Deep Cerebral Infarcts in the People's Republic of China

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We reviewed computed tomograms and clinical characteristics for 1,124 consecutive stroke patients from Shanghai, People's Republic of China. While there were many similarities between lacunar syndromes in these patients and patients from Europe and the United States, several differences were noted. Deep cerebral infarcts identified by computed tomography were more common in patients from Shanghai and accounted for 27% of all ischemic infarcts. Small deep infarcts occurred more commonly in women from Shanghai (44%) than in women from the West. Deep cerebral infarcts in patients from Shanghai were larger than those usually seen in patients from the West. The mean infarct volume in patients from Shanghai was 2.4 ml, and 49% were giant lacunes (>15 mm in maximal diameter). (Stroke 1990;21:394-396)

Cerebral vascular disease is a major cause of morbidity and mortality in the People's Republic of China. In 1982, an epidemiologic study found cerebrovascular disease to account for 27% of deaths.1 The prevalence ratio of completed strokes for six Chinese cities was 719/100,000 population, and the incidence of first stroke was 220/100,000 population2; 51% of first strokes were due to cerebral infarction. It has been difficult to classify cerebral infarcts as deep (lacunar) or superficial (nonlacunar) because autopsies are seldom done in China, cerebral arteriography is seldom performed on stroke patients, and computed tomography (CT) was generally not available before 1984.2 We report the clinical and CT characteristics of patients with deep cerebral infarcts seen at two Shanghai teaching hospitals during 30 months.

Subjects and Methods

The stroke patients were seen at the Ren Ji Hospital and the Second Hospital of the Shanghai Textile Industry of the Shanghai Second Medical University. All CT scans indicating a diagnosis of cerebrovascular disease between January 1986 and July 1988 were reviewed without knowledge of the patient's clinical signs. CT studies were performed without contrast on a SIMA 9000T device. A patient was included in the study if the lesion on CT was hypodense and sharply marginated without a mass effect, if it was subcortical or near the basal ganglia, if the patient's clinical signs were consistent with a lacunar syndrome,3 and if the CT scan was performed ≤6 weeks after clinical onset. We grouped the patients by onset of neurologic symptoms. If hemiparesis reached maximum severity within 1 day, the onset was abrupt; if weakness got progressively worse for up to 4 days, the onset was gradual. We excluded patients with brainstem lacunes. The maximum diameters of the CT lesion were measured at right angles to each other, corrected for the reduced size of the CT image, and classified as giant (>15 mm) or small (≤15 mm). Infarct volume was estimated according to Nelson et al4 (volume=1/2 length×width×depth). Most patients seen in the emergency room or outpatient clinic with a possible acute stroke underwent CT. Emergency room, inpatient, and outpatient records and CT request forms were reviewed for each patient. Information regarding stroke risk factors, clinical signs, and stroke outcome was recorded for each patient. Not all information was available for each patient. Muscle strength was scored from 0 to 5 according to the Medical Research Council rating scale. Hemiparesis was categorized as absent (score of 5), mild (score of 4), moderate (score of 3), or severe (score of 0–2).

Fisher's exact test was used to compare the proportions of patients in each of the four hemi-
TABLE 1. Location of Lacunar Infarcts in 144 Patients by Computed Tomography Without Contrast

<table>
<thead>
<tr>
<th>Location</th>
<th>No.</th>
<th>%</th>
</tr>
</thead>
<tbody>
<tr>
<td>Lenticular nucleus</td>
<td>72</td>
<td>50</td>
</tr>
<tr>
<td>Caudate nucleus</td>
<td>32</td>
<td>22</td>
</tr>
<tr>
<td>Thalamus</td>
<td>19</td>
<td>13</td>
</tr>
<tr>
<td>Internal capsule</td>
<td>129</td>
<td>90</td>
</tr>
<tr>
<td>Genu/anterior limb</td>
<td>46</td>
<td>32</td>
</tr>
<tr>
<td>Posterior limb</td>
<td>74</td>
<td>51</td>
</tr>
<tr>
<td>Corona radiata</td>
<td>16</td>
<td>11</td>
</tr>
</tbody>
</table>

Infarcts often involved more than one site.

Paresis severity categories with infarcts in a given location. The \( \chi^2 \) test was used to compare the demographic and clinical characteristics of the patients in the two age classes, the two infarct diameter classes, and the two onset groups. The level of significance was taken as \( p < 0.05 \).

Results

We reviewed noncontrasted CT scans from 1,124 consecutive stroke patients; 540 patients (48%) had ischemic infarcts, and 144 (27%) of the 540 patients with ischemic infarcts had deep cerebral infarcts. The mean \( \pm \) SD age of these 144 patients was 64 \( \pm \) 11 years; 63 (44%) were women. Of the patients in whom information was available, 74% had a history of hypertension (blood pressure of \( > 160/95 \) mm Hg), 3% had a history of diabetes mellitus, and 19% had experienced a previous stroke.

A history consistent with a lacunar syndrome was found in all 144 patients. Hemiparesis was present in 97%. Weakness involved the right side in 51% and was mild in 55%, moderate in 18%, and severe in the remaining 27%. In 56% of the patients the degree of arm and leg weakness was equal, in 33% the arm was weaker than the leg, and in the remaining 11% the leg was weaker than the arm. Limb sensory loss occurred in 13%, and speech disturbances (mainly dysarthria) were found in 23%. None had homonymous hemianopsia or parietal lobe signs. Motor recovery occurred in \( \leq 2 \) weeks in 25%, in 2–4 weeks in 23%, and in \( > 4 \) weeks in the remaining 52%.

