Heart Failure Prevention Is the Best Option To Stem High Costs and Disease Burden

Research for More Effective Heart Failure Treatment Is Needed

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In this issue of Circulation: Cardiovascular Quality and Outcomes, Okin and colleagues¹ report on the incidence of heart failure in a cohort of patients with hypertension enrolled in the Losartan Intervention for End Point Reduction in Hypertension (LIFE) study. This study, an international randomized trial, demonstrated that losartan, when compared with atenolol, provided superior reduction of events in high-risk patients with hypertension and no history of heart failure before enrollment.² Over a 5-year follow-up period, the incidence of heart failure in blacks was 7%, significantly higher than the 3.1% incidence rate found in nonblack patients. This difference persisted even when appropriately adjusted for differences such as the heavier risk factor burden, younger age, sex, renal disease, diabetes mellitus, randomized treatment, incident myocardial infarction, in-treatment QRS duration, strain and left ventricular hypertrophy as measured by ECG, and diastolic and systolic pressure. Additionally, an echocardiographic substudy demonstrated adverse differences in left ventricular structure and performance after 2 years of antihypertensive therapy in blacks compared to nonblacks.

These findings are consistent with several previous reports detailing heart failure differences in blacks, who, when receiving their diagnosis of heart failure, are younger; have greater risk factor burden; are more overweight; and are more likely to have diabetes, renal disease, less coronary disease, and higher heart failure-related hospitalization and death rates than whites.³ ⁴ Unique in this report is that the cohort is entirely high-risk patients with hypertension. The small numbers of black participants (497) compared with white participants (8199), and thus, small numbers of new heart failure cases in blacks, is an important recognized limitation.

The opinions expressed in this article are not necessarily those of the editors or of the American Heart Association.

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adherence, blacks are consistently undertreated and experience disproportionate disease burden. A National Heart, Lung, and Blood Institute-initiated program to improve hypertension control rates in blacks supports several community-based projects and offers multiple strategies. The results of this program are not yet reported. Aggressive programs to prevent CVD, and thus heart failure, by controlling conventional cardiovascular risk factors, especially hypertension, will decrease burden of disease for all patients at risk. Prevention programs that target high-risk blacks are urgently needed.

The second imperative—to identify novel mechanisms of heart failure in black patients with hypertension—is also important. Blacks have the highest prevalence of hypertension in the United States, and black patients with heart failure have more hypertension and less coronary disease. Among otherwise similar patients with hypertension, why should some black patients have an accelerated progression of myocardial dysfunction and heart failure? Investigations of potential alternate disease pathways may offer new insight or strategies for antihypertensive and myocardial protective therapeutic targets.

Elucidating mechanisms of heart failure is the Holy Grail for many established and emerging basic and clinical investigators. Racial differences in response to drug treatment have gained interest in treating heart failure. Some well-documented examples include response to diuretics, β-adrenergic blockade, and angiotensin-converting enzyme (ACE) inhibitors, and all have been evaluated with the hope of identifying mechanisms for observed differences and how these might affect patient management. Pharmacogenomics is an emerging field of high interest and may provide an opportunity for tailoring pharmacological intervention to individual or population characteristics in the treatment of heart failure. Indeed, the differential changes in left ventricular structure and function observed in black LIFE participants, which cannot be accounted for by known differences in risk factors, might be explained by genetic variation in disease mechanisms. This finding should spark further investigation. However, the context of race for elucidating disease mechanisms has proven problematic. Using self-identified race as a proxy for socioeconomic and environmental factors, and without a common gene pool, blacks experience a disproportionate burden of CVD.

The implications of racial differences have become an albatross for the patient and provider. The biological significance of race is correctly suspect because race is a social construct, a proxy for socioeconomic and environmental factors, and without a common gene pool. Historically, race and ethnicity have been exploited, even in medicine, to promote or provide a rationale for unjust practices. Providers often are not comfortable discussing race with their patients. Black patients, having appropriately learned to question the notion of “separate but equal,” are reluctant to accept treatments marketed for blacks, regardless of the strength of the science. Public health officials, investigators, providers, and patients cannot afford to be ambivalent about identification and aggressive treatment of high-risk profiles, including evaluation of the role of race. Discomfort, lack of trust, and suspicion are regrettable and will keep us mired in past failures. Aggressive heart failure prevention programs can begin immediately and will quickly save lives and reduce healthcare costs. A robust research agenda, including investigation of the influence of race on disease manifestations and treatment, will require more time to improve therapeutic options. Even though it may be difficult, meeting the public health imperative to reduce disability and death in blacks is both feasible and correct.

Disclosures

None.

References


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