One-Year Outcome After Cerebral Infarction in Whites, Blacks, and Hispanics

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Little is known about outcome after cerebral infarction for different ethnic groups. Of 590 stroke patients hospitalized from 1983 to 1986 at the Neurological Institute, cerebral infarction over age 39 years occurred in 135 whites, 177 blacks, and 82 Hispanics. Outcome after cerebral infarction differed by ethnicity. The 1-month mortality rate was similar in whites and blacks and least in Hispanics. Whites had a slightly greater risk of recurrent stroke or death than blacks or Hispanics until 6 months after infarction, when their risk stabilized, while the risk in blacks and Hispanics continued to rise for the entire year of follow-up. By 1 year, the rate of recurrent stroke or death was 34.8±4.2% in whites, 31.1±3.6% in blacks, and 21.4±4.8% in Hispanics (p<0.04). Differences were found in the distribution of various stroke risk factors in the three ethnic groups. A Cox proportional hazards model demonstrated that the ethnic differences in stroke risk factors and infarct subtype were responsible for the ethnic differences in outcome. An abnormal first electrocardiogram was a risk factor for stroke recurrence or death in all three ethnic groups, while a nonlacunar infarct subtype and a history of diabetes were significant only in Hispanics. Understanding the associations of stroke determinants with ethnicity may lead to more focused secondary prevention of recurrent stroke. (Stroke 1991;22:305–311)

Blacks accounted for 12% of the US population in 1980 and are projected to show a 15.8% growth in number in the decade 1980–1990. The US Hispanic population has increased 34% since the 1980 census, growing five times faster than the white population.1 This population growth has led to a greater number of blacks and Hispanics at risk for stroke or recurrent stroke.

Current studies indicate that stroke recurrence rates after cerebral infarction vary from 4% to 12% per year, but there has not been universal agreement on the predictors of outcome.2–8 Most cohorts were largely white; therefore, no study to date has separately characterized stroke recurrence and survival rates in blacks and Hispanics. When ethnicity has been studied, it has largely been restricted to whites versus nonwhites. Nonwhites often consist primarily of blacks; Hispanics have not been identified independently. Since most epidemiologic studies have been conducted in white populations and since there is a growing number of blacks and Hispanics in the United States with stroke, there is a need for better understanding of ethnic differences in outcome after stroke.

Subjects and Methods

From 1983 to 1986, patients with stroke admitted to the Neurological Institute of Columbia–Presbyterian Medical Center and prospectively evaluated by the Stroke Service were followed for 2 years as part of the Stroke Data Bank.9 Each patient was personally examined ≤1 week after stroke onset by one of the neurologists on the Stroke Service. A large core of information was collected on each patient concerning the details of medical, neurologic, and social history, general and neurologic examinations, laboratory studies, and final diagnosis, with special collection procedures for complications, stroke evolution, stroke recurrence, and death. In the Stroke Data Bank, race was defined as white, black, or other. For this analysis, ethnicity was defined subsequently by skin pigmentation and surname as white (non-Hispanic), black (non-Hispanic), Hispanic, or other.

At the time of hospital discharge, a diagnosis was determined taking into account all the available data regarding the neurologic and medical history, neurologic symptoms and signs, head computed tomography...
Recurrence stroke was defined as a cerebrovascular event subsequent to the initial stroke that clearly resulted in a new deficit and was in a different stroke subtype. Follow-up visits with the nurse and stroke subtype. Follow-up permitted the determination of a variety of outcomes, including stroke recurrence and death. Recurrent stroke was defined as a cerebrovascular event subsequent to the initial stroke that clearly resulted in a new deficit and was in a different anatomic or vascular territory or was of a different stroke subtype. Follow-up visits with the nurse and investigator were scheduled at 6 months, 1 year, and 2 years after the stroke. Every effort was made to have in-person follow-up. However, a telephone interview was arranged for those patients unable to come to the medical center in person.

Event rates were calculated for death and stroke recurrence. Actuarial life table analyses were used to determine the cumulative probability of stroke recurrence or death for all infarcts and for individual ethnic groups. Losses to follow-up were censored in the life table analyses at the date of last contact. Only 6% (23 of 394) of the patients were lost to follow-up at 1 year. Statistical analysis included the calculation of the cumulative probability of stroke recurrence or death and its standard error for individual ethnic groups. The Wilcoxon test was used to judge the similarity of the rates for each ethnic group. Event rates were stratified by age at stroke, but because of small sample sizes, this was limited to two age groups (<65 and ≥65 years).