CT was performed an average of 7 (range 1–40) days after the onset of neurologic signs. Table 1 lists the locations of the infarcts. Twenty patients (14%) had old lacunes and three (2%) had old superficial infarcts. The maximum diameters of the lacunes are shown in Table 2; 49% were giant lacunes. The mean \( \pm \) SD volume of the infarcts was 2.4 \( \pm \) 2.7 ml.

TABLE 2. Maximum Diameter of Lacune in 144 Patients on Computed Tomograms Without Contrast

<table>
<thead>
<tr>
<th>Diameter (mm)</th>
<th>No.</th>
<th>%</th>
</tr>
</thead>
<tbody>
<tr>
<td>0–9</td>
<td>12</td>
<td>8</td>
</tr>
<tr>
<td>10–15</td>
<td>61</td>
<td>42</td>
</tr>
<tr>
<td>16–20</td>
<td>37</td>
<td>26</td>
</tr>
<tr>
<td>&gt;20</td>
<td>34</td>
<td>24</td>
</tr>
</tbody>
</table>

No significant differences were found between the 72 patients <65 years old and the 72 patients \( \geq 65 \) years old with respect to history of hypertension, diabetes mellitus, or previous stroke, to severity of hemiparesis, to infarct diameter or volume, and to time to recovery.

Infarct location correlated with hemiparesis severity. Infarcts involved only the anterior limb of the internal capsule in seven of the eight patients (88%) with no hemiparesis, 17 of the 55 patients (31%) with mild hemiparesis, some of the 52 patients with moderate hemiparesis, and five of the 29 patients (17%) with severe hemiparesis (\( p < 0.01 \)). Infarcts involved the posterior limb of the internal capsule in none of the eight patients with no hemiparesis, 31 of the 55 patients (56%) with mild hemiparesis, some of the 52 patients with moderate hemiparesis, and 21 of the 29 patients (72%) with severe hemiparesis (\( p < 0.001 \)).

No significant differences were found between patients with giant and small deep infarcts with respect to mean age, to sex ratio, to history of hypertension, diabetes mellitus, or previous stroke, or to severity of hemiparesis. However, patients with small infarcts recovered significantly faster (\( p < 0.05 \)).

In 56% of the patients hemiparesis developed abruptly, but in the remaining 44% the weakness had a gradual onset. Patients with a gradual onset of neurologic signs were less likely to be hypertensive (54% and 79%, respectively; \( p < 0.05 \)) and more likely to have mild hemiparesis (74% and 46%, respectively; \( p < 0.05 \)) than patients with an abrupt onset. No significant differences were found between these groups for mean age, sex ratio, history of diabetes mellitus or previous stroke, infarct volume, or time to recovery.

Discussion

Our study was potentially limited by the lack of cerebral arteriographic or autopsy confirmation of the deep infarcts. Unfortunately, arteriography and autopsies are seldom performed in China, so little is known about the cause and location of the occlusion. Thus, it is possible that some of the larger deep infarcts have been caused by middle cerebral artery stenosis and therefore do not conform to the common definition of a lacune.

In many ways, patients from Shanghai with deep infarcts had clinical and CT characteristics similar to those seen in patients from the United States and Europe. Compared with patients in Western stroke registries, patients from Shanghai were similar with respect to age, clinical signs, frequency of gradual onset of neurologic signs, incidence of hypertension, and anatomic location of the infarct.

Stroke patients in Shanghai also differed from those in the United States and Europe. First, Chinese patients had a higher proportion of deep infarcts. In our study, 27% of the 540 patients with an ischemic stroke by CT had deep infarcts, the same figure reported in a stroke registry from Taiwan, Republic of China. In Western stroke registries,
however, the percent of ischemic infarcts diagnosed as lacunes range from 12% to 23%.7–9,13 While the formats of the Western stroke registries differ, most define a lacune by clinical characteristics and not by CT findings. Several stroke registries have found that the CT scan was positive in 35–50% of patients with lacunes.8,14,15 This makes the difference between the frequency of lacunar syndromes identified by CT in Shanghai and the West even more striking. Second, more women suffered lacunes in Shanghai than in the West. Chinese women comprised 44% of the patients with lacunar infarcts, compared with an average of 28% in the West.4,5,11 The Oxfordshire Community Stroke Registry, however, has reported no substantial sex difference in the risk of small deep infarction.15 Third, deep infarcts were generally larger in Shanghai patients than in patients from the West. We found the deep infarcts to have a mean volume of 2.4 ml and a mean diameter of 17 (range 1.0–40) mm; 71 patients (49%) had deep infarcts with a maximum diameter of >15 mm. In several Western studies, mean infarct volume varied from 0.7 to 1.8 ml and mean diameter from 7 to 13 (range 3–32) mm6,10,16; giant lacunes generally represented <10% of all lacunes.5,6 However, one study reported giant lacunes in 58% of patients with infarcts in the territory of deep perforators of the internal carotid artery system.17

The reasons for the differences between deep cerebral infarcts seen in patients from the People’s Republic of China and those from the West are poorly understood. Hypertension appears to be a predisposing factor in both. However, autopsies and cerebral arteriographic studies are needed in Chinese patients to ascertain if the anatomic bases for the deep infarcts are the same.

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References

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