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Left Ventricular Hypertrophy and Cardiovascular Mortality by Race and Ethnicity

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Abstract

Background—Left ventricular hypertrophy is a major independent risk factor for cardiovascular mortality. The contribution of left ventricular hypertrophy to racial and ethnic differences in cardiovascular mortality is poorly understood.

Methods—We used data from the Third National Health and Nutrition Examination Survey and from the National Death Index to compare mortality for those with an electrocardiographic (ECG) diagnosis of left ventricular hypertrophy to those without left ventricular hypertrophy separately for whites, African Americans, and Latinos. We used Cox proportional hazards regression to control for other known prognostic factors.

Results—ECG left ventricular hypertrophy was significantly associated with ten-year cardiovascular mortality in all three racial/ethnic groups, both unadjusted and adjusted for other known prognostic factors. The hazard ratio for this association was significantly greater for African Americans (2.31, 95% CI 1.55–3.42) than for whites and Latinos (1.32, 95% CI 1.14–1.76 and 2.11, 95% CI 1.35–3.30 respectively) independent of systolic blood pressure.

Conclusions—ECG left ventricular hypertrophy contributes more to the risk of cardiovascular mortality in African Americans than it does in Whites. Using regression of ECG left ventricular hypertrophy as a goal of therapy might be a means to reduce racial differences in cardiovascular mortality; prospective validation is required.

Keywords

left ventricular hypertrophy; electrocardiography; cardiovascular risk assessment; racial/ethnic differences

Rates of cardiovascular mortality in the United States are higher for minority patients¹. Hypertension might play a major role in the genesis of this mortality difference. The incidence of hypertension is greater in African Americans than in whites and Latinos. Although the incidence of hypertension is comparable in whites and Latinos, rates of *controlled* hypertension are significantly lower in Latinos¹.

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Left ventricular hypertrophy is a consequence of poor hypertension control as well as a risk factor for cardiovascular mortality independent of elevation in blood pressure^{2,3} perhaps because it identifies a sub-population that is particularly sensitive to the adverse effects of hypertension. Differences in prevalence of left ventricular hypertrophy by race and ethnicity might be expected to explain differences in cardiovascular mortality, but data supporting this view are scarce. In one study confined to patients with coronary heart disease, left ventricular hypertrophy by echocardiography was associated with a higher population attributable risk for mortality in African American patients than in whites⁴.

The aim of the current study was therefore to assess the degree to which the presence of left ventricular hypertrophy predicts mortality across race and ethnicity in community-based adults unselected with regard to co-morbidity. The study used electrocardiographic (ECG) criteria for determining left ventricular hypertrophy that are based on thresholds in the relationship between ECG parameters and mortality⁵. We chose to use ECG criteria because the electrocardiogram is less expensive and more widely available compared with echocardiography or magnetic resonance imaging, and thus may be more suited for application to population-wide hypertension management.

Methods

Data Sources

The data sources for the study were the Third National Health and Nutrition Examination Survey (NHANES III)⁶ database and the NHANES III Linked Mortality File⁷. NHANES III was an epidemiologic survey designed to obtain a sample representative of the civilian, non-institutionalized population of the United States greater than 2 months old between 1988 and 1994. Twelve-lead electrocardiograms were obtained from all subjects older than 40 years. Tracings were recorded at the mobile examination center and later transferred to a central location for electronic analysis.

The NHANES III Linked Mortality File contains the results of matching NHANES III subject identifiers with data available in the National Death Index⁸ as of December 31, 2000. Date of death and cause of death available in the National Death Index are derived from death certificates.

Inclusion criteria

We included all subjects for whom data was available to calculate the ECG measure, for whom vital status at ten years was known, and who were identified as Caucasian, African American or Hispanic. In addition, we included only those subjects with a QRS duration < 0.12 msec, since validity of the ECG measure in the presence of bundle branch block has not been established.

Definitions

We defined left ventricular hypertrophy with an ECG measure, the Novacode estimate of left ventricular mass index. We chose this measure because it had the strongest associations with mortality and the greatest percent population attributable risk compared with other validated measures in a previous study⁵. The reasons for the superior performance of this measure over other ECG measures for predicting mortality likely include that it accounts for the presence of obesity and for the presence of the “strain pattern” that has been demonstrated to predict mortality independently^{9–11}. The Novacode system has also been found to predict cardiovascular endpoints in other prior studies^{12,13}. This measure is calculated from a set of linear regression equations, one for African American women, one for white (including Latino) women, and one for men¹⁴.