To ascertain the effect of potential confounders, the frequency of patients admitted from the northern Manhattan community (defined by the five ZIP codes in the area surrounding Columbia-Presbyterian Medical Center) and the frequency of patients of a given socioeconomic status (defined by level of education) were compared in the three ethnic groups. The frequencies of various stroke risk factors and clinical findings were determined in each ethnic group. We calculated distributions of the variables age and sex; stroke diagnostic subtype; initial neurologic syndrome, cerebral site, systolic and diastolic blood pressures, and blood sugar concentration; medical history of hypertension, myocardial infarction, valvular heart disease, atrial fibrillation, other arrhythmias, angina, congestive heart failure, diabetes, and previous stroke or transient ischemic attack (TIA); stroke severity measured by total weakness scores; and CT findings. Treatment differences were difficult to assess since the Stroke Data Bank was not designed to determine treatment efficacy; therefore, only the frequency of use of heparin in the hospital or warfarin upon discharge was compared. The variables were examined for significantly different distributions among the three ethnic groups using 2 × 3 χ² contingency tables with 2 degrees of freedom. The probability values were reported, and p<0.05 was considered significant. Variables found to differ by univariate analyses among the three ethnic groups at p<0.05 were selected for regression analyses.

Recurrent stroke or death ≤1 year after cerebral infarction was analyzed using the Cox proportional hazards model for the entire cohort and separately for each ethnic group. The Cox model was judged appropriate based on the linearity of the log-log survival distribution curves and the lack of significance in the model of a categorical variable for the time to recurrent stroke or death.

Modeling was done using the stroke risk factors as independent variables and the time to stroke recurrence or death as the dependent variable. The linear logistic regression technique was used to identify risk factors simultaneously predictive of outcome. Using this method, the conditional probability of stroke recurrence or death (given the values of X₁, X₂, . . .) is expressed as a function of predictive factors 1/[1 + e⁻^(B₀+B₁X₁+B₂X₂+...)], where B₀, B₁, . . . are parameters to be estimated by maximum likelihood estima-
tion from the data and the $X_i$, $X_2$, ... is the set of variables that passed the univariate screen for a particular individual. There was no assumption of multivariate normality for these covariates. Odds ratios and confidence intervals were calculated from the $B$ coefficients and their standard errors. 11

A model was constructed using the entire cohort, with ethnicity as an independent variable. Estimated odds ratios and their 95% confidence intervals were calculated for a categorical ethnicity variable after adjusting for socioeconomic status and other stroke risk factors. The significant variable were also entered in three separate models for each ethnic group, and odds ratios for the risk factors were calculated.

Results

From 1983 to 1986, 590 patients with stroke were examined and entered into the Stroke Data Bank at the Neurological Institute. Of 394 patients with cerebral infarction over age 39 years, there were 135 whites, 177 blacks, and 82 Hispanics (Table 1). The mean age at stroke did not differ significantly among the three ethnic groups. Blacks and Hispanics were more likely to live in the local community and to have less than a high school education.

Prognosis after cerebral infarction differed by ethnicity (Table 2). The life table 1-month mortality rate was greatest in whites, similar to that in whites in blacks, and least in Hispanics. By 12 months, 25.0±3.8% of the whites, 21.3±3.2% of the blacks, and only 13.6±4.0% of the Hispanics were dead. Among those who died, the cause of death was directly or indirectly related to stroke in 61.8% of the whites, 55.6% of the blacks, and 30.0% of the Hispanics.

By 1 year, stroke had recurred in 17.9±3.5% of the whites, 16.5±2.9% of the blacks, and 12.3±3.9% of the Hispanics (Table 2). When the end point of death was combined with that of stroke recurrence, the 1-year rates differed significantly among ethnic groups (Table 2, $p=0.04$). Within each ethnic group, the 1-year outcome rates did not differ by age group.

Inspection of the cumulative risk plots (Figures 1 and 2) shows that the risk for all three ethnic groups was highest during the first month after cerebral infarction. Thereafter, whites had a greater risk than blacks or Hispanics until 6 months, when their risk stabilized, while the risk for blacks and Hispanics continued to rise for the entire year of follow-up. Therefore, there was a decrease in the risk difference between whites and blacks and between whites and Hispanics at 1 year versus that at 6 months.

