Extracranial Carotid Atherosclerosis and Vascular Risk Factors in Different Types of Ischemic Stroke in Taiwan

Jiann-Shing Jeng, MD; Ming-Yao Chung, MD; Ping-Keung Yip, MD; Bao-Show Hwang, BS; Yang-Chyuan Chang, MD

Background and Purpose: The clinical patterns of stroke and the angiographic distribution of cerebral atherosclerosis in Chinese people are different from those in whites. Studies relating carotid atherosclerosis and vascular risk factors to various types of stroke in Chinese people are lacking.

Methods: Based on clinical information, we separated 367 stroke patients living in Taiwan into four subgroups: cortical infarction (CI), subcortical infarction (SCI), vertebrobasilar artery infarction (VBAI), and cardioembolic infarction (CEI). We assessed the extent and severity of extracranial carotid artery atherosclerosis in different types of ischemic stroke using duplex ultrasonography. Vascular risk factors and carotid atherosclerosis were then correlated with each subgroup of ischemic stroke.

Results: Our data revealed that 32% of the CI subgroup, 3% of the SCI subgroup, 7% of the VBAI subgroup, and 21% of the CEI subgroup possessed severe carotid stenosis (≥50% stenosis or occlusion). The extent of atherosclerosis of extracranial carotid arteries, measured by plaque score, was also more severe in the CI subgroup than in the other subgroups. Diabetes mellitus was more frequent in the CEI subgroup. Cardiomegaly and left ventricular hypertrophy were more commonly seen in the CEI subgroup. The VBAI subgroup was younger than the other subgroups. There were no differences in hypertension, prior stroke, alcohol intake, or serum levels of glucose, uric acid, hematocrit, lipids, and lipoproteins among the subgroups.

Conclusions: Of the Chinese patients living in Taiwan, the extent and severity of extracranial carotid artery atherosclerosis were more prominent in patients with CI than in patients with other types of ischemic stroke. In Chinese patients with CI, severe carotid stenosis is not uncommon; in Chinese patients with SCI, however, the frequency of carotid stenosis is quite low. (Stroke. 1994;25:1989-1993.)

Key Words: • atherosclerosis • carotid arteries • cerebrovascular disorders • Chinese • stenosis

Atherosclerosis has always been regarded as an important component of the pathogenesis of ischemic cerebrovascular disease (CVD). However, the pathogenesis of CVD is heterogeneous. Therefore, the atherosclerotic pattern of the cerebral arteries and the associated risk factors may be dissimilar in different types of ischemic CVD.

Pathological and angiographic studies indicate that variations in the occlusive lesion sites in various segments of the cerebrovascular tree in CVD may stem from racial differences. Chinese patients were shown to have fewer extracranial lesions and more severe intracranial lesions than whites. However, these studies were biased toward carefully selected patients with more severe or vulnerable conditions. Duplex carotid sonography is a noninvasive, reproducible, and sensitive method to evaluate the extent and severity of extracranial carotid artery (ECCA) atherosclerosis, thus providing the opportunity to determine the role of ECCA atherosclerosis in various types of ischemic CVD in Chinese patients.

This study assesses the extent and severity of ECCA atherosclerosis quantitatively in different types of ischemic CVD for those Chinese patients living in Taiwan, and it compares the associated vascular risk factors among them.

Subjects and Methods

The study population comprised 367 consecutive Chinese patients (222 men, 145 women) with acute ischemic stroke who were admitted to National Taiwan University Hospital between July 1990 and October 1993. Stroke was defined as a rapidly developing episode of focal loss of cerebral function with symptoms lasting more than 24 hours and with no apparent cause other than that of vascular origin. Patients with any of the following were excluded: intracerebral hemorrhage, subarachnoid hemorrhage, transient ischemic attack, and embolic stroke from rheumatic heart disease or brain tumors. Patients with liver, biliary, renal, or thyroid diseases were also excluded because these illnesses might influence lipid and lipoprotein metabolism.

