Cerebrovascular Disease in Hong Kong Chinese

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Our prospective study of cerebrovascular disease in Hong Kong confirms a previous clinical impression that stroke in the Chinese has a pattern different from that in Caucasians. We studied 540 patients (aged 20–70 years) with stroke. Computed tomography or autopsy was obtained in 86.1% and showed an increase in the proportion with lacunar infarction, striatocapsular infarction, and parenchymal hemorrhage relative to the frequencies in Caucasians. This increase in the incidence of cerebral hemorrhage occurs not only in semicomatose and comatose patients but also in alert patients (16.9%) and those with a lacunar syndrome (12.5%). Our findings suggest that cerebrovascular disease in the Chinese selectively affects small vessels, causing lacunes and hemorrhages. In future community studies on stroke prevalence, researchers should be cautious about interpreting similar prevalence rates as reflecting similar risk factors or pathologies. (Stroke 1990;21:230–235)

The pattern of cerebrovascular disease in a community is important for diagnostic, therapeutic, rehabilitative, and preventive purposes. While important epidemiology data are obtained from community-based surveys, accurate categorization of the types of stroke can be achieved best with hospital-based studies in which neurologic opinion, computed tomography (CT), or autopsy are available.

Between 6.3% and 11.9% of strokes in Caucasians are due to intracerebral hemorrhage; in the Chinese, however, 21–48% of strokes are estimated to be hemorrhagic. This difference has been attributed to possible differences in admission policy or diagnostic accuracy, as well as to the age distribution of the populations. Two recent community-based studies in China reported an even higher incidence of intracerebral hemorrhage, but the diagnosis was based on clinical characteristics and cerebrospinal fluid (CSF) examination, without CT or autopsy confirmation.

In this hospital-based study, we attempted to determine among the Chinese living in Hong Kong the pathologic categorization of stroke within different clinical stroke types.

Subjects and Methods

The population of Hong Kong is almost entirely urban; 1.1 million people live on the island of Hong Kong itself. In 1984, only 2.3% of the population was >74 years old (Table 1). The annual age-specific death rate between 1978 and 1980 was close to that of many Western countries in 1961, with a crude annual death rate of approximately 65/100,000 population.

Queen Mary Hospital is the major public hospital on the island of Hong Kong. There are also one small voluntary hospital and three private hospitals on the island, but it is estimated that Queen Mary Hospital admits approximately 80% of all acute medical cases. All stroke patients coming to the emergency room are admitted as a matter of policy. The hospital is within an hour by car from all parts of the island, and 86% of all stroke admissions present to the hospital ≤24 hours after the onset of the illness. Patients with stroke aged 20–70 years and of Chinese descent who were admitted from April 1, 1984, through March 31, 1985, into the University Medical Unit were entered into this study, which represents approximately two thirds of the stroke cases admitted to the hospital since this unit alternates with another one on a 2:1 admission day schedule. Stroke was defined as rapidly developing signs of focal or global disturbance of cerebral function leading to death or lasting >24 hours with no apparent cause other than vascular. Patients with transient ischemic attacks (TIA), defined as deficits lasting <24 hours, were excluded, as were patients referred from other hospitals or units.

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TABLE 1. Age and Sex Distribution of Hong Kong Island Population, 1984-1985

<table>
<thead>
<tr>
<th>Age (yr)</th>
<th>Male</th>
<th>Female</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt;20</td>
<td>204,440</td>
<td>189,610</td>
</tr>
<tr>
<td>20-44</td>
<td>252,628</td>
<td>219,263</td>
</tr>
<tr>
<td>45-54</td>
<td>60,664</td>
<td>51,098</td>
</tr>
<tr>
<td>55-64</td>
<td>47,501</td>
<td>45,067</td>
</tr>
<tr>
<td>65-74</td>
<td>26,210</td>
<td>30,742</td>
</tr>
<tr>
<td>≥75</td>
<td>8,303</td>
<td>18,181</td>
</tr>
<tr>
<td>Total</td>
<td>599,746</td>
<td>553,961</td>
</tr>
</tbody>
</table>

Data are number of people.

and those who experienced the onset of stroke >5 days before admission.

Each patient was examined by a neurologist ≤24 hours after admission and was subsequently followed. On admission, the patients were categorized as alert, semicomatose, or comatose. Coma was defined as a lack of spontaneous speech, purposeful movement, or eye opening or the absence of these responses to verbal stimuli; noncomatose patients who were confused, retarded in response, or lacking in spontaneity were categorized as semicomatose; and those with a normal degree of alertness without any impairment of sensorium were categorized as alert.