Differences were found in the distribution of various stroke risk factors in the three ethnic groups (Table 3). Hypertension was slightly more prevalent in blacks and Hispanics. History of myocardial infarction, atrial fibrillation, other arrhythmias, angina, congestive heart failure, diabetes, and prior stroke and TIA did not differ among the three ethnic groups. Combined cardiac disease, defined as at least one of the listed cardiac conditions, was greatest in whites and least in Hispanics. Initial diastolic and systolic blood pressures were higher in blacks and Hispanics, while initial blood glucose concentration and hematocrit showed no differences. Whites had the greatest frequency of a symptomatic lesion on cerebral angiography (when performed). The first electrocardiogram (ECG) and echocardiogram were least likely to be abnormal in Hispanics.

There were differences in the distribution of initial infarct subtypes in the three ethnic groups: whites more frequently had ATH than blacks and Hispanics, Hispanics more frequently had LAC and less frequently had EMB, and blacks had the highest frequency of IUC. The use of intravenous heparin during hospitalization was less frequent in blacks, but the use of warfarin upon discharge did not differ significantly among the three ethnic groups (Table 3).
Using the entire cohort, a Cox proportional hazards model demonstrated that the ethnic differences in stroke risk factors and infarct subtype were responsible for the ethnic differences in outcome. Age at initial stroke was not a significant predictor of outcome in the overall model. Education, a possible marker of socioeconomic status, was not an independent predictor of outcome and accounted for a minimal amount of the ethnic differences. After adjusting for education and other stroke risk factors, however, the magnitude of the odds ratio for the Hispanic versus the non-Hispanic group decreased and was no longer independently significant (Table 4).

When each ethnic group was analyzed separately, there was evidence of a varying effect of some of the stroke risk factors (Table 5). Nonlacunar infarction was a strong independent predictor of outcome, particularly in Hispanics, with a nearly sevenfold increase in the odds of stroke recurrence or death compared with a twofold increase in blacks and whites. An abnormal first ECG was associated with a twofold-to-fourfold increase in the odds of stroke recurrence or death in whites, blacks, and Hispanics. History of diabetes had the greatest effect in Hispanics.

Discussion
The determinants of long-term outcome after cerebral infarction for blacks and Hispanics are poorly understood. In one study of patients with anterior-circulation infarcts followed up for 36 months, 45% of blacks survived compared with 52% of whites. In the Community Hospital-based Stroke Program, survival at 1 year was similar in whites and nonwhites, and nonwhite survivors at 1 year were less likely to return to work; however, nonwhites accounted for only 16% of the entire cohort. Most prospective epidemiologic studies have been conducted in predominantly white cohorts, limited by small numbers or relying on death certificates for diagnosis.

The hospital-based cohort at Columbia-Presbyterian Medical Center provides an unusual opportunity to explore ethnic differences in stroke epidemiology. The hospital serves as a primary health care provider for the ethnically mixed population of northern Man-

![Figure 1. Life table cumulative risk of stroke recurrence after cerebral infarction in whites, blacks, and Hispanics within 1 year.](image1)

![Figure 2. Life table cumulative risk of stroke recurrence or death after cerebral infarction in whites, blacks, and Hispanics within 1 year.](image2)
TABLE 3. Ethnic Distribution of Cerebral Infarction Characteristics