Based on clinical characteristics and brain computed tomography (CT) and/or magnetic resonance imaging (MRI) studies, the stroke was classified into four subgroups: cortical infarction (CI), subcortical infarction (SCI), vertebrobasilar artery infarction (VBAI), and cardioembolic infarction (CEI). The strokes were subtyped without knowledge of the ultrasonographic results. The CI subgroup was composed of patients with clinical evidence of stroke with cortical deficits whose brain CT and/or MRI studies showed an infarct involving the cortex of the carotid artery territory without evidence of cardiogenic source of embolus. The SCI subgroup was composed of patients whose brain CT and/or MRI studies showed...
a small infarct in the subcortical areas of the carotid artery territory that was responsible for the stroke. Patients with combined cortical and subcortical involvement, as seen on the images, were included in the CI subgroup. The VBAI subgroup consisted of patients with clinical evidence of neurological deficits in the territory of posterior circulation and compatible findings on brain CT and/or MRI. CI with evidence of emboli that originated from cardiogenic conditions was classified as CEI. These cardiogenic conditions include atrial fibrillation or flutter, valvular heart disease excluding rheumatic heart disease, prosthesis heart valve, recent (<6 weeks) myocardial infarction, endocarditis, and ischemic cardiomyopathy.

A detailed history of potential vascular risk factors and conditions assumed to be associated with carotid atherosclerosis was obtained from the patient and from the hospital records. Included were age, sex, history of hypertension, diabetes mellitus, previous stroke, cigarette smoking (>10 cigarettes daily for >10 years), and alcohol drinking. After a 10-hour overnight fast, a venous blood sample was drawn from each patient on the second day of admission (usually within the first 7 days after ictus) for the measurement of serum levels of blood glucose, uric acid, cholesterol, triglyceride, low-density lipoprotein cholesterol, and high-density lipoprotein cholesterol. On a standard chest roentgenogram, cardiomegaly was defined as a cardiothoracic ratio ≥50%. Left ventricular hypertrophy (LVH) on electrocardiogram (ECG) was defined as voltage of SV_{1} plus RV_{1} of ≥3.5 mV with ST-segment and T-wave changes of left ventricular strain. Ischemic heart disease was coded as present if the patient had a history of myocardial infarction or angina pectoris and/or old myocardial infarction or ischemic changes on the ECG.

An ultrasonicographic assessment of ECCA atherosclerosis was carried out in every patient. A Diasonics DRF 400 duplex ultrasound system with a 7.5-MHz scanning frequency in real-time B-mode and 3.0-MHz scanning frequency in pulsed-Doppler mode was used for the evaluation. The duplex scanning was performed by trained physicians or an experienced ultrasonographer while the patient was lying in the supine position. Scan images were recorded on super VHS videotapes and were interpreted independently by two different readers (B.-S.H. and J.-S.J.) who were blinded to the stroke subtypes.

The extent and severity of ECCA atherosclerosis in each patient were evaluated by two indexes: plaque score and maximum stenosis. The plaque scoring system is a modification of the method of Sutton et al.\textsuperscript{12} The examination included longitudinal and transverse views of the proximal common carotid artery (≥20 mm proximal to bulb bifurcation), distal common carotid artery (<20 mm proximal to bulb bifurcation), carotid bulb bifurcation areas, internal carotid artery, and external carotid artery bilaterally. A grade was assigned for each chosen segment: grade 0, normal or no observable plaque; grade 1, one small plaque with diameter stenosis <30%; grade 2, one medium plaque with 30% to 49% diameter stenosis or multiple small plaques; grade 3, one large plaque with 50% to 99% diameter stenosis or multiple plaques with at least one medium plaque; and grade 4, 100% occlusion. The plaque score was computed by summing the plaque grades at each of the segments of the ECCA. The assessment of the degree of stenosis was based on the kilohertz shift, the waveform, and the real-time B-mode images. Percent stenosis was computed by measuring the residual lumen diameter and the original diameter at the site of maximal stenosis in each segment of the arteries and dividing the difference by the original diameter. Stenosis ≥50% was also defined in the presence of peak systolic frequency ≥2.4 kHz (or peak systolic velocity ≥1.248 m/s). The maximum stenosis, selected by the maximum percent stenosis of the ECCA in each patient, was coded as follows: no lesion, mild to moderate stenosis (<50%), and severe stenosis (≥50%).

