Extracranial Carotid Atherosclerosis in Black and White Patients With Transient Ischemic Attacks

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To evaluate the association between extracranial carotid atherosclerosis, race, and transient ischemic attack, we carried out a retrospective hospital chart review and quantified the extent of noninvasively determined extracranial carotid atherosclerosis in 25 black patients >45 years old with transient ischemic attacks. Two sex- and age-matched white patients with transient ischemic attacks were similarly studied for each black patient. Extent of extracranial carotid atherosclerosis (expressed as B-mode score) was similar for blacks and whites. B-mode score was only slightly less in patients with posterior- than in those with anterior-circulation transient ischemic attacks. Fifty-six patients (35 white, 21 black) had unilateral anterior-circulation transient ischemic attacks. Of the 32 patients with more extensive extracranial carotid atherosclerosis ipsilateral to the affected hemisphere, 23 (66% of 35) were white; only nine (43% of 21) were black. In the 35 white patients, the extent of disease in the ipsilateral carotid artery was significantly greater ($p<0.03$) than that in the contralateral carotid artery. When B-mode scores in the left and right carotid arteries were combined for the subgroup of patients with unilateral anterior-circulation transient ischemic attacks, blacks had slightly more atherosclerosis in the extracranial arteries than whites. (Stroke 1989;20:1133-1137)

The information relating transient ischemic attacks (TIAs) in black and white patients to atherosclerosis in the extracranial arteries has been obtained using angiography rather than B-mode ultrasound, and these studies suggest more severe disease ipsilateral to the hemisphere responsible for the symptoms and that blacks are less likely to have ipsilateral disease than whites. These data have been extrapolated to imply that blacks also have less extracranial carotid artery disease overall than whites. This conclusion contrasts sharply with older results from the International Atherosclerosis Project, which demonstrated that blacks have as much as or more extracranial atherosclerosis than whites. Similarly, our earlier study of an asymptomatic population showed more extensive extracranial atherosclerosis in blacks than in whites. Studies using angiographic evidence of atherosclerosis are likely to overestimate the prevalence of extracranial atherosclerotic disease in the population with TIA because only patients with an increased likelihood of extracranial disease are studied. Whether this bias causes blacks with extracranial disease to be less likely than whites to undergo angiography is unknown. Furthermore, in none of the angiographic studies has the overall extent of disease in blacks been compared with that in whites. We compared the extent of extracranial carotid atherosclerosis in black and white patients with TIA using B-mode ultrasound. We hoped in this way to choose a population that was less likely to have been selected for a high pretest likelihood of carotid atherosclerosis.

Subjects and Materials
Charts of all black patients discharged from North Carolina Baptist Hospital between March 1983 and February 1988 with a diagnosis of TIA ($n=129$) were retrospectively reviewed by a neurologist to ensure conformity with the standards of the National...
Institute of Neurological and Communicative Disorders and Stroke Ad Hoc Committee on Cerebrovascular Disease. Of these 129 patients, 80 were eliminated because they were misclassified or diagnostic information in the chart was incomplete. Of the remaining 49, 14 were eliminated because they were <45 years old (carotid atherosclerosis is negligible in such individuals) or had a history of stroke (previous stroke alters the prognosis and, in the case of ipsilateral stroke, complicates the diagnosis). Of the remaining 35 black patients, five had both noninvasive (B-mode ultrasound) and invasive (carotid angiography) evaluations of the carotid arteries, 22 had noninvasive evaluations only, three had invasive evaluations only, and five had neither.

Four of these latter five patients had apparent cardiac etiologies for their TIAs (atrial fibrillation, digitalis intoxication, or overmedication with anti-hypertensive agents), and one was severely debilitated due to metastatic carcinoma. Thus, 27 of 30 black patients (90%) whose carotid arteries were evaluated had noninvasive studies, whereas only eight of 30 (27%) had angiography. Although 27 black patients had B-mode ultrasound, two tests were performed at other institutions. Thus, 25 black patients remained for study. Fifty white patients were similarly identified and matched for age (±5 years) and sex to the 25 black patients. Of all white TIA patients reviewed, 51% also underwent angiography. The following information on risk factors for carotid atherosclerosis was obtained from admission notes and discharge summaries and recorded for each patient: age, race, and sex; smoking status (ever vs. never) and patient- or physician-reported histories of stroke, hypertension, diabetes, myocardial infarction, coronary artery bypass graft surgery, or angioplasty were recorded for most patients. Coronary disease was coded as present if the patient had ever had a heart attack, coronary artery bypass surgery, angioplasty, or a coronary angiogram demonstrating ≥50% stenosis of a coronary artery.