<table>
<thead>
<tr>
<th>Risk factors (risk per 100)</th>
<th>White (n=135)</th>
<th>Black (n=177)</th>
<th>Hispanic (n=82)</th>
<th>Total (N=394)</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hypertension</td>
<td>59.9</td>
<td>70.4</td>
<td>73.2</td>
<td>67.4</td>
<td>0.07</td>
</tr>
<tr>
<td>Cardiac comorbidity</td>
<td>38.5</td>
<td>34.5</td>
<td>22.0</td>
<td>33.3</td>
<td>0.04</td>
</tr>
<tr>
<td>Myocardial infarction</td>
<td>20.5</td>
<td>14.7</td>
<td>11.1</td>
<td>15.9</td>
<td>NS</td>
</tr>
<tr>
<td>Atrial fibrillation</td>
<td>10.5</td>
<td>8.7</td>
<td>3.8</td>
<td>8.3</td>
<td>NS</td>
</tr>
<tr>
<td>Other arrhythmias</td>
<td>7.5</td>
<td>5.9</td>
<td>3.8</td>
<td>6.0</td>
<td>NS</td>
</tr>
<tr>
<td>Angina</td>
<td>18.7</td>
<td>14.9</td>
<td>8.8</td>
<td>15.3</td>
<td>NS</td>
</tr>
<tr>
<td>Congestive heart failure</td>
<td>11.2</td>
<td>12.1</td>
<td>3.9</td>
<td>10.4</td>
<td>NS</td>
</tr>
<tr>
<td>Diabetes</td>
<td>20.7</td>
<td>26.4</td>
<td>29.6</td>
<td>25.5</td>
<td>NS</td>
</tr>
<tr>
<td>Prior transient ischemic attack</td>
<td>22.3</td>
<td>15.2</td>
<td>22.8</td>
<td>19.3</td>
<td>NS</td>
</tr>
<tr>
<td>Prior stroke</td>
<td>26.6</td>
<td>26.8</td>
<td>25.9</td>
<td>26.6</td>
<td>NS</td>
</tr>
</tbody>
</table>

Laboratory and diagnostics (%)

| Initial systolic blood pressure >160 mm Hg | 35.8 | 51.7 | 48.8 | 45.7 | 0.02 |
| Initial diastolic blood pressure >100 mm Hg | 17.9 | 38.6 | 24.4 | 28.6 | 0.001 |
| Initial glucose concentration >140 mg/dl | 29.3 | 32.0 | 30.4 | 30.7 | NS    |
| Initial hematocrit >45% | 31.0 | 26.4 | 27.1 | 28.1 | NS    |
| Initial first electrocardiogram abnormal | 53.0 | 44.6 | 39.5 | 46.4 | NS    |

If performed:

| Abnormal angiogram | 80.0 | 56.0 | 40.6 | 61.4 | 0.001 |
| Abnormal carotid Doppler | 53.9 | 37.5 | 34.5 | 44.0 | NS    |
| Abnormal echocardiogram | 39.3 | 49.5 | 26.7 | 41.7 | 0.03  |
| Abnormal first electrocardiogram | 62.0 | 54.0 | 33.8 | 52.8 | <0.001 |

Initial infarct subtype (%)

| Atherosclerotic | 30.4 | 13.6 | 13.4 | 19.3 | 0.001 |
| Lacunar         | 24.4 | 28.8 | 52.4 | 32.2 | 0.001 |
| Cardiembolic    | 17.8 | 14.7 | 4.9  | 13.7 | 0.02  |
| Undetermined cause | 27.4 | 42.9 | 29.3 | 34.8 | 0.009 |

Anticoagulant use (%)

| Heparin in hospital | 65.4 | 46.0 | 58.5 | 55.2 | 0.014 |
| Warfarin at discharge | 20.0 | 16.7 | 12.3 | 16.9 | NS    |

NS, p<0.05 by x² with 2 degrees of freedom.

TABLE 4. Adjusted Non-Hispanic Versus Hispanic Odds Ratio of Stroke Recurrence or Death Calculated by Cox Proportional Hazards Models

<table>
<thead>
<tr>
<th>Model</th>
<th>Odds ratio</th>
<th>95% confidence interval</th>
</tr>
</thead>
<tbody>
<tr>
<td>Unadjusted</td>
<td>1.75</td>
<td>1.03–2.97</td>
</tr>
<tr>
<td>Adjusted for education</td>
<td>1.63</td>
<td>0.90–2.95</td>
</tr>
<tr>
<td>Adjusted for education and stroke risk factors</td>
<td>1.09</td>
<td>0.56–2.11</td>
</tr>
</tbody>
</table>

Stroke risk factors included history of abnormal first electrocardiogram, nonlacunar stroke subtype, and diabetes.

Some ethnic differences in 1-year outcome after cerebral infarction occurred in our cohort. Whites had the greatest 1-month mortality rate, followed by blacks and Hispanics. For death, stroke recurrence, or both, whites still had the greatest rates; intermediate rates were found in blacks, and the lowest rates were found in Hispanics. However, even though whites had the greatest rates, the differences between the rates decreased by the end of the first year. This may indicate a greater risk in blacks and Hispanics.