$$\text{Novacode LVMI}_{\text{African American women}} = 0.0216(R \text{ aVL}) + 0.0184(R V_6 + S V_2) - 0.0143(R V_2) - 0.0693(Q \text{ or } S V_6) + 0.199(T \text{ aVL}) + 0.746(\text{QRS duration}) - 22.3064$$

$$\text{Novacode LVMI}_{\text{white women}} = 0.0178(R V_5) + 0.0528(Q \text{ or } S V_5) - 0.1128(Q \text{ or } S I) + 0.1075(T V_1) + 0.1701(T \text{ aVF}) - 0.0939(T V_6) + 88.4357$$

$$\text{Novacode LVMI}_{\text{men}} = 0.01(R V_5) + 0.0203(Q \text{ or } S V_1) + 0.0287(Q \text{ or } S III) + 0.1819(T V_6) - 0.1482(T \text{ aVR}) + 1.0485(\text{QRS duration}) - 36.429$$

We considered left ventricular hypertrophy to be present when Novacode LVMI is greater than 115 gms/m² for women and greater than 130 gms/m² for men⁵.

We selected ten-year cardiovascular mortality as the primary outcome of interest. We defined cardiovascular deaths as those for which the death certificate principal cause of death was ICD-9 codes 390.0–459.9 (for deaths before or during 1999) or ICD-10 codes I10–I15, I20–I25, I50–I51, I60–I69, I70–I73 (for deaths after 1999). In secondary analyses, we assessed the association between left ventricular hypertrophy and five-year cardiovascular mortality, and five- and ten-year total mortality. We also explored five- and ten-year coronary heart disease (CHD) mortality as possible outcomes of interest. Because of small numbers of CHD deaths (at ten years, whites 71 deaths, African Americans 17 deaths, Latinos 8 deaths), possibly as a result of under-coding of CHD on death certificates, multivariable analyses with CHD as an outcome are not feasible with this data set.

Statistical Analysis

Descriptive statistics and frequency distributions were generated for sociodemographic and clinical variables by race/ethnicity. Comparisons of patient characteristics across racial/ethnic groups for these variables were made with one-way analysis of variance for continuous variables and chi-square tests for binary variables.

We used Cox proportional hazards models to test whether left ventricular hypertrophy was associated with cardiovascular mortality separately among African American, Latino, and white subjects. In the first set of models (unadjusted), the presence or absence of left ventricular hypertrophy was the only independent variable. It should be noted that there is an accounting for gender inherent in this “unadjusted” model, since the presence of left ventricular hypertrophy is defined separately for men and women. In the second set, age and systolic blood pressure were added to left ventricular hypertrophy. In the third set, smoking, total and HDL cholesterol, and diabetes were added to left ventricular hypertrophy, age, and blood pressure.

We used Cox proportional hazards models with a race/ethnicity $\times$ left ventricular hypertrophy interaction term to test whether left ventricular hypertrophy was differentially associated with outcome across the three racial/ethnic categories. In the first model we included race/ethnicity, presence of left ventricular hypertrophy, and the interaction term. In the second model we included the above terms along with age and systolic blood pressure as continuous variables. In the third model we added smoking, total and HDL cholesterol, and diabetes to race/ethnicity, left ventricular hypertrophy, the interaction term, age, and blood pressure. Hazards ratios associated with left ventricular hypertrophy were estimated for the three race/ethnicity groups for all models.

Statistical analysis was performed using SAS, Version 9.1 for personal computer.

Results

There were 7495 subjects in the NHANES III data set with ECG, vital status, and race/ethnicity data that met the requirements for inclusion in the study. The mean age of the included patients

was 59.5 years. Among included patients, 52.3% were women. The sample was 24.4% African American, 51.7% white, and 23.9% Latino. Clinical characteristics of the study sample are shown in Table 1. As a result of the large sample size, racial/ethnic differences were statistically significant for all variables and of small magnitude for most. The notable exception is that the prevalence of ECG left ventricular hypertrophy in African Americans was approximately twice that in Latinos and whites despite rather small differences in mean systolic blood pressure across the three groups; the prevalence of left ventricular hypertrophy by hypertension categorization did not differ across racial/ethnic categories.

In unadjusted analyses, left ventricular hypertrophy was significantly associated with ten-year cardiovascular mortality for all three racial/ethnic groups (Table 2), and remained significant after adjusting first for age and systolic blood pressure and then for smoking, total and HDL cholesterol, and diabetes in addition to age and systolic blood pressure. The results of the secondary analyses with five-year cardiovascular mortality and five- and ten-year total mortality as the outcomes (Table 3) parallel the results of the primary analysis.

We also assessed whether or not the effect of left ventricular hypertrophy on mortality differed across the three racial/ethnic groups (African American vs. white and Latino vs. white). As shown in Table 2, the magnitude of the association between electrocardiographically-estimated left ventricular mass index and cardiovascular mortality was not significantly different for Latino and white subjects. For African American subjects, however, the hazard ratio associated with left ventricular hypertrophy was significantly greater than the hazard ratio for white subjects in the model adjusting for age and blood pressure and was borderline greater in the fully adjusted model.