With the patient seated, the sonographer used an 8-MHz high-resolution Biosound Compact scanner (Indianapolis, Indiana) to measure the thickness of the arterial wall along the common, external, and internal carotid arteries in longitudinal and transverse planes from the anteroposterior, lateral, and postero-lateral projections. Extent of disease in the extracranial carotid arteries was scored by summing the maximum thickness of the near and far walls of the left and right common, high common, and low internal carotid arteries.

Data are reported as mean±SD B-mode score to describe patients by race and sex. Otherwise, various categories of patients are compared using mean±SEM B-mode score. Split-plot analysis of variance was used to assess differences between mean B-mode scores among the matched black and white patients. A number of other risk factors were compared using Mantel-Haenszel tests. Paired t tests were used to assess the relation between the side-specific B-mode scores and the hemisphere in which unilateral anterior-circulation TIAs were diagnosed.

**Results**

Mean±SD B-mode scores by sex and race are presented in Table 1. The mean score for blacks was very similar to that for whites; the difference was 0.6 units (95% confidence interval -2.5–3.7 units). Age had a significant positive relation to B-mode score (p=0.009, data not shown). Table 2 presents the results of analyses of covariance to assess the relations between B-mode score and sex, presence of coronary disease, histories of hypertension or diabetes, and smoking status while covarying for age. No other risk factor was significantly related to B-mode score when age was used as a covariate, although the increased (age-adjusted) mean±SEM B-mode score of males (14.8±1.1) vs. females (11.8±1.2) was of borderline significance (p=0.08).

Table 3 indicates that the black and white patients were comparable with respect to the prevalence of risk factors for carotid atherosclerosis.
A subgroup of 56 patients had unilateral anterior-circulation TIA; 32 (23 white and nine black) had more plaque on the side ipsilateral while 24 (12 black and 12 white) had as much or more plaque on the side contralateral to the TIA. Among the 35 whites in the subgroup, there was a significant ($p=0.03$) positive relation between the side-specific B-mode scores and the side of the TIA (Table 4); this relation was not apparent among the 21 blacks in the subgroup. On the other hand, blacks with unilateral anterior-circulation TIA had (nonsignificantly) more extensive extracranial carotid atherosclerosis than whites. Among the 19 patients (15 whites and four blacks) with vertebrobasilar TIs, mean B-mode score was 12.1.

**Discussion**

Prevalence studies suggest that blacks have fewer TIA than whites, and extrapolation from data obtained on patients undergoing angiography has led to the conclusion that blacks have less extracranial (but more intracranial) atherosclerosis than whites. However, autopsy studies do not confirm this. Solberg and McGarry reported that New Orleans blacks in all age groups had more fatty streaks in the carotid arteries than whites and that older black women had significantly more ($p<0.05$) raised lesions than whites. When measurements were compared with a large sample from Oslo, Norway, New Orleans blacks in almost all age groups had more raised lesions. The report concludes that blacks have at least as much carotid atherosclerosis as do whites. This conclusion was borne out by a later study. Fatty streaks were present in the carotid arteries of black men and women to a greater extent than in white men and women, and black women had consistently more raised lesions than white women. Our previous studies of asymptomatic patients also showed more extensive extracranial disease in blacks. Angiographic results should not be extrapolated to larger populations because patients undergoing angiography are not representative of asymptomatic people. Furthermore, patients who undergo angiography constitute a variable fraction of those who have a diagnosis of TIA. In general, only otherwise healthy TIA patients with a high pretest likelihood of a surgically correctable lesion undergo angiography. We found the frequency of angiography for blacks and whites with TIA to be dissimilar; approximately half of the white patients we studied but only 27% of the blacks underwent this procedure. This suggests that the angiographic results are biased and provides further rationale for the use of B-mode ultrasound (performed in 90% of TIA patients at our institution) rather than angiography to compare atherosclerotic disease in black and white patients with TIA.

In addition to selection bias, a second difference between angiography and autopsy studies is the focus of the former on stenosis and of the latter on disease extent. It is now evident that extensive disease may be present without significant stenosis. In this regard, compared with angiography studies, ours may be more similar to autopsy studies in that while angiography measures stenosis, B-mode ultrasound measures wall thickness and extent of disease as well as lumen diameter.