Statistical analyses were performed using the SAS package.\textsuperscript{13} Analysis of variance with Scheffé's test was used to detect the difference in continuous risk factors among the subgroups of stroke. The Mantel-Haenszel χ^{2} test was applied to examine the difference in categorical risk factors among the subgroups of stroke. Multivariate linear regression with stepwise procedure was used to fit the best model for the extent of ECCA atherosclerosis.

### Results

This analysis comprised 367 patients with ischemic stroke. Each patient underwent brain CT and/or MRI studies at least once. Of these, 96 (26%), 177 (48%), 61 (17%), and 33 (9%) patients were included in the CI, SCI, VBAI, and CEI subgroups, respectively.

The clinical characteristics of the four subgroups are shown in Table 1. Patients in the VBAI subgroup were younger than the other subgroups (mean age). The VBAI subgroup included more male patients than the CEI subgroup. History of diabetes mellitus was more frequently in the CI subgroup. Presence of cardiomegaly on chest roentgenogram and LVH on ECG were more frequently seen in the CEI subgroup. In regard to the other vascular risk factors, including hypertension, previous stroke, alcohol drinking, serum levels of blood glucose, uric acid, hematocrit, lipid, and lipoproteins, our data showed no significant differences among these subgroups.

Of the total of 367 patients, 48 patients (13%) had severe carotid stenosis (≥50% stenosis or occlusion), 179 patients (49%) had mild to moderate carotid stenosis (1% to 49% stenosis), and 140 patients (38%) had no carotid stenosis (Table 2). A further division in each stroke subgroup revealed that 32% of the CI subgroup, 21% of the CEI subgroup, 7% of the VBAI subgroup, and 3% of the SCI subgroup possessed severe carotid stenosis. The CI and CEI subgroups had significantly more severe carotid stenosis than the SCI (P<.05) or the VBAI (P<.05) subgroups. There was no significant difference in maximum stenosis between the CI and CEI subgroups (P=.23).

The extent of ECCA atherosclerosis is presented in Table 3. The mean plaque scores of the CI, SCI, VBAI, and CEI subgroups were 5.3, 2.0, 2.4, and 2.9, respectively. The plaque score was significantly higher in the CI patients than in the SCI patients (P<.001), the VBAI patients (P=.002), and the CEI patients (P=.02). There was no significant difference in the plaque score among the SCI, VBAI, and CEI subgroups. Age, male sex, LVH on ECG, diabetes, and cholesterol emerged as independent predictors of the extent of ECCA atherosclerosis in multivariate analyses (Table 4).

### Discussion

The pathogenesis of ischemic stroke, including atherothrombosis of the larger arteries, arteriosclerosis of the smaller arteries, cardiac or arterial embolization, and hemodynamic ischemia, is heterogeneous.\textsuperscript{5} Therefore, the atherosclerotic pattern of the cerebral arteries and the associated risk factors may be different in the various types of ischemic stroke. Large-artery thrombosis was estimated to cause approximately 40% of all ischemic strokes in the Harvard Cooperative Stroke Registry study.\textsuperscript{14} Information from the Stroke Data Bank indicates that after thorough investigation, ap-
proximately 24% of ischemic strokes can be attributed
to large-artery stenosis or thrombosis, and 31% are
considered to be lacunar infarction.\textsuperscript{15} CI is charac-
terized by large-artery atherosclerosis.\textsuperscript{2-16} We used SCI
instead of lacunar infarction because the definition of
lacunar infarction varies from study to study and be-
cause the clinical manifestations of some patients were
not typical lacunar syndromes or their neuroimages did
not show typical sites for lacunar infarction. Lacunar
infarction remains a controversial entity\textsuperscript{19} because deep
infarcts can also be caused by large-artery occlusion,
emboli, or intracranial branch atheromatous occlusion
in addition to a small penetrating artery disease, as
originally described by Fisher.\textsuperscript{20-21} We thought that the
subgroup of SCI in this study might include supratentor-
ial lacunar infarction.