The clinical deficits were designated as lacunar syndrome, complicated motor paresis, no weakness, minimal weakness, and totally nonresponsive. A lacunar syndrome was diagnosed when the patient had either pure motor hemiparesis, ataxic hemiparesis, dysarthria–clumsy hand syndrome, pure sensory stroke, sensorimotor stroke, or isolated supranuclear facial palsy.16-18 Sensorimotor stroke was diagnosed only when there was incomplete motor or sensory impairment.17 The absence of any disturbance of alertness, dysphasia, intellectual change, neglect, hemianopsia, or seizures accompanying the illness was required for the diagnosis of a lacunar syndrome. Patients who had at least one limb with motor power less than Medical Research Council grade 4 but who did not meet the criteria for any of the above lacunar syndromes were designated as having complicated motor paresis. Patients who were admitted in a coma and lacking motor responses were designated as nonresponsive. Those with evidence of weakness of grade 4 or better, such as is manifested by a drift, were designated as having a minimal motor deficit.

Subarachnoid hemorrhage was diagnosed when there was focal or generalized collection of blood in the basal cisterns on CT, convexity of the subarachnoid space on CT, or blood-stained CSF without CT evidence of a parenchymal hematoma. An effort was made to perform CT or autopsy in all cases. Patients who had a lumbar puncture showing heavily blood-stained CSF but had no CT or autopsy were classified as having intracranial hemorrhage (type unspecified). Patients who had neurologic deficits lasting >24 hours and a nondiagnostic CT scan were classified as having ischemic stroke after exclusion of subarachnoid hemorrhage. We defined subcortical ischemic lesions of <4 ml3 as lacunes and those larger as subcortical infarcts.

**Results**

We included 540 patients who satisfied the entry criteria in our study; we excluded another 51 who presented with TIA during the same period. CT was obtained in 433 of the 540 patients (80.2%) and autopsy was performed in 32 more (5.9%); therefore, the lesion was confirmed in 86.1%. Of the 433 patients who had CT, it was performed during the first 3 days after stroke onset in 231 (53.4%), 4-7 days after stroke onset in 81 (18.7%), 2-3 weeks after stroke onset in 101 (23.3%), during the fourth week after stroke onset in six (1.4%), and >4 weeks by guest on November 10, 2017 http://stroke.ahajournals.org/ Downloaded from

TABLE 2. Age and Sex Distribution of 540 Stroke Patients by Stroke Type

<table>
<thead>
<tr>
<th>Stroke type</th>
<th>Total</th>
<th>Ischemic</th>
<th>Cerebral hemorrhage</th>
<th>Subarachnoid hemorrhage</th>
<th>ICH</th>
<th>Other pathology</th>
<th>Unknown</th>
</tr>
</thead>
<tbody>
<tr>
<td>20-40 yrs</td>
<td>540</td>
<td>270</td>
<td>165</td>
<td>20</td>
<td>19</td>
<td>10</td>
<td>56</td>
</tr>
<tr>
<td>M</td>
<td>12</td>
<td>3</td>
<td>8</td>
<td>0</td>
<td>1</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>F</td>
<td>13</td>
<td>5</td>
<td>3</td>
<td>4</td>
<td>0</td>
<td>0</td>
<td>1</td>
</tr>
<tr>
<td>41-50 yrs</td>
<td>103</td>
<td>47</td>
<td>41</td>
<td>3</td>
<td>5</td>
<td>2</td>
<td>5</td>
</tr>
<tr>
<td>M</td>
<td>23</td>
<td>9</td>
<td>11</td>
<td>1</td>
<td>1</td>
<td>0</td>
<td>1</td>
</tr>
<tr>
<td>F</td>
<td>12</td>
<td>6</td>
<td>3</td>
<td>2</td>
<td>0</td>
<td>0</td>
<td>1</td>
</tr>
<tr>
<td>51-60 yrs</td>
<td>167</td>
<td>92</td>
<td>45</td>
<td>1</td>
<td>5</td>
<td>3</td>
<td>21</td>
</tr>
<tr>
<td>M</td>
<td>57</td>
<td>20</td>
<td>25</td>
<td>5</td>
<td>4</td>
<td>1</td>
<td>2</td>
</tr>
<tr>
<td>F</td>
<td>153</td>
<td>88</td>
<td>29</td>
<td>4</td>
<td>3</td>
<td>4</td>
<td>25</td>
</tr>
</tbody>
</table>

Data are number of patients. ICH, intracranial hemorrhage (type unspecified); M, male; F, female.
The age and sex distribution of the patients is presented in Table 2; 88.9% of the patients were >50 years old, and the sex ratio was 1.3:1. Male predominance was particularly evident among patients 41–61 years of age.

