Explaining Racial Disparities in Coronary Outcomes in Women

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There remains no more combustible a set of issues in American life than concern over race and gender. Much of this concern is fundamentally over economic issues relating to disenfranchisement. This translates in medicine into disparities in access to care, care delivered, and patient outcomes.

Differences in care and outcome of coronary disease have been shown to differ between white and black women, but the literature offers a mixed and complex picture. There would be little doubt about the presence of a disparity if there could be shown to be a disparity in survival at the population level. In the community-based Charleston Heart Study, 741 white women and 454 black women were monitored for up to 30 years. The mortality rate from coronary disease was 2.1 (1.6 to 2.6) per 1000 person-years for white women and 3.2 (2.3 to 4.0) for black women. This trend, with a relative risk of 1.5 comparing black with white women, did not achieve statistical significance (P=0.17). However, the cardiac mortality rate was low, as only 3% of women had coronary disease at baseline, and the sample size was somewhat small. Similar findings were noted in the Evans County study. Overall, the literature comparing long-term cardiovascular mortality in white and black women is quite limited, making it difficult to draw firm conclusions concerning the societal level of risk.

Much of the concern in the literature has been about referral to diagnostic cardiac catheterization and subsequent referral to revascularization. This issue was systematically reviewed by Sheifer et al, who found decreased rates of catheterization and revascularization in almost all studies, from various types of patient populations. As an example from this review, Peterson et al examined referral to revascularization after diagnostic cardiac catheterization in 12,402 patients in the Duke University database. Men and women were not analyzed separately. After adjustment for severity of disease, blacks were 13% less likely than whites to undergo percutaneous coronary revascularization and 32% less likely to undergo coronary artery bypass surgery. This disparity in care was more marked in patients with more severe disease, ie, those who might have benefited more from these interventional procedures. This study also included outcome data; unadjusted 5-year mortality was 27% among blacks and 20% among whites. After adjusting for baseline differences, blacks were 18% more likely to die. After adjusting for baseline risk factors and also initial treatment (ie, revascularization), there was no longer a statistically significant difference in mortality. Therefore, racial differences in revascularization explained the mortality differences. The most obvious interpretation of these findings is that revascularization improves patients' outcomes, and the lower revascularization rate among blacks is in part responsible for their higher mortality. However, this study, like other studies on this topic, was unable to take into account whether these differences might have been due to suitability to revascularization. For example, if blacks have more often distal or diffuse coronary occlusions, this fact might make them less often ideal candidates for intervention than whites.

Differences in revascularization after initial coronary angiography may underestimate disparity in access, as there may be a greater disparity in access to the initial diagnostic angiogram. Indeed, Schulman et al conducted a controlled simulation that suggested that black women in particular are less likely to be referred to angiography.

The issue of disparity in care has also been considered for the care of patients suffering an acute myocardial infarction. Canto et al studied this issue in the Cooperative Cardiovascular Project. Among 10,026 white women and 732 black women who were eligible for reperfusion, black women received reperfusion 44% of the time and white women 56%. After adjustment for baseline differences, black women were 10% less likely than white women to receive thrombolysis. Schneider et al used a managed care database to show that use of ß blockers after myocardial infarction was 13% lower in blacks (64%) than in whites (74%, P<0.005). Similar results were reported by Rathore et al. Care and outcome of blacks (n=943) and nonblacks (n=2375) with non-ST elevation MI were compared in the TIMI III registry (41.7% women). Blacks had more hypertension and diabetes but less severe disease than whites and were less likely to undergo angiography or revascularization (risk ratio 0.65; P<0.001). However, there was no difference in death or myocardial infarction for all blacks versus whites or black women versus white women at 42 days, and blacks had less recurrent ischemia.

Overall, the literature indicates that there are disparities in care, but they tend to be small. Furthermore, the reasons for these differences are unclear, and many studies cannot rule out the influence of residual confounding (due, for example, to presence or severity of comorbid conditions), patient preferences, and, for coronary procedures, angiographic indi-
cations. In addition, the question of whether there is a difference in outcome has only been addressed in a few studies, with inconsistent results. Finally, whether differences in outcome can be attributed to disparities in care remains uncertain.