Discussion

Using data from a nationally representative epidemiological survey, we found that the presence of left ventricular hypertrophy determined by ECG was a strong predictor of subsequent cardiovascular mortality for whites, African Americans, and Latinos. The ECG indicator of left ventricular hypertrophy remained a significant predictor of outcome across all three groups after adjustment for systolic blood pressure. Furthermore, left ventricular hypertrophy was a stronger predictor of cardiovascular mortality in African Americans than it was in whites. The hazard ratios in Latinos were consistently greater than the hazard ratios in whites and consistently similar to the hazard ratios in African Americans, suggesting that the relatively low number of events in Latinos might have rendered this analysis underpowered to detect meaningful differences for this ethnic group. With five- and ten-year total mortality as outcomes the differences by race and ethnicity are muted, perhaps reflecting excess non-cardiovascular mortality in minority communities during the 1990s when the NHANES III cohort was created¹⁵.

Comparison with prior literature

Prior studies have suggested that the ECG is a less useful tool for assessing for the presence of left ventricular hypertrophy in African Americans. Lee et al¹⁶ compared the sensitivity and specificity of ECG indicators of left ventricular hypertrophy in African Americans and whites compared with echocardiography. Among 122 African American and 148 white subjects with hypertension, each of the five ECG indicators of left ventricular hypertrophy had lower specificity in African Americans compared with whites. The authors concluded that “standard ECG criteria will yield a higher number of false positive diagnoses of left ventricular hypertrophy in black hypertensives than in white hypertensives”. Chapman and colleagues¹⁷ likewise reported lower specificity for ECG indicators of left ventricular hypertrophy in African Americans compared with whites among 408 patients with hypertension. Arnett et al¹⁸ found sensitivity and specificity of ECG criteria for left ventricular

hypertrophy in a community-based group of 196 African Americans to be similar to that previously reported for whites; direct racial comparisons were not reported. Okin and colleagues¹⁹, reporting data from a randomized trial of anti-hypertensive therapy, reported lower specificity for African American subjects compared with white subjects using the Sokolow-Lyons ECG indicator of left ventricular hypertrophy (R wave amplitude in lead V₁ + S wave amplitude in lead V₅ or V₆ > 35 mm) but not the Cornell voltage indicator (R wave amplitude in lead aVL + S wave amplitude V₃ > 20 mm in women or 28 mm in men). In a study of patients with coronary heart disease, left ventricular hypertrophy by echocardiography was associated with a higher population attributable risk for mortality in African American patients (20%) compared with whites (16%)⁴.

In contrast to the relatively large number of studies of the diagnosis and prognostic implications of left ventricular hypertrophy for African Americans, data for Latinos are scarce. It has been reported that ECG voltages in Latinos varies systematically from other ethnic groups²⁰. A study of a database containing information on over 45,000 electrocardiograms at a single Veterans Administration hospital found a lower prevalence of left ventricular hypertrophy using the Romhilt-Estes criteria in Latinos (3.3%) compared with non-Latinos (5.3%)²¹. The proportion of the non-Latino group that was African American is not given. Although the presence of left ventricular hypertrophy by echocardiography has been demonstrated to be an independent predictor of vascular events in Latinos²², comparisons of the strength of the association with other demographic groups has not been reported.

The prevalence of left ventricular hypertrophy by ECG in our study is somewhat lower than the prevalence reported by Drazner and colleagues using magnetic resonance imaging and careful control for body size and composition in the Dallas Heart Study²³. These investigators studied 2193 African American or white Dallas residents selected at random, with over-sampling of African American subjects in order to ensure that adequate racial comparisons could be made. They report a prevalence of left ventricular hypertrophy of 2.8% in white women and 7.7% in African American women, and 4.5% and 8.2% in white and African American men respectively. Despite differences in absolute values, the white:African American ratio of prevalences in the Dallas Heart Study is similar to that seen in our study.

Our study differs from most previous studies in three important ways. First, our indicator of left ventricular hypertrophy was defined against mortality rather than echocardiographically-determined left ventricular mass. Second, we used a relatively underutilized ECG indicator of left ventricular hypertrophy, the Novacode estimate of left ventricular mass index, which has a stronger relationship with mortality than other indicators. This indicator incorporates the T wave inversion found in the strain pattern that is associated with higher mortality^{9–11}, partially accounts for obesity, and uses gender- and race-specific regression equations. Third, we assessed the impact of left ventricular hypertrophy on mortality in a community sample rather than in a sample containing only patients with hypertension or coronary heart disease.