CT or autopsy was performed in 90.4% of the patients aged <61 years and in 83% of those aged 61–70 years. Whereas hemorrhagic stroke predominated in those younger than 61, older patients tended to have more ischemic lesions, even when bias due to lack of pathologic confirmation in comatose patients was taken into account (Table 2). While there was little difference in the incidence of ischemic disease due to sex, significantly more men than women had cerebral hemorrhage (p<0.01); on the other hand, subarachnoid hemorrhage occurred significantly more frequently among women (p<0.01).

On admission, 55.8% of the patients were fully alert, 22.0% were semicomatose, and 22.2% were comatose (Table 3). CT or autopsy was obtained in 68.3% of the comatose and 90.8% of the semicomatose patients; 91.4% of the alert patients had CT.

Among the 540 patients, 144 (26.7%) had a lacunar syndrome, 222 (41.1%) had complicated motor paresis, and 124 (23.0%) had no or only minimal weakness (Table 4). Among those with no or only minimal weakness, other neurologic deficits included dysphasia in nine, homonymous hemianopsia or visual neglect in 14, and limb ataxia in 19 patients.

By definition all 144 patients with a lacunar syndrome were alert, while only 70 (56.5%) of the 124 with no or only minimal weakness and 57 (25.7%) of the 222 with complicated motor paresis were alert. Among the 120 comatose patients, 66 (55.0%) had hemiplegia and five (4.2%) had no or only minimal weakness; the other 49 had neither spontaneous nor provoked motor response.

Among the 270 patients with ischemic lesions, lacunar infarcts were found in 30.0%, cortical infarcts in 25.6%, and brainstem or cerebellar infarcts in 5.6% (Table 5). Of the subcortical infarcts, 4% involved the striatocapsular and 5% the corona radiata regions. On admission, 93.8% of the patients with lacunes, 90.3% of those without CT abnormality, 84.6% of those with subcortical infarcts, and 80.0% of those with brainstem or cerebellar infarcts were mentally alert. In contrast, only 55.1% of the patients with cortical infarcts were alert on admission.

Complicated motor paresis is more likely to be the result of hemorrhage than ischemia. When due to ischemia, complicated motor paresis was found in 57.9% of the 95 patients with cortical or subcortical infarcts but in only 12.3% of those with lacunar infarcts and 18.1% of those without CT abnormality.
On the other hand, patients with no or only minimal weakness were more likely to have an ischemic lesion than a hemorrhage when subarachnoid hemorrhage had been excluded (Table 4).

Among the 465 patients with a confirmed diagnosis, intracranial hemorrhage was clearly more likely with decreasing degrees of alertness and with increasing degrees of motor deficit. Intracranial hemorrhage was confirmed in 92.8% of the 83 comatose patients so examined and in 96% of the 49 with no motor responses. Among the 540 patients overall, 30.5% had confirmed parenchymal intracerebral hemorrhage, another 3.7% had subarachnoid hemorrhage, and 3.5% had an unspecified type of intracranial hemorrhage (Table 4).

Among the 56 patients with unknown pathology and 19 patients with intracranial hemorrhage (type unspecified), 11 were semicomatose and hemiplegic while 38 were comatose (Table 3). Among the remaining 465 patients in whom the pathologic process of the stroke was known, 47.2% of the 108 who were semicomatose and hemiplegic had cerebral hemorrhage while 76.8% of the 82 comatose patients had cerebral hemorrhage. This suggests that of the 56 patients with an undetermined cause and the 19 patients with intracranial hemorrhage (type unspecified), 34 could have had cerebral hemorrhage. Inclusion of these probable cases would mean that cerebral hemorrhage accounted for 37% of all strokes.

Ten patients satisfied the clinical criteria for stroke but were found to harbor other pathology on investigation. In addition to one case each of meningioma, metastatic tumor, and probable incidental osteoma, there were seven subdural hematomas. However, no history or evidence of head trauma was otherwise present. Thus, the clinical criteria used to diagnose stroke in many epidemiology studies had a false-positive rate of 1.9%.

**Discussion**

Although our study is hospital-based, the clinical characteristics of the study population such as alertness and motor deficit are fairly similar to those reported in community-based studies from Europe and Israel. Therefore, it seems unlikely that our study is significantly distorted because of either excessive admission or nonadmission of more severe cases of stroke.

CT or autopsy documented the lesion in 86.1% of all patients and in >91% of the noncomatose patients. In addition, another 3.5% of the patients had CSF examination, which established the diagnosis to be intracranial hemorrhage of unspecified type. In comparison, among studies of Caucasians only the Pilot Stroke Data Bank and the Oxfordshire Community Stroke Project have achieved similar rates of confirmation of the clinical diagnosis by CT, angiography, surgery, or autopsy. Nevertheless, several differences emerge.

