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Heart Failure with Preserved Ejection Fraction in African-Americans – The Atherosclerosis Risk in Communities (ARIC) Study

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Abstract

Objectives—In an entirely African-American cohort, we compared clinical characteristics, cardiac structure and function, and all cause mortality in heart failure (HF) with preserved ejection fraction (HFpEF) in relation to HF with reduced ejection fraction (HFrEF) and those without HF.

Background—African-Americans are at increased risk for HF. Nevertheless, there are limited phenotypic and prognostic data in African-Americans with HFpEF compared to those with HFrEF and those without HF.

Methods—Middle-aged African-Americans from the Jackson cohort of the Atherosclerosis Risk in Communities study (n=2,445) underwent echocardiography between 1993 and 1995. HF prevalence was available in 1,962 for whom left ventricular ejection fraction (LVEF) could be quantified. Participants with HF were categorized as having HFpEF (LVEF ≥ 50%) or HFrEF (LVEF < 50%), or no HF, with comparisons made between groups.

Results—HF was identified in 116 (5.9%) participants (n=85 [73%] HFpEF; n=31 [27%] HFrEF). Compared to those without HF, those with HFpEF were older, more likely to be female, had more frequent comorbidities, and concentric hypertrophy. In relation to HFrEF, those with HFpEF were more likely female, but less likely to have coronary heart disease, diabetes mellitus, chronic kidney disease, left atrial enlargement, and eccentric hypertrophy. Over a median 13.7 years of follow up, risk of death differed between groups, with age and sex adjusted hazard ratios

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of 1.51 (95%CI 1.01–2.25) for HFpEF vs. those without HF, and 2.50 (95%CI 1.37–4.58) for HFrEF vs. HFpEF.

Conclusions—In this cohort of middle-aged African-Americans, HFpEF was the most common form of HF, and was associated with a substantially better prognosis than HFrEF, but worse than those without HF.

Keywords

African-Americans; heart failure with preserved ejection fraction; heart failure with reduced ejection fraction; echocardiography; mortality

Introduction

African-Americans, as compared to other racial groups, are at increased risk for the development of heart failure (HF) (1–3). Additionally, HF in African-Americans occurs at a younger age and is associated with a higher prevalence of cardiovascular risk factors, particularly hypertension and diabetes mellitus, but lower frequency of coronary heart disease (CHD) (4–6). It has been suggested that the prevalence of HF and preserved ejection fraction (HFpEF) may be higher in African-Americans than current estimates of HFpEF in the general population (5,7,8). However, findings are mixed regarding survival in African-Americans with HF (3,4,6,9) and information is limited concerning prognosis in African-Americans with HFpEF (10,11). We therefore aimed to describe differences in clinical characteristics, cardiac structure and function, and prognosis in a community based sample of African-Americans with HFpEF as compared to those with HF and reduced ejection fraction (HFrEF) and those without HF.

Methods

Study population

The Atherosclerosis Risk in Communities (ARIC) study is an ongoing, prospective observational study of the natural history of atherosclerotic diseases and cardiovascular risk factors. Detailed study rationale, design, and procedures have been previously published (12). The original cohort was recruited between 1987–1989 using probability sampling of middle aged (45–64 years old) men and women from 4 communities in the United States (Forsyth County, NC; Jackson, MS; Minneapolis, MN; and Washington County, MD). The Jackson field center enrolled an entirely African American cohort. Subsequent follow up visits occurred at 3 year intervals up to 1998, with annual telephone interviews conducted between visits. Institutional review boards from each site approved the study and informed consent was obtained from all participants.

Transthoracic echocardiography was only performed in the Jackson cohort during visit 3 (1993–1995). Of 2,445 participants who underwent transthoracic echocardiography, 1,962 were included in this analysis after sequential exclusion of 318 in whom left ventricular ejection fraction (LVEF) could not be calculated, 32 with missing HF status, 3 in whom incident HF occurred after visit 3 but before the echocardiogram, and 130 with missing covariate data.