The issue of whether there are outcome differences, and whether differences in outcome can be explained by differences in the quality of care, is addressed in this issue of Circulation by Jha et al.10 The authors compared 218 African-American women to 2,451 white women in the Heart and Estrogen/progestin Replacement Study (HERS). HERS was a randomized trial evaluating the effect of hormone replacement therapy on cardiovascular outcomes. As there was no treatment effect, the treatment arms were combined in the present study. During an average of 4.1 years of follow-up, cardiovascular events were twice as common in black as in white women (6.4 versus 3.1 per 100 person-years). Black women had higher rates of hypertension, diabetes, and hyperlipidemia but were less likely to be treated with aspirin or statins. Black women were less likely to have optimal blood pressure (56% versus 63%) or LDL cholesterol (30% versus 38%). Differences in pharmacological therapy were small; the only statistically significant difference in recommended treatments according to guidelines was in use of statins in women with LDL cholesterol >100 mg/dL (35% is whites and 26% in blacks). There were no differences in cardiac catheterization and revascularization procedures, which is in contrast with previous studies as described above. This finding, however, cannot be generalized, as women in a prospective trial may be referred to revascularization at quite different rates than women in the wider community.

Mortality, however, was higher in black women than in white women. In unadjusted models, the hazard ratio for black compared with white women was 2.05 (95% CI 1.52 to 2.77) for cardiovascular death or MI, 2.00 (95% CI 1.40 to 2.86) for nonfatal MI, and 2.21 (95% CI 1.39 to 3.70) for cardiovascular death. After adjusting for baseline differences, including demographic factors, geographic location, comorbidity, presence of multivessel disease, heart failure, diabetes, and hypertension, black women still had a 60% greater risk of coronary events in follow-up. After adjusting for differences in baseline characteristics as well as therapy, black women still had over a 50% higher risk of nonfatal myocardial infarction or cardiovascular death than white women (hazard ratio 1.52; 95% CI 1.04 to 2.21). However, looking at the individual components, there was only a slight trend for increased cardiovascular death (hazard ratio 1.17; 95% CI 0.63 to 2.17), whereas a strong relationship to nonfatal MI remained (hazard ratio 1.77; 95% CI 1.12 to 2.77).

This study found a higher event rate in black than in white women. The difference in mortality was largely explained by differences in baseline characteristics and to some extent by differences in treatment. However, the difference in nonfatal myocardial infarction could not be explained by the measured variables. Thus, there remain either differences in patient characteristics, therapy, or lifestyle, which have not been examined in this study but which must account for the increased event rates in black women. It follows that it remains unclear, from the results of this study, why there are higher rates of events in black than in white women. Presumably there is nothing about skin color per se, and that if the proper covariates are found, then the differences between black and white women could be completely accounted for.

Thus, we come back to black women either being at higher risk because they are sicker or because they receive worse care. Although both of these possibilities seem to be true in the current series, neither were, or could be, measured perfectly. However, given the available information, black women were undeniably sicker with more depression, higher body mass index, more renal dysfunction, more hypertension and more diabetes, greater inactivity, and more heart failure. The differences in care were more modest and had only a modest effect on outcome. Perhaps, then, there are additional risk factors or some of these risk factors may have been inadequately measured, which explains the observed differences between black and white women.

Can the data in this study, showing that black women with coronary disease are sicker, with more comorbidity and heart failure and more recurrent events than white women, be generalizable to the wider community? One possibility is that this trial is not representative of the population, and that black women with more severe disease more often choose to be in the trial to get better care. Alternatively, less sick black women may have chosen not enter the trial as they distrusted the investigators more than white women did. Indeed, not all studies have found blacks to be sicker than nonblacks. But if the difference in severity of disease could be generalized, then the question becomes, why are black women sicker and at greater risk? Perhaps they are at greater risk on a genetic basis, for example, through a predisposition to develop diabetes and hypertension. However, there are no data to support such a genetic basis. Alternatively, black women may be sicker because of environmental influences that have caused them to develop more risk factors such as diabetes and hypertension.

Thus, we find that this provocative paper by Jha et al has raised more questions than it answers. Clearly, these black women are at greater risk. Coexisting disease and risk factors, and differences in treatment, explain some but not all of the outcome differences between black and white women. Thus, we remain uncertain as to the cause of racial disparities in outcome, and unsure about the generalizability of this difference. We are also not sure of the cause of racial disparities in severity of disease and comorbidity. Further research will be necessary to understand the extent of these disparities and their causes in populations that are as unbiased as possible. This being said, it is important to realize that race is a social construct, not a meaningful scientific classification, and racial identification is uncertain at best.11 These are critical issues, because until disparities are well understood, it is difficult to design optimal programs to overcome disparities and give all people, no matter what their color, the best chance of the best outcome as efficiently as we can.

References


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