TABLE 5. Estimated Odds Ratio of Stroke Recurrence or Death Calculated by Separate Cox Proportional Hazards Models for Each Ethnic Group

<table>
<thead>
<tr>
<th>Risk factors</th>
<th>White</th>
<th>Black</th>
<th>Hispanic</th>
</tr>
</thead>
<tbody>
<tr>
<td>Nonlacunar infarction</td>
<td>2.05</td>
<td>1.59</td>
<td>6.91*</td>
</tr>
<tr>
<td>Abnormal first electrocardiogram</td>
<td>2.13*</td>
<td>1.80*</td>
<td>3.64*</td>
</tr>
<tr>
<td>History of diabetes</td>
<td>1.41</td>
<td>1.17</td>
<td>3.84*</td>
</tr>
</tbody>
</table>

*p<0.05 different from 1.0 by x².
later after infarction. Further follow-up is needed to determine if this trend continues.

Prior studies have demonstrated that blacks have a greater incidence of stroke than whites of comparable age, sex, and residence. Many of these observations stem mainly from mortality data. For all stroke, nonwhites generally have a higher mortality rate than whites. Some studies suggest that this difference may be due to the higher prevalence of hypertension in blacks. It is difficult to find separate information on rates of cerebral infarction since most larger demographic studies combine cerebral infarction with cerebral hemorrhage. Some of the reported increased mortality rates for blacks may stem from the higher frequency of intracerebral hemorrhage, which uniformly has a higher mortality than cerebral infarction.

Ethnic differences in vascular pathology and stroke risk factors may account for differences in stroke incidence and mortality. Some investigators have suggested that race helps to predict the location of the vascular lesion based on the finding of a greater frequency of intracranial atherosclerosis in blacks. Other researchers have found that hypertension and diabetes are more frequent in nonwhite women with stroke while TIA's are less frequently diagnosed in nonwhites. Specific studies of Mexican-Americans have noted a greater prevalence of non-insulin dependent diabetes and higher concentrations of serum cholesterol and triglyceride. In clinical trials, control of hypertension in blacks decreased the cerebrovascular disease death rates below that of blacks in the general population. However, information is lacking on the effect of different risk factors in individual ethnic groups on the risk of recurrent stroke.

Our model demonstrates that ethnicity was not an independent predictor of outcome. Differences in the distributions of recognized stroke risk factors and stroke diagnostic subtypes explained the differences in outcome. Therefore, ethnicity was a marker of the magnitude of various stroke risk factors. When each ethnic group was analyzed separately, nonlacunar infarction was a stronger predictor of poor outcome in Hispanics. An abnormal first ECG increased the odds of death or stroke recurrence in all three ethnic groups, while a history of diabetes was significant only in Hispanics. The varying effect of the individual risk factors in each ethnic group suggests that the presence of a risk factor needs to be treated more aggressively depending on the ethnicity of the patient. More aggressive management of Hispanics with nonlacunar infarction or diabetes may be needed. Further studies are necessary to identify other stroke risk factors that help account for ethnic differences. This may direct the emphasis for secondary prevention in different ethnic groups.

Some cautions must be emphasized in interpreting the results of this and other hospital-based studies. If the frequency of nonhospitalized stroke differs by ethnic group, the results of a hospital-based study may be misleading. Referral bias may also affect analyses of outcome. However, when an indicator of community of origin was entered into our model, it did not significantly predict outcome. Differential follow-up can also affect the results, but this effect is less serious when the period of observation is limited to 1 year. Stroke subtype was not determined blinded to race, but the strict diagnostic definitions and the existence of the IUC category helped ensure the homogeneity of the stroke subtypes. We hope the estimates provided by this study will be corroborated by future population-based studies.

Recurrent stroke is frequent and is responsible for major stroke morbidity and mortality. The growing number of blacks and Hispanics in the US population has alerted the National Institute of Neurological Disorders and Stroke to call for more focused epidemiologic investigations on the risk of recurrent stroke in these ethnic groups. More extensive follow-up may show additional differences in the long-term outcome after stroke for different ethnic groups. Better understanding of the association of stroke risk factors with ethnicity in the determination of stroke outcome will help in the design of clinical trials and the development of more selective secondary prevention strategies.

References

KEY WORDS • cerebrovascular disorders • epidemiology • racial differences
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