Echocardiography

Two-dimensional, M-mode, and Doppler images were acquired with an Acuson 128XP/10c cardiac ultrasound machine with 2.5, 3.5, and 5.0 MHz transducers (Acuson, Malvern, PA). Quality control measures have been previously described (13). Left ventricular (LV) end diastolic diameter (LVEDD), LV end systolic diameter, septal and posterior wall thickness

were measured from 2-dimensional images according to American Society of Echocardiography criteria. LV mass was calculated using the simplified cubed equation and indexed (LVMI) to height (meters$^{2.7}$) with left ventricular hypertrophy (LVH) defined as an LVMI ≥51g/m$^{2.7}$ (14). Relative wall thickness was calculated as 2 × posterior wall thickness/LVEDD with ≤ 0.42 considered normal. LVEF was calculated utilizing 2-D Teichholz method, with a LVEF ≥ 50% and <50% defining preserved and reduced LVEF, respectively. Diastolic dysfunction was determined based upon transmitral Doppler E to A ratio, with ≤ 0.75 or > 1.5 considered abnormal (15).

Definition of Heart Failure

Prevalent HF at visit 3 was defined by a) stage 3 or manifest HF according to Gothenburg criteria or the use of medications for HF at visit 1 (n=82) or b) hospitalization with ICD-9 code for HF (428.x) listed at discharge between visit 1 and 3 (n=34) (3). The Gothenburg criterion is a validated scoring system composed of 3 components a) cardiac, b) pulmonary, and c) therapy, in which stage 3 or manifest HF requires 1 point from each component (16). Participants with quantifiable LVEF by echocardiography and prevalent HF were categorized as HFpEF (LVEF ≥ 50%) or HFrEF (LVEF < 50%).

Covariates

Established definitions for hypertension, obesity, diabetes mellitus, CHD, stroke, smoking status, and medication use as previously described in ARIC were utilized (17). Electrocardiographic LVH was determined by Cornell criteria. Pulmonary disease was defined as self reported history of physician diagnosed lung disease or asthma. Retinopathy, estimated glomerular filtration rate, ankle brachial index, hematologic parameters, lipids, and glucose were measured according to standardized protocols with chronic kidney disease (CKD) and peripheral arterial disease defined as previously described (12,18–20). Ankle brachial index was available in 1,239 participants. All covariates were ascertained at visit 3, except for hemoglobin, white blood cell count, and creatinine, for which the most recent prior measures were used.

Outcome

The primary outcome was death from any cause. The follow up period was defined as the time elapsed from the date of echocardiography to the date of death, date of last contact for those lost to follow-up, or December 31, 2008. Deaths were ascertained through annual phone calls to participants and ongoing surveillance of health department certificate files.

Statistical Methods

For each of the 3 groups (non HF, HFpEF, and HFrEF), summary statistics for covariates were calculated as counts and percentages, and medians and inter-quartile ranges for categorical and continuous data, respectively. Comparisons were then made between a) HFpEF and non-HF and b) HFpEF and HFrEF. Chi squared or Fisher's exact test and Wilcoxon rank-sum test were used to compare baseline characteristics. All cause mortality rates were calculated (number of deaths divided by person-time at risk). Survival analysis was performed according to the Kaplan Meier method with the log-rank test used to assess for differences. Univariable and multivariable hazard ratios for death were estimated using Cox proportional hazards regression. Covariates included in multivariable models included age, gender, LVEF, and clinical characteristics. Propensity scores for HFpEF vs. non HF and HFrEF vs. HFpEF were calculated using clinical characteristics that significantly differed in univariate analyses between groups. The propensity score was then included in Cox proportional hazards models. Two-sided p values <0.05 were considered significant. Analyses were performed using Stata 11.2 (Stata Corp., College Station, Texas).

Results

Prevalent HF was identified in 116 (5.9%) participants and further classified as HFpEF (n=85, 73%) and HFrEF (n=31, 27%). Those with HFpEF were older than those without HF, but similar in age to those with HFrEF. Female sex was significantly more common in HFpEF as compared to those without HF and HFrEF. There were no differences with regard to heart rate or blood pressure. As expected, symptoms of HF, such as orthopnea, paroxysmal nocturnal dyspnea, and lower extremity edema were more common among those with HF as compared to those without HF (Table 1).

Comorbidities were common among those with HFpEF (Table 1). In particular, hypertension, obesity, diabetes mellitus, pulmonary disease, peripheral arterial disease, and CHD were more frequent among those with HFpEF as compared to those without HF. Of note, hypertension was highly prevalent even in those without HF, approaching 60%. Comorbidities were also common among those with HFrEF, with diabetes mellitus, CHD, and CKD present more frequently than in HFpEF.