Autopsy and angiographic studies have shown that
ECCA disease is frequently seen in cortical or nonlacu-
lar infarction and infrequently in lacunar infarction.\textsuperscript{2,3,18,22} However, these studies were subject to bias
toward highly selected patients with more severe or
vulnerable conditions. Only a few studies have enough
samples that are unbiased to provide sufficient data
regarding the extent and severity of atherosclerosis of
cerebral arteries in the different types of ischemic
stroke. High-resolution carotid duplex scanning has
been widely recognized as a noninvasive, reproducible,
and accurate method of evaluating ECCA atherosclero-
sis. The sensitivity of duplex scanning in the detection of
carotid stenosis is high (88\% to 95\%)\textsuperscript{23-25}; the accuracy
of duplex (79\% to >90\%) was established by a blind
comparison with angiography.\textsuperscript{23,26}

We used 50\% stenosis as the cutoff point because
lesions of <50\% diameter reduction carry a very low
risk of stroke\textsuperscript{23,27} and a high degree of carotid stenosis is
significantly correlated with ischemic stroke, progress-
ing carotid artery stenosis, and ischemic heart dis-
ease.\textsuperscript{23,27-31} Many published reports have demonstrated
that racial differences exist in the distribution of cere-
bral atherosclerosis in stroke victims. Whites tend to
have more extracranial vascular lesions, whereas Orien-
tals and blacks tend to have more intracranial vascular
lesions.\textsuperscript{2,3,6-9} However, these results are primarily based
on angiography or autopsy. Only a few studies have used
carotid ultrasonography to evaluate the degree of ca-
rotid artery atherosclerosis in different subtypes of
stroke. Boiten and Lodder\textsuperscript{16} found that 37\% of 94
patients with CI had ipsilateral internal carotid artery
stenosis of \(\geq 50\%\). Only 13\% of 103 patients with
lacunar infarction had ipsilateral internal carotid artery
stenosis of \(\geq 50\%\). Tegeler et al\textsuperscript{17} yielded similar results,
in which 41\% of 54 patients with nonlacunar infarction
and 13\% of 55 patients with lacunar infarction had
ipsilateral internal carotid artery stenosis of \(\geq 50\%\).

<table>
<thead>
<tr>
<th>Characteristics</th>
<th>CI (n=96)</th>
<th>SCI (n=177)</th>
<th>VBAI (n=61)</th>
<th>CEI (n=33)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age, y</td>
<td>Mean (SD)</td>
<td>68.3 (9.5)*</td>
<td>64.8 (10.0)*</td>
<td>60.4 (11.0)*</td>
</tr>
<tr>
<td>Range</td>
<td></td>
<td>32-87</td>
<td>33-88</td>
<td>37-79</td>
</tr>
<tr>
<td>Male</td>
<td></td>
<td>64</td>
<td>66</td>
<td>42*</td>
</tr>
<tr>
<td>Hypertension</td>
<td></td>
<td>65</td>
<td>66</td>
<td>28*</td>
</tr>
<tr>
<td>Diabetes mellitus</td>
<td></td>
<td>42*</td>
<td>28*</td>
<td>26*</td>
</tr>
<tr>
<td>Previous stroke</td>
<td></td>
<td>23</td>
<td>19</td>
<td>21</td>
</tr>
<tr>
<td>IHD</td>
<td></td>
<td>49</td>
<td>40</td>
<td>35†</td>
</tr>
<tr>
<td>Smoking</td>
<td></td>
<td>43</td>
<td>39</td>
<td>53</td>
</tr>
<tr>
<td>Drinking alcohol</td>
<td></td>
<td>17</td>
<td>18</td>
<td>21</td>
</tr>
<tr>
<td>Cardiomegaly</td>
<td></td>
<td>36*</td>
<td>25*</td>
<td>18*</td>
</tr>
<tr>
<td>LVH on ECG</td>
<td></td>
<td>37*</td>
<td>25*</td>
<td>20*</td>
</tr>
</tbody>
</table>