First, we show an increase in the incidence of both lacunar syndrome and lacunar infarction. Among our 540 stroke cases, 26.7% were considered on clinical grounds to be classic lacunar syndromes (Table 4), while 30% of the 270 ischemic cases were found to harbor lacunes after CT examination (Table 5). This compares with a clinical incidence of 3–14% and CT findings of 12.2% reported by the Harvard Cooperative Stroke Registry and the Pilot Stroke Data Bank. In addition, whereas striatocapsular territory infarction is uncommon among Caucasians based on a reported incidence of 0.69%, our series also shows an increase in large subcortical infarcts involving the striatocapsular (4%) and corona radiata (5.6%) regions.

Cerebral hemorrhage occurred more frequently among our stroke cases (30.6%) than in Caucasians. Two other studies with comparable diagnostic reliability report incidences of only 9–10.9%. Although other series had fewer confirmatory tests, experience in Rochester, Minnesota, and elsewhere has shown that the use of CT increases the number of cerebral hemorrhages diagnosed by only 1.7–24%. Thus, even though cerebral hemorrhage may have been underdiagnosed in many studies of Caucasian patients, the actual incidence would still not approach that seen in our study. This higher incidence of cerebral hemorrhage is seen not only in semicomatose and comatose patients, but also among patients who were alert on admission (16.9%). Correlation with mental state is seldom reported in the literature. However, in the Tilburg study, only 4.8% of fully conscious patients had intracranial hemorrhage.

Preferential admission of more severe stroke cases decreases the percentage of patients admitted with cerebral hemorrhage and normal alertness. In the Tilburg and Mayo clinic studies, 19–23% of patients with intracranial hemorrhage were fully conscious on admission. In our series, 30.9% of the
proven cases of cerebral hemorrhage were alert on admission. Thus, the increase in the incidence of cerebral hemorrhage cannot be an artifact caused by a disproportionate admission of more disabled patients.

Is this increase due to more cerebral hemorrhages or to fewer ischemic strokes? Elsewhere we have argued that there is probably an absolute increase in the number of cerebral hemorrhages in our population.\(^{23}\) Even if there were fewer ischemic strokes, the increased number of hemorrhages found among patients presenting with a lacunar syndrome would require a different explanation.

Hematoma has previously been considered an uncommon finding in the lacunar syndrome. No hematomas were seen in one study of 37 patients with a lacunar syndrome\(^{24}\) and only five were seen in 63 patients with pure motor hemiplegia.\(^{25,26}\) In our study, however, 12.5% of the 144 patients presenting with lacunar syndromes had cerebral hemorrhage, suggesting that the proportional increase in cerebral hemorrhage is due to an actual increase in the number and not to just a relative decrease in ischemic lesions.

Definite subarachnoid hemorrhage was present in 3.7% of the patients, and another 3.5% had an unspecified type of intracranial hemorrhage. This incidence of subarachnoid hemorrhage is not much different from the 5–13% found in other stroke studies.\(^{1–8}\)

Racial differences in the pattern of stroke have previously been reported to show similar increases in the incidence of hemorrhagic stroke among the Japanese\(^ {27}\) and blacks.\(^ {6}\) The intracranial vessels are also more heavily compromised by atherosclerosis in the Japanese and blacks.\(^ {28,29}\) Our study suggests that the Chinese may have either a decreased occurrence of larger cerebral artery disease or are similar to the Japanese and blacks in having more intracranial vascular disease. It would therefore be of paramount interest to determine in future studies whether stroke in other Chinese and Japanese communities is also characterized by predominantly small-vessel pathology that results in lacunar infarction and hemorrhage. We need to evaluate further whether risk factors for cerebrovascular disease vary in relative importance across communities and whether the increase in cerebral hemorrhage is directly or inversely proportional to large-vessel atherosclerosis.

Community surveys can provide much-needed information about the prevalence of cerebrovascular disease in different areas. However, our study shows that similar prevalence rates should not necessarily be interpreted as reflecting similar underlying pathologies. Careful hospital-based studies that complement community-based epidemiology surveys are necessary if the mix of various pathologic stroke types and their respective risk factors are to be determined for different populations. Clinicopathologic correlation for each community is mandatory if more in-depth analysis of epidemiologic data is to be made.

Descriptions of clinical characteristics, such as degree of alertness and extent of motor deficits, would furnish information about the reliability and completeness of hospital- and community-based studies and would provide insight into the pattern of stroke that exists in each community. Furthermore, in view of the increasing recognition of racial differences in stroke patterns, the applicability of stroke prevention trials and stroke risk identification across different populations requires reassessment.

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References


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