Cardiac structure and function differed between groups (Table 2). Those with HFpEF had increased wall thickness and LVMI compared to those without HF, although prevalence of diastolic dysfunction and left atrial enlargement did not differ between these two groups. In contrast, those with HFrEF had the highest LV wall thickness and LVMI. Additionally, left atrial enlargement and mitral regurgitation were more frequent among those with HFrEF as compared to those with HFpEF. Concentric hypertrophy was the most common LV geometry; however, it was significantly more frequent in HFpEF as compared to those without HF. Hypertrophy was present in nearly all participants with HFrEF, with eccentric hypertrophy more frequent in HFrEF as compared to HFpEF.

Over a median follow up of 13.7 years, deaths occurred in 21%, 31%, and 61% of those without HF, with HFpEF, and with HFrEF, respectively (Table 3). Death rates in HFpEF were increased compared to those without HF, but were lower than those with HFrEF, even when adjusted for age. In age and gender adjusted Cox Proportional Hazards Models, risk of death differed between groups. HFpEF was associated with a 51% increased risk of death as compared to those without HF (HR 1.51, 95%CI 1.01–2.25). HFrEF was associated with the worst survival, with 2.5 times the risk of death as compared to those with HFpEF (HR 2.50, 95%CI 1.37–4.58). When further adjusted for LVEF, HFpEF was associated with 61% increased risk of death as compared to those without HF (HR 1.61, 95%CI 1.08–2.41). Adjustment for differences in clinical characteristics attenuated the mortality risk associated with HFpEF vs. those without HF; however, those with HFrEF remained at significantly increased risk of death compared to those with HFpEF (Table 3, Figure 1).

Discussion

In a community based sample of middle-aged African-Americans, we found that demographic and clinical characteristics as well as cardiac structure and function significantly differed between HFpEF, HFrEF, and those without HF. By comparing HFpEF to those without HF, we found that older age, female sex, hypertension, obesity, diabetes mellitus, and concentric hypertrophy were more common in HFpEF. Similarly, diabetes mellitus, CKD, CHD, and left atrial enlargement were more common in HFrEF than HFpEF. Survival differed between groups, with HFpEF portending a worse prognosis than those without HF, but not as severe as HFrEF. Together, these findings suggest that in African-Americans HFpEF and HFrEF may be distinct syndromes.

Representation of African-Americans in observational studies and clinical trials is typically low, thus HF in this population is not well understood (21,22). Moreover, few HF studies

focus specifically on African-Americans (23), as most literature involving race in HF addresses differences between racial groups. This is in spite of a higher prevalence of cardiovascular risk factors and greater burden of HF among African-Americans. The existing literature suggests the development and progression of HF in African Americans may be characterized by predominantly non-ischemic etiologies, more severe natural history, and possibly a different response to pharmacotherapy when compared to predominantly Caucasian studies (9,21). By evaluating an entirely African-American cohort, our findings may advance understanding of HF in this high risk population.

Approximately three quarters of African-Americans with HF in our cohort had HFpEF. This is concordant with two population based studies of ambulatory HF patients, where 57–76% had preserved systolic function (7,24), but differs from an ambulatory Veterans Administration population where HFpEF was present in 25% (25). However, only 6% of those in the Veterans Administration study were female, and therefore may not be representative of the typical HFpEF population (25). In comparison, 65% of the Jackson cohort was female, which may be one explanation for the higher prevalence of HFpEF. Our findings are also consistent with population studies of ambulatory HF patients that included multiple ethnicities and demonstrated a higher prevalence of HFpEF than HFrEF (26–28). However, our results contrast with studies evaluating African-Americans hospitalized with acute decompensated HF where HFpEF was prevalent in 29–43% (5,6,29), suggesting the relative frequency of HFpEF and HFrEF may differ between hospitalized and ambulatory settings (30). Additionally, the finding that HFpEF was more common than HFrEF may be explained, in part, by the high prevalence of hypertension and relatively low frequency of CHD.