CI indicates cortical infarction; SCI, subcortical infarction; VBAI, vertebrobasilar artery infarction; CEI, cardioem-bolic infarction; IHD, ischemic heart disease; LVH, left ventricular hypertrophy; and ECG, electrocardiogram. Values (except age) are percentages.

\*P<.05, \†P=.07 by Mantel-Haenszel \(x^2\) test.

proximately 24% of ischemic strokes can be attributed
to large-artery stenosis or thrombosis, and 31% are
considered to be lacunar infarction.\textsuperscript{15} CI is charac-
terized by large-artery atherosclerosis.\textsuperscript{2-16} We used SCI
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<table>
<thead>
<tr>
<th>Subgroups</th>
</tr>
</thead>
<tbody>
<tr>
<td>CI</td>
</tr>
<tr>
<td>SCI</td>
</tr>
<tr>
<td>VBAI</td>
</tr>
<tr>
<td>CEI</td>
</tr>
<tr>
<td>Total</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>ECCA Stenosis</th>
<th>No.</th>
<th>%</th>
<th>No.</th>
<th>%</th>
<th>No.</th>
<th>%</th>
<th>No.</th>
<th>%</th>
<th>No.</th>
<th>%</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt;50%</td>
<td>65</td>
<td>68</td>
<td>171</td>
<td>97</td>
<td>57</td>
<td>93</td>
<td>26</td>
<td>79</td>
<td>319</td>
<td>87</td>
</tr>
<tr>
<td>(\geq 50%)</td>
<td>31</td>
<td>32</td>
<td>6</td>
<td>3</td>
<td>4</td>
<td>7</td>
<td>7</td>
<td>21</td>
<td>48</td>
<td>13</td>
</tr>
</tbody>
</table>

ECCA indicates extracranial carotid artery; CI, cortical infarction; SCI, subcortical infarction; VBAI, vertebrobasilar artery infarction; and CEI, cardioembolic infarction.
data disclosed that 32% of CI patients and 3% of SCI patients possessed severe carotid stenosis. Significant carotid stenosis (≥50% stenosis) was nine times more prevalent in patients with CI compared with patients with SCI. The findings that the severity of ECCA atherosclerosis was more prominent in CI than in other types of ischemic infarction have been shown again in the Chinese patients.

The methods for the quantitation of the extent of ECCA atherosclerosis were introduced recently.12,32 We used a scoring system, modified from the method of Sutton et al.,12 to present the extent of ECCA atherosclerosis in our stroke patients. The plaque score showed a significant trend toward higher scores in the CI subgroup than the other subgroups. There was no significant difference in the ECCA plaque score among the SCI, VBAI, and CEI subgroups. The extent of ECCA atherosclerosis, reflecting generalized atherosclerosis and atherothrombotic brain infarction,6,34-35 has been found to correlate better with vascular risk factors than the severity of the disease.32,33 In this group of stroke patients, age, male sex, LVH on ECG, diabetes, and cholesterol level were the independent predictors of the extent of ECCA atherosclerosis in multivariate analyses.

