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Heart Disease and Kidney Failure in the Black Community

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In the United States, Black Americans suffer from a disproportionately high prevalence of cardiometabolic disorders including cardiovascular disease (CVD) and chronic kidney disease/end-stage kidney disease (CKD/ESKD) compared with their White peers.[1,2] The reasons for these disparities are multifactorial. These disparities are primarily driven by societal factors, racial- and ethnic-based inequities in the allocation of health-affirming resources and opportunities, which is commonly termed structural racism.[3] This is often manifested as long-standing community-level disinvestment in support for health (eg, employment, education, neighborhood safety, physical activity, food security, environmental justice), health care (health insurance, limited access to quality care, trust/distrust of the medical system), and more that may lead to poor cardiometabolic health and other medical conditions.[4]

There are also common racial group differences in the prevalence of abnormal biologic factors from blood pressure to structural cardiac abnormalities and more. Some of these may be more directly related to a greater burden of personal and societal-levied psychological insults (eg, discrimination, marginalization, invalidation) leading to group differences in anxiety, stress, and states of hypervigilance leading to neurohormonal activation, enhanced expression of mediators that increase inflammation and suppress immune function, and epigenetic modifications.[5] The confluence of these as well as other related insults contributes to the higher rates of development of CVD and CKD/ESKD, their risk factors, and ultimately the progression to premature CVD- and CKD/ESKD-associated morbidity and mortality.[2]

Racial and ethnic differences in health conditions, outcomes, and treatment response inform us that there are many important elements differentially affecting socially assigned racial and ethnic groups and task us as researchers to unravel which factors are most relevant and for which disease states. Depending on the question being asked, this work can be performed through health disparities research (examining differences between groups) or minority health research (examining the nuances of a health condition within a minoritized group). The strength of a minority health research approach is the ability to examine many covariates and comorbidities within a racial or ethnic group; whereas there is much social heterogeneity within groups, it is much less than that between socially designated racial and ethnic groups.[6]

To better understand the potential relation of echocardiographic parameters of cardiac structure and function and the risk of incident ESKD, Kou et al[7] analyzed data from more than 2000 Black participants from the Jackson, Mississippi, site of the Atherosclerosis Risk in Communities (ARIC) study during a nearly 25-year period of observation (January 1, 1993, through July 31, 2017). They found that abnormalities in several measures of cardiac structure (interventricular septal thickness, left ventricular and left ventricular mass index, and posterior wall thickness) were strongly associated with progression to ESKD across clinical and demographic subgroups after multiple statistical adjustments. By contrast, measures of cardiac function (left ventricular ejection fraction and the ratio of early diastolic peak flow velocity to late diastolic peak flow velocity) were not significant after accounting for demographic and clinical factors with the exception of a lower ejection fraction in participants younger than 60 years, the only subgroup in which cardiac function appeared to be independently associated with an increased risk of ESKD.

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The limitations of the study included recruitment from a single community site, a single baseline echocardiographic measurement, and the lack of inclusion of inflammatory or oxidative stress mediators. These could be important as prior studies have reported that elevated plasma markers of inflammation in patients with CKD have been associated with endothelial dysfunction, increase in left ventricular mass index over time, and rapid loss of kidney function.[8,9] Other markers, such as allostatic load, scores for constructs such as lifetime discrimination or financial stress, or the potential association of treatment with different medications such as inhibitors of the renin-angiotensin system, could also have provided greater insight into potential mechanisms through which cardiac structure may relate to an increased risk of ESKD. However, there were several strengths to the study, such as echocardiographic assessment of cardiac structure and function, being able to control for several important cardiometabolic medical conditions and laboratory markers, and leveraging of data from a well-documented and well-characterized community-based cohort that was observed for almost 25 years. This long-term observation was critical for ascertaining the risk of ESKD development.

In conclusion, Kou and colleagues[7] found that echocardiographic parameters of left ventricular structure, but not function, had a robust association with the future risk of incident ESKD in Black Americans with CKD, a cohort with an overall very high risk for development of ESKD. Their findings support the early measurement of left ventricular structure in patients with CKD to help identify those even more likely to progress to ESKD. Although this was not an intervention study, based on their findings, it would seem prudent to consider aggressive guideline-based care, such as the use of renin-angiotensin system inhibitors and sodium-glucose co-transporter 2 inhibitors, in persons with CKD and abnormal left ventricular structure to reduce CKD progression, especially given their established effects on patients with key CKD progression factors, such as albuminuria, hypertension, and diabetes, as well as their ability to reduce adverse cardiovascular events and to lower mortality risk in patients with CKD and CVD.[10]

POTENTIAL COMPETING INTERESTS

The authors report no competing interests.

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REFERENCES

1. Fernandes-Silva MM, Shah AM, Hegde S, et al. Race-related differences in left ventricular structural and functional remodeling in response to increased afterload: the ARIC study. *JACC Heart Fail*. 2017;5(3):157-165.
2. Norton JM, Moxey-Mims MM, Eggers PW, et al. Social determinants of racial disparities in CKD. *J Am Soc Nephrol*. 2016;27(9):2576-2595.
3. Eneanya ND, Boulware LE, Tsai J, et al. Health inequities and the inappropriate use of race in nephrology. *Nat Rev Nephrol*. 2022;18(2):84-94.
4. Smedley BD, Stith AY, Nelson AR; Institute of Medicine, Committee on Understanding and Eliminating Racial and Ethnic Disparities in Health Care. *Unequal Treatment: Confronting Racial and Ethnic Disparities in Health Care*. National Academies Press; 2003.
5. Nicholas SB, Kalantar-Zadeh K, Norris KC. Socioeconomic disparities in chronic kidney disease. *Adv Chronic Kidney Dis*. 2015;22(1):6-15.
6. Alvidrez J, Castille D, Laude-Sharp M, Rosario A, Tabor D. The National Institute on Minority Health and Health Disparities research framework. *Am J Public Health*. 2019;109(S1):S16-S20.
7. Kou M, Hishida M, Mathews L, et al. Echo-based cardiac structure parameters for the long-term risk of end-stage kidney disease in Black individuals: the ARIC study. *Mayo Clin Proc*. 2022;97(10):1794-1807.
8. Amdur RL, Feldman HI, Gupta J, et al. Inflammation and progression of CKD: the CRIC study. *Clin J Am Soc Nephrol*. 2016;11(9):1546-1556.
9. Ioannou K, Stel VS, Dounousi E, et al. Inflammation, endothelial dysfunction and increased left ventricular mass in chronic kidney disease (CKD) patients: a longitudinal study. *PloS One*. 2015;10(9):e0138461.
10. Brosius FC, Cherney D, Gee PO, et al. Transforming the care of patients with diabetic kidney disease. *Clin J Am Soc Nephrol*. 2021;16(10):1590-1600.