Regardless of preserved or reduced LVEF, comorbidities were common in HF. However, the pattern of clinical characteristics differed between HFpEF and HFrEF. In addition to diabetes mellitus and CKD, CHD was more frequent in HFrEF as compared to HFpEF, although it was only present in one third of those with HFrEF despite the high prevalence of atherosclerotic risk factors. Overall, CHD was not as common in HF as typically described in predominantly white populations, which may be partially explained by the relatively high proportion of women and middle age range in this study. Hypertension, however, was present in 85% of those with HF. Together, these findings suggest that hypertension, along with other comorbidities, such as obesity, diabetes mellitus, and CKD, rather than CHD, may be relatively more important factors in HF in African-Americans.

The most striking finding related to cardiac structure was the marked prevalence of LVH not only in those with HF, but also among those without HF. While hypertension may be the most common contributor to hypertrophy, other factors including obesity (31,32), diabetes mellitus (33,34), the metabolic syndrome (35), and CKD (36), have previously been demonstrated to be associated with LVH. Several studies have shown that these comorbidities are common in HF particularly among African-Americans and Hispanics (4,37). In our analysis, these comorbidities were frequent in HF, and a particularly worrisome finding was that concentric hypertrophy was present in 60% of those without HF. The high prevalence of hypertension and concentric LVH, a known marker of increased cardiovascular risk in African-Americans (38), portends a potential increase in HF among this group (39).

There is also uncertainty regarding the risk of death in chronic HF among African-Americans (5). Registries of hospitalized HF patients suggest similar or better survival in African-Americans vs. whites (4–6,40). This is congruent with previous reports from ARIC demonstrating similar mortality rates between races at 30 days and 1 year; however, the longer follow up time in ARIC revealed higher fatality rates in African-Americans at 5 years

post HF hospitalization (3). Few studies have evaluated survival in HFpEF in African-Americans. In a predominantly male Veterans Administration population, survival over 5 years of follow up did not differ according to race among those with HFpEF, although among African-Americans HFpEF appeared to be associated with a similar or slightly better prognosis than HFrEF (10). Results from the Duke Databank of Cardiovascular Disease suggest a better 5 year survival in HFpEF (68%) (11), as compared to HFrEF (51%) (41), although a direct comparison of mortality between HFpEF and HFrEF in African-Americans was not made.

We found that HFpEF was associated with a more benign prognosis than HFrEF. This is consistent a meta-analysis of nearly 42,000 patients with HF that demonstrated a 32% lower risk of death in HFpEF as compared to HFrEF, although stratification according to race was not reported (42). It is also consistent with the Candesartan in Heart failure Assessment of Reduction in Mortality and morbidity (CHARM) trial (43), but differs from epidemiologic data from Olmsted County, Enhanced Feedback for Effective Cardiac Treatment, and the Framingham Heart Study where mortality rates were similar between HFpEF and HFrEF (44–46). This may, in part, be explained by differences in study populations, as those that capture patients during or immediately following an acute hospitalization find that HFpEF and HFrEF have similar mortality, particularly among the elderly (30). In contrast, ambulatory HF patients, such as those in the Cardiovascular Health and Strong Heart studies, which enrolled an older biracial population and Native Americans, respectively, demonstrate lower fatality rates in HFpEF vs. HFrEF (26,27). However, representation of African-Americans in these studies was generally low, limiting applicability to this race.

We also found the mortality rate in HFpEF to be greater than that of those without HF. The risk of death in HFpEF was 61% higher than age, gender, and LVEF matched participants without prevalent HF. However, adjustment for additional clinical characteristics attenuated this risk. It has been proposed that HFpEF is a collection of comorbidities and that the syndrome of HFpEF may not even exist (47). In contrast, pooled analyses of clinical trials of HFpEF and cardiovascular trials of patients without HF demonstrate higher mortality rates in HFpEF as compared to those without HF (48). Our data in African-Americans are consistent with these pooled analyses. Although adjustment for comorbidities attenuated this risk, the relatively small numbers of deaths in our HFpEF population limited our statistical power. Nevertheless, our findings highlight the importance of comorbidities in African Americans with HFpEF, but do not mitigate the broader literature demonstrating that HFpEF is associated with a worse prognosis than those without HF.

Recent literature also suggests that half of all deaths in HFpEF may not be related to cardiovascular causes (49–51), again emphasizing the impact of comorbidities (37,52). We extend these findings by showing that non-cardiovascular comorbidities are frequent among African-Americans with HF. Furthermore, using ICD codes (