Another important difference between our study and previous angiography studies relates to the selection of cases. Most previous angiography studies included patients with a variety of cerebrovascular symptoms, whereas ours was limited to patients whose symptoms conformed strictly to the standard definition of a TIA. We did this partly to eliminate possible bias from differences in case mix between blacks and whites that might result if we included both TIA and stroke patients. It is likely that the extent to which study populations reflect stroke in blacks is (inversely) related to the extent of extracranial carotid atherosclerosis in that intracerebral vascular disease may be more often responsible for stroke in blacks. For our study, patients whose diagnosis was in any way suspect, as well as patients whose symptoms did not completely resolve within 24 hours, were excluded. Thus, of 129 black patients with a discharge diagnosis of TIA, only 49 actually had had a TIA. Extent of disease was very...
similar in this selected group of blacks and in an age-matched group of white TIA patients.

The only important risk factor for extracranial carotid atherosclerosis that emerged in our study was age (data not shown). Other cardiovascular disease risk factors were equally prevalent between races in this population (presumably because of selection by TIA status), and prevalence of coronary disease was comparable for blacks and whites as has been reported. However, in contrast to our previously reported results in a population undergoing coronary angiography and free of cerebrovascular symptoms, presence of heart disease, histories of hypertension and diabetes, and smoking status did not relate to the extent of carotid atherosclerosis. This may result partly from the retrospective nature of our risk factor evaluation and from the small sample size. Only a small proportion of patients in this study underwent coronary angiography, and historical information alone would have failed to identify asymptomatic patients with obstructive coronary artery disease. Alternatively, these risk factors may not be as closely related to atherosclerosis in TIA patients as in asymptomatic patients. In general, our TIA patients had more extensive carotid atherosclerosis than either the symptomatic or the contralateral side (of these 23, 11 were black).

To compare our data with those of other investigators who used angiography to study symptomatic patients, we evaluated the subgroup of 56 patients who had had unilateral anterior-circulation TIAs; 32 had more plaque ipsilateral to the hemisphere responsible for symptoms (66% of the 35 whites but only 43% of the 21 blacks) while 23 had more disease on the contralateral side (of these 23, 11 were black). These findings are quite similar to those reported by Russo, who reviewed charts and angiograms of 50 consecutive TIA patients and found only 21 with an abnormality in the extracranial portion of the appropriate carotid artery; of these, 33% were black and 67% were white. Of the 29 who had no demonstrable ipsilateral lesion, 62% were black. In our 35 white patients with unilateral anterior-circulation TIAs, the extent of extracranial carotid disease ipsilateral to the hemisphere responsible for the symptoms was greater than that on the contralateral side, but this was not true for blacks. However, in the subgroup of patients with anterior-circulation TIAs, the overall extent of disease (both sides combined) was actually greater for blacks than for whites. In addition, the overall extent of disease for patients with posterior-circulation TIAs was nearly as great (mean B-mode score 12.1) as that for patients with anterior-circulation TIAs (mean B-mode score 13.8).

In conclusion, our data suggest that blacks with TIAs have no less extensive extracranial carotid atherosclerosis than do whites. For patients with unilateral anterior-circulation TIAs, blacks had more extensive disease than whites, but in whites extracranial disease was more closely linked to the symptomatic hemisphere than in blacks. However, in these patients nearly half of those with anterior-circulation TIAs had bilateral disease and, in general, patients with symptoms related to the anterior circulation had only slightly more extensive disease than those with symptoms related to the vertebrobasilar arteries. Both subgroups had extensive disease compared with control populations previously studied (patients free of cerebrovascular symptoms and those free of coronary disease).

Our studies may partly reconcile apparent discrepancies between carotid angiography studies and studies of autopsied or asymptomatic populations. In agreement with angiography studies of patients with anterior-circulation TIAs, we found more severe disease ipsilateral to the symptomatic hemisphere in whites than in blacks; however, in this same subgroup extracranial disease was more (or no less) extensive in blacks than in whites. Overall, blacks had no less extensive extracranial disease than did whites. The data suggest that extrapolation from the severity of disease (site-specific percent stenosis) to the extent of disease (B-mode score, plaque size on autopsy) must be undertaken with caution and that patients with TIA, irrespective of site, have extensive carotid atherosclerosis.

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