Hypertension is a well-established risk factor for stroke and is presumed to be the most common risk factor for lacunar infarction. Fisher6,34 originally suggested that the smaller penetrating intracranial vessels are often damaged by hypertension. Seventy-five percent of patients clinically diagnosed as having lacunar infarction in the Harvard Cooperative Stroke Registry had hypertension.1 In the present study, except for the CEI subgroup, the percentage of hypertension in the other subgroups was equally high (61% to 66%). This might indicate that hypertension contributes similarly to large- and small-artery diseases in the Chinese stroke patients.

Diabetes imparts a definite independent risk of atherosclerosis and atherothrombotic brain infarction.34,35 Many studies have failed to find a correlation between diabetes or blood glucose levels and CI and lacunar infarction.14,16,17,36 However, our analysis revealed that a history of diabetes was more prevalent in the CI subgroup than in other subgroups.

A number of investigations relating blood lipids and lipoproteins with CVD have been reported; however, the results are inconsistent.37-40 Some recent investigations have suggested that separation of ischemic CVD into subgroups may help to elucidate this association.2,15,39 In the Akita Pathology Study, serum cholesterol levels were positively associated with CI and inversely associated with cerebral hemorrhage.2 The data of Adams et al.39 showed that patients with lacunar infarction had higher concentrations of low-density lipoprotein cholesterol than patients with CI. Our results did not show significant differences in blood lipids and lipoproteins among the subgroups of stroke but only disclosed that cholesterol was an independent risk factor for the extent of ECCA atherosclerosis.

In conclusion, our study revealed that severe carotid stenosis in Chinese patients with CI is not uncommon; however, the frequency of carotid stenosis is quite low in patients with SCI.

### References


### Table 3. Distribution of Extracranial Carotid Artery Atherosclerosis of 367 Patients With Different Types of Ischemic Stroke

<table>
<thead>
<tr>
<th>Subgroups</th>
<th>CI</th>
<th>SCI</th>
<th>VBAI</th>
<th>CEI</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Plaque Score</td>
<td>No.</td>
<td>%</td>
<td>No.</td>
<td>%</td>
<td>No.</td>
</tr>
<tr>
<td>0</td>
<td>24</td>
<td>25</td>
<td>79</td>
<td>45</td>
<td>22</td>
</tr>
<tr>
<td>1-3</td>
<td>19</td>
<td>20</td>
<td>61</td>
<td>34</td>
<td>23</td>
</tr>
<tr>
<td>4-6</td>
<td>21</td>
<td>22</td>
<td>24</td>
<td>14</td>
<td>9</td>
</tr>
<tr>
<td>&gt;6</td>
<td>32</td>
<td>33</td>
<td>13</td>
<td>7</td>
<td>7</td>
</tr>
</tbody>
</table>

CI indicates cortical infarction; SCI, subcortical infarction; VBAI, vertebrobasilar artery infarction; and CEI, cardioembolic infarction.

### Table 4. Relation of Risk Factors to Plaque Score: Multivariate Analysis

<table>
<thead>
<tr>
<th>Risk Factor</th>
<th>Regression Coefficient</th>
<th>SE</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age, y</td>
<td>.103</td>
<td>.0196</td>
<td>.0001</td>
</tr>
<tr>
<td>Male sex</td>
<td>1.389</td>
<td>.4157</td>
<td>.0018</td>
</tr>
<tr>
<td>LVH</td>
<td>1.432</td>
<td>.4425</td>
<td>.0019</td>
</tr>
<tr>
<td>Diabetes</td>
<td>1.275</td>
<td>.4332</td>
<td>.0038</td>
</tr>
<tr>
<td>Cholesterol, mg/dL</td>
<td>.0115</td>
<td>.0047</td>
<td>.0017</td>
</tr>
</tbody>
</table>

LVH indicates left ventricular hypertrophy on electrocardiogram.


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doi: 10.1161/01.STR.25.10.1989
*Stroke* is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
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Print ISSN: 0039-2499. Online ISSN: 1524-4628

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