Stroke Subtypes Among Hispanics Living in Guayaquil, Ecuador
Results From the Luis Vernaza Hospital Stroke Registry

Oscar H. Del Brutto, MD; Aurelio Mosquera, MD; Xavier Sánchez, MD; José Santos, MD; Carlos A. Noboa, MD

Background and Purpose: Racial differences in stroke subtypes have been documented. Asians have a higher rate of cerebral hemorrhage than whites; however, there is little information about stroke subtypes among Hispanics. The purpose of this study was to determine the patterns of stroke subtypes in a population of Hispanics.

Methods: Five hundred consecutive patients with a first stroke were included. Patients were collected from hospital wards, the emergency department, and the outpatient clinic to ensure inclusion of patients with a wide range of stroke severity. Computed tomography was available in all cases. Patients with pure subarachnoid hemorrhage were excluded.

Results: There were 313 (62.6%) patients with an infarct and 187 (37.4%) with a hemorrhage. Hypertensive arteriopathy was the most common cause of both infarcts and hemorrhages. The carotid territory was involved in 70.6% of the 313 patients with infarcts, the vertebrobasilar territory in 17.9%, multiple territories in 6.7%, and a watershed area in 4.8%. Hemorrhages were most often lobar (36.4%), followed by putaminal (30.5%), brain stem (9.1%), cerebellar (8%), thalamic (8%), ventricular (5.3%), and caudate (2.7%).

Conclusions: This hospital-based stroke registry suggests that stroke in Hispanics has a pattern different from that in whites but similar to that in Asians. Cerebral hemorrhages occur three times more frequently in Hispanics than in whites. (Stroke. 1993;24:1833-1836.)

Key Words • cerebrovascular disorders • racial differences • South America

Stroke registries have shown racial differences in stroke subtypes. Chinese\(^\text{6,7}\) and other Asian people\(^\text{6,7}\) have a disproportionately higher rate of cerebral hemorrhage than whites.\(^\text{6,9}\) Stroke is a major cause of disability and death in Hispanics; however, there is little information about stroke subtypes in this ethnic group. Because this knowledge is important for preventive, diagnostic, and therapeutic purposes, we conducted this study to outline the patterns of stroke in a population of Hispanics.

This is a hospital-based registry, which may underestimate the frequency of very mild or rapidly lethal strokes. Nevertheless, hospital-based studies provide the best opportunity for precise categorization of stroke subtypes.\(^\text{3,7,10}\) Luis Vernaza Hospital is the major public hospital in Guayaquil, Ecuador, serving people of low socioeconomic status who belong to the Mestizo population (a racial admixture of Ecuadorian natives and Spaniards).

Subjects and Methods

This stroke registry included 500 consecutive Hispanic patients with a first stroke evaluated at Luis Vernaza Hospital. People from other ethnic groups were excluded. To ensure inclusion of patients with a wide range of stroke severity, the registry comprised patients admitted to hospital wards or the emergency department as well as patients attending the outpatient clinic of the Department of Neurology. All patients had a computed tomography (CT) of the head within 2 weeks of the onset of symptoms. Systematic evaluation also included a routine metabolic workup, a 12-lead electrocardiogram, and chest roentgenograms. Cerebral angiography was performed in 180 patients (106 of these patients had a cerebral infarct, and 74 had a hemorrhage). Two-dimensional echocardiography was performed in 120 patients (98 with infarcts and 22 with hemorrhages). Cerebrospinal fluid examination and other special tests were performed in selected patients.