Potential mechanisms

There are several potential reasons why Novacode left ventricular mass index above threshold might be associated with cardiovascular mortality to a greater degree in African American patients. The pattern of concentric left ventricular hypertrophy, characterized by both increased wall thickness and increased cavity dimension, has been associated with poorer prognosis in most studies^{24–26}, and African American²⁷ and Latino²⁸ patients are more likely to develop concentric hypertrophy compared with whites. It is possible that an increase in the Novacode left ventricular mass index reflects not only increases in mass but also changes in geometry. Genetic factors may play a role. Polymorphisms that cause a more vigorous hypertrophic response to hypertension may be more common in African Americans. There may linkage in African Americans between a predisposition to left ventricular hypertrophy and predisposition

to atherosclerotic disease or cardiomyopathy. Environmental factors common among socio-economically disadvantaged patients such as chronic psychological stress or a diet more likely to result in obesity may also play a role, particularly if these environmental factors interact with genetic factors. Finally, treatment factors may play a role, with disadvantaged patients receiving less vigorous treatment subsequent to the initial examination as a result of poorer access to care.

Limitations

There are important limitations to our study. First, the study relies on cross-sectional data. Treatment patterns and blood pressure subsequent to the initial NHANES III examination are not known. Second, our definition of cardiovascular mortality relies on death certificate diagnoses rather than medical record review. Finally, our methodology relies on an ECG measure that requires computer-assisted interpretation, potentially limiting generalizability.

Conclusions

An ECG indicator of left ventricular hypertrophy, the Novacode estimate of left ventricular mass index, predicted cardiovascular mortality independent of systolic blood pressure in whites, African Americans, and Latinos. The hazard ratio for the association between left ventricular hypertrophy and outcome was greater for African Americans compared with whites. If validated in other populations, these findings suggest that electrocardiographic left ventricular hypertrophy identifies a particularly high-risk group among minority patients, who may benefit from more aggressive intervention. Electrocardiography might be a useful means of following response to treatment in minority patients with hypertension and as such might augment efforts to reduce health disparities attributable to hypertension.

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Table 1

Demographic and clinical characteristics of the study sample

	White	African American	Latino	P
N	3874	1832	1789	
Mean age (sd)	63.2 (13.8)	56.5 (12.3)	56.4 (11.9)	<0.001
Gender (% female)	53.4%	53.2%	49.4%	0.014
Smoker	19.0%	32.4%	20.5%	<0.001
Total cholesterol, mean±sd, mg/dl	221 ± 43.2	215 ± 46.0	215 ± 44.4	<0.001
(mmol/l [*])	(5.72 ± 1.12)	(5.57 ± 1.19)	(5.57 ± 1.15)	
HDL cholesterol, mean±sd, mg/dl	50.6 ± 16.0	54.7 ± 18.3	48.4 ± 14.4	<0.001
(mmol/l)	(1.31 ± 0.41)	(1.42 ± 0.47)	(1.25 ± 0.37)	
Diabetes	8.3%	12.5%	15.8%	<0.001
Systolic blood pressure, mean±sd	133 (19.5)	134 (20.5)	132 (19.7)	0.003
(mmHg)				
Left ventricular hypertrophy, N (%)	66 (1.7%)	69 (3.8%)	25 (1.4%)	<0.001
In pts with sys BP < 140	17 (0.4%)	20 (1.1%)	6 (0.3%)	
In pts with sys BP > 140	49 (1.3%)	49 (2.7%)	19 (1.1%)	0.86

* conversion factor 0.0259

Table 2
Hazard ratios for 10 year cardiovascular mortality associated with left ventricular hypertrophy

	White (413 events)		African American (124 events)		Latino (92 events)	
	HR	95% CI	HR	95% CI	HR	95% CI
Unadjusted	2.54	2.08–3.12				
Adjusted for age and systolic blood pressure	1.32	1.07–1.62	2.55 [*] 2.31 ^{**}	1.78–3.67 1.61–3.34	3.10 [*] 2.11 [*]	2.02–4.76 1.37–3.25
Adjusted for age, blood pressure, smoking, total and HDL cholesterol, and diabetes	1.42	1.14–1.76	2.30 [†]	1.55–3.42	2.11 [*]	1.35–3.30

^{*} p = NS for comparison with White
^{**} p = 0.008 for comparison with White
[†] p = 0.052 for comparison with White

Table 3

Hazard ratios for associations between secondary outcome measures and left ventricular hypertrophy, controlling for age, blood pressure, smoking, total and HDL cholesterol, and diabetes

	White		African American		Latino	
	HR (95% CI)	# of events	HR (95% CI)	# of events	HR (95% CI)	# of events
5-year cardiovascular mortality	1.35 (0.99–1.86)	211	2.94 (1.67–5.19)	62	2.04 (1.04–4.02)	46
5-year total mortality	1.11 (0.89–1.39)	465	1.49 (1.02–2.17)	154	1.33 (0.85–2.10)	124
10-year total mortality	1.19 (1.02–1.39)	917	1.41 (1.08–1.83)	334	1.15 (0.83–1.60)	249