Stroke was defined as a rapidly developing focal or global loss of cerebral function with symptoms leading to death or lasting more than 24 hours with no apparent cause other than vascular.\(^\text{11}\) Cerebral infarct was diagnosed when CT showed a hypodense area corresponding to the clinical manifestations or when the CT was normal in the setting of a clinically definite stroke. Cerebral hemorrhage was diagnosed when there was CT evidence of parenchymal or ventricular bleeding unrelated to trauma. Patients with pure subarachnoid hemorrhage or with a previous stroke were excluded. Risk factors were defined according to current criteria.\(^\text{12}\)

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Cerebral infarcts were classified according to their location: (1) carotid territory infarcts (cortical/subcortical and lacunar infarcts); (2) vertebrobasilar territory infarcts (brain stem/cerebellar and posterior cerebral artery infarcts); (3) watershed infarcts; and (4) multiple territory infarcts (CT evidence of cerebral infarcts in the territory of two or more of the major cerebral arteries). We used the CT-anatomic template of Damasio for identification of cerebral vascular territories. Thereafter, patients with infarct were entered in one of the following mechanistic stroke subtypes: (1) large-artery atherosclerotic disease (stenosis greater than 60% or occlusion of the corresponding extracranial artery or major intracranial artery); (2) hypertensive arteriolopathy (infarct in the territory of a deep perforating artery in a patient with arterial hypertension [blood pressure greater than 160/90 mm Hg at least twice], without evidence of large-artery disease); (3) embolism of cardiac origin; (4) other etiologies; and (5) infarct of undetermined cause. For patients with more than one possible etiology, we chose the most likely cause of the infarct (eg, if a patient had arterial hypertension and mitral stenosis, and a cortical infarct in the territory of the middle cerebral artery, we chose cardiac embolism as the cause of the infarct).

Cerebral hemorrhages were also classified according to their location in (1) putaminal, (2) lobar, (3) caudate, (4) thalamic, (5) primary ventricular, (6) cerebellar, and (7) brain stem. Possible etiologies of hemorrhages included (1) hypertension arteriolopathy, (2) saccular aneurysms (documented by angiography), (3) arteriovenous malformations (documented by angiography), (4) other etiologies, and (5) undetermined cause.

### Results

Our study included 296 men and 204 women whose ages ranged from 15 to 94 years (mean±SD age, 58.4±15.2 years). An infarct was present in 313 (62.6%) patients and a hemorrhage in 187 (37.4%). The infarct was seen on CT in 232 (74%) of the 313 patients with diagnosis of cerebral infarct. The mean±SD age for patients with infarct was 61.4±14.2 years (range, 20 to 80 years) and for patients with hemorrhage was 53.0±18.0 years (range, 15 to 94 years). Overall, 301 (60.2%) patients had arterial hypertension, 85 (17%) had diabetes mellitus, and 80 (16%) had cardiac disease (including 44 patients with a potential cardiac source of emboli and 36 patients with coronary artery disease). The carotid territory was involved in 221 (70.6%) of the 313 patients with infarcts, the vertebrobasilar territory in 56 (17.9%), multiple territories in 21 (6.7%), and a watershed area in 15 (4.8%). Ninety-nine (44.8%) of the 221 patients with involvement of the carotid territory had a cortical/subcortical infarct, and 122 (55.2%) had a lacunar infarct. Thirty-three (58.9%) of the vertebrobasilar territory infarcts were in the brain stem/cerebellum, and 23 (41.1%) were in the territory of the posterior cerebral artery. A probable cause of the infarct was determined in 221 (70.6%) of the 313 patients. Hypertensive arteriolopathy was the main cause, accounting for 135 cases of infarct (cerebral angiography was performed in 38 of these patients and was normal in each case). Embolism of cardiac origin was the presumed cause of the infarct in 44 patients; nonvalvular atrial fibrillation was the most common cause of embolic gen heart disease (24 patients), followed in order of frequency by rheumatic mitral stenosis (12 patients), left ventricular akinesis (3 patients), non-rheumatic valvulopathy (3 patients), infective endocarditis (1 patient), and cardiac myxoma (1 patient). Atherosclerotic stenosis greater than 60% or occlusion of a large artery was angiographically documented in 23 patients; of these, 12 had involvement of the extracranial carotid artery, 6 of the vertebral arteries, and 5 of the middle cerebral artery. Other etiologies of cerebral infarct were found in 19 patients, including 6 with tuberculous meningitis, 4 with arteriovenous malformations, 2 with migraine, 1 with cysicercotic angiitis, and one with Takayasu's disease. Finally, the etiology of the infarct could not be determined in 92 (29.4%) patients; 27 of these patients had arterial hypertension, but they were not included under the hypertensive arteriolopathy category because they had large cerebral infarcts (cerebral angiography was performed in 30 of the 92 patients with infarct of unknown etiology). Correlation between the etiology and the location of the infarct is shown in Table 1.

Lobar hemorrhages were found in 68 (36.4%) of the 187 patients with cerebral hemorrhage. Lobar intracerebral hematomas were predominantly located in the temporal lobe in 20 patients, the parietal lobe in 18, the frontal lobe in 15, and the occipital lobe in 8; the

### Table 1. Correlation Between Etiology and Location in 313 Patients With Cerebral Infarct

<table>
<thead>
<tr>
<th>Etiology</th>
<th>Cortical/Subcortical</th>
<th>Lacunar</th>
<th>Brain Stem/Cerebellar</th>
<th>PCA</th>
<th>Watershed</th>
<th>Multiple</th>
</tr>
</thead>
<tbody>
<tr>
<td>Large-artery atherosclerosis*</td>
<td>9</td>
<td>3</td>
<td>3</td>
<td>3</td>
<td>5</td>
<td>...</td>
</tr>
<tr>
<td>(n=23)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hypertensive arteriolopathy</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>20</td>
</tr>
<tr>
<td>(n=135)</td>
<td></td>
<td>85</td>
<td>20</td>
<td>10</td>
<td>...</td>
<td>20</td>
</tr>
<tr>
<td>Cardiogenic embolism†</td>
<td>32</td>
<td>6</td>
<td>2</td>
<td>3</td>
<td>1</td>
<td>...</td>
</tr>
<tr>
<td>(n=44)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Other causes</td>
<td>8</td>
<td>9</td>
<td>...</td>
<td>2</td>
<td>...</td>
<td></td>
</tr>
<tr>
<td>(n=19)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Not determined</td>
<td>50</td>
<td>19</td>
<td>8</td>
<td>5</td>
<td>9</td>
<td>1</td>
</tr>
</tbody>
</table>

PCA indicates posterior cerebral artery.

*Eleven and nine of these patients also had arterial hypertension.

**Etiology**
- Large-artery atherosclerosis* (n=23)
- Hypertensive arteriolopathy (n=135)
- Cardiogenic embolism† (n=44)
- Other causes (n=19)
- Not determined (n=92)

**Cortical/Subcortical**:
- Large-artery atherosclerosis: 9
- Hypertensive arteriolopathy: 32
- Cardiogenic embolism: 32
- Other causes: 8
- Not determined: 50

**Lacunar**:
- Large-artery atherosclerosis: 3
- Hypertensive arteriolopathy: 6
- Cardiogenic embolism: 6
- Other causes: 9
- Not determined: 19

**Brain Stem/Cerebellar**:
- Large-artery atherosclerosis: 3
- Hypertensive arteriolopathy: 2
- Cardiogenic embolism: 2
- Other causes: 2
- Not determined: 8

**PCA**:
- Large-artery atherosclerosis: 3
- Hypertensive arteriolopathy: 3
- Cardiogenic embolism: 3
- Other causes: 2
- Not determined: 5

**Watershed**:
- Large-artery atherosclerosis: 5
- Hypertensive arteriolopathy: 1
- Cardiogenic embolism: 1
- Other causes: ... 2
- Not determined: 9

**Multiple**:
- Large-artery atherosclerosis: ... 5
- Hypertensive arteriolopathy: 20
- Cardiogenic embolism: ... 20
- Other causes: ... ... 20
- Not determined: 1

**Locations**:
- Large-artery atherosclerosis: ... 20
- Hypertensive arteriolopathy: ... 20
- Cardiogenic embolism: ... 20
- Other causes: ... ... 20
- Not determined: 1
remaining 7 patients had multiple lobar hematomas. Putaminal hemorrhages were found in 57 (30.5%) patients, brain stem hemorrhages in 17 (9.1%), cerebellar hemorrhages in 15 (8%), thalamic hemorrhages in 15 (8%), primary ventricular hemorrhages in 10 (5.3%), and caudate hemorrhages in 5 (2.7%). The etiology of the hemorrhage was determined in 151 (80.7%) patients. Hypertensive arteriolopathy was the most common cause, accounting for 119 cases. Rupture of a saccular aneurysm or an arteriovenous malformation was the cause of the hemorrhage in 24 patients. Other causes of hemorrhage included brain tumor in 2 patients and idiopathic thrombocytopenic purpura, infective endocarditis, anticoagulant therapy, acute alcohol intoxication, venous thrombosis, and eclampsia in 1 patient each. The etiology of the hemorrhage could not be determined in 36 (19.3%) patients (20 of these 36 patients had a normal cerebral angiography). Table 2 correlates etiology with location of the hemorrhage.

**Discussion**

This hospital-based stroke registry shows that the pattern of stroke in Hispanics is different from that reported in other ethnic groups. The prevalence of cerebral hemorrhage is two to three times higher in Hispanics than in whites but is similar to that observed in Chinese, in whom at least 30% of all strokes are due to hemorrhage. To confirm this assertion we have compared our data with similar hospital-based stroke registries including people from other ethnic groups (Table 3). All of these studies may suffer from a bias of selection, but, as noted by the authors, they provide useful information on patients with a first stroke who are evaluated in a hospital.

As previously noted, few studies have evaluated the subtypes of stroke in a population of Hispanics, and the need for more information on the subject was recently urged in a statement of the American Heart Association. Thereafter, Sacco et al compared stroke subtypes in three ethnic groups (whites, blacks, and Hispanics) living in the same geographic area (northern Manhattan). They found that 11% of Hispanics and 7% of whites had intracranial hemorrhages, a figure that suggested that hemorrhagic strokes may be more common in Hispanics than in whites. It must be noted, however, that people living in the same geographic area usually share environmental risk factors; therefore, the actual prevalence of stroke subtypes in a given population should be better evaluated in the natural geographic and socioeconomic habitat. To our knowledge, such a study has not yet been reported.

Mechanisms by which intracerebral hemorrhages are more common in certain ethnic groups are not totally understood. Shi et al recently reviewed the subject and hypothesized that genetic influences, environmental factors, and dietary habits may explain such racial

**Table 2. Correlation Between Etiology and Location in 187 Patients With Cerebral Hemorrhage**

<table>
<thead>
<tr>
<th>Etiology</th>
<th>Putaminal (n=67)</th>
<th>Lobar (n=68)</th>
<th>Caudate (n=5)</th>
<th>Thalamic (n=15)</th>
<th>Ventricular (n=10)</th>
<th>Cerebellar (n=15)</th>
<th>Brain Stem (n=17)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hypertensive arteriolopathy (n=119)</td>
<td>55</td>
<td>19</td>
<td>2</td>
<td>14</td>
<td>3</td>
<td>13</td>
<td>13</td>
</tr>
<tr>
<td>Saccular aneurysm (n=13)</td>
<td>...</td>
<td>12</td>
<td>...</td>
<td>...</td>
<td>1</td>
<td>...</td>
<td>...</td>
</tr>
<tr>
<td>Arteriovenous malformations (n=11)</td>
<td>...</td>
<td>5</td>
<td>2</td>
<td>...</td>
<td>1</td>
<td>2</td>
<td>1</td>
</tr>
<tr>
<td>Other causes (n=8)</td>
<td>...</td>
<td>6</td>
<td>...</td>
<td>1</td>
<td>...</td>
<td>1</td>
<td>...</td>
</tr>
<tr>
<td>Not determined (n=36)</td>
<td>2</td>
<td>26</td>
<td>1</td>
<td>1</td>
<td>4</td>
<td>...</td>
<td>2</td>
</tr>
</tbody>
</table>

**Table 3. Proportion of Stroke Subtypes in Hospital-Based Stroke Registries Including People From Different Ethnic Groups**

<table>
<thead>
<tr>
<th>Stroke Registry</th>
<th>Race</th>
<th>Cerebral Infarct</th>
<th>Cerebral Hemorrhage</th>
<th>Unknown</th>
</tr>
</thead>
<tbody>
<tr>
<td>Oxfordshire6 (n=642)*</td>
<td>White</td>
<td>84.9%</td>
<td>10.3%</td>
<td>4.8%</td>
</tr>
<tr>
<td>Lausanne7 (n=1000)</td>
<td>White</td>
<td>89.1%</td>
<td>10.9%</td>
<td>...</td>
</tr>
<tr>
<td>Lehigh Valley8 (n=2675)</td>
<td>White</td>
<td>89.9%</td>
<td>8.9%</td>
<td>1.3%</td>
</tr>
<tr>
<td>Northern Manhattan16 (n=1008)*</td>
<td>Black</td>
<td>84.5%</td>
<td>13.3%</td>
<td>2.2%</td>
</tr>
<tr>
<td>Queen Mary Hospital9 (n=501)*</td>
<td>White</td>
<td>93.9%</td>
<td>7.6%</td>
<td>...</td>
</tr>
<tr>
<td>Shatin9 (n=777)</td>
<td>Asian</td>
<td>68.4%</td>
<td>27.1%</td>
<td>4.5%</td>
</tr>
<tr>
<td>Akta4 (n=1862)*</td>
<td>Asian</td>
<td>64.5%</td>
<td>35.5%</td>
<td>...</td>
</tr>
<tr>
<td>Luis Vernaza Hospital (n=500)</td>
<td>Hispanic</td>
<td>62.6%</td>
<td>37.4%</td>
<td>...</td>
</tr>
</tbody>
</table>

*Adjusted rates after exclusion of patients with subarachnoid hemorrhage.
differences in stroke subtypes. Some of the environmental risk factors that may play a role in the increased prevalence of cerebral hemorrhage in Asians (e.g., cold weather) do not apply to people living in Guayaquil, where the annual temperature is 20 to 30°C without marked seasonal variations because of its proximity to the equator. Genetic predisposition to certain intracranial arteriopathies that are more common in Asians (e.g., moyamoya disease) do not seem to occur in Hispanics, in whom these conditions are extremely rare. Nevertheless, it must be remembered that a possible common ethnic origin between Orientals and native Americans may explain the increased prevalence of cerebral hemorrhage in both Asians and Hispanics. Finally, a risk factor that may be shared by both Asians and Hispanics is diet. The Chinese and the Ecuadorian diets are generally higher in carbohydrates and lower in fats than the diet in whites. Whether dietary habits per se could explain the increased rate of cerebral hemorrhages in Asians and Hispanics remains to be determined.

Apart from showing the high prevalence of cerebral hemorrhage in Hispanics, our series showed that the pattern of cerebral hemorrhage in Hispanics is different from that reported in whites, in whom putaminal hemorrhages usually account for the majority of hemorrhagic strokes. In contrast, lobar hemorrhages predominated in our series. This high prevalence of lobar hemorrhages was not related to an increased number of patients with sacular aneurysms or arteriovenous malformations, since most of the patients with lobar hemorrhages had a normal cerebral angiography. It is possible, however, that small angiomata or undiagnosed cerebral amyloidosis accounted for some of these cases. Another possibility is that chronic use of aspirin, nonsteroidal anti-inflammatory drugs, or phenylpropanolamine may have predisposed people to the development of lobar hemorrhages in our population. We did not systematically inquire regarding over-the-counter drug ingestion in our patients; however, this practice is very common in Ecuador (as well as in other developing countries) where people are self-medicating for common diseases. Finally, chronic alcohol consumption may be responsible for some of the cases of lobar hemorrhage in our series. There is increasing evidence that chronic drinkers have a greater chance of developing a cerebral hemorrhage than nondrinkers. Unfortunately, we have not determined the prevalence of alcohol consumption among our patients, but results from this study will encourage further research on this subject.

Stroke registries in Asians have not classified cerebral hemorrhages according to their location. If the findings and hypothesis presented here could be confirmed in Asians and in other populations of Hispanics, it may be concluded that the increased prevalence of cerebral hemorrhage in these ethnic groups is related to an increased rate of lobar hemorrhages, which, in turn, could be related to chronic drug or alcohol ingestion or to dietary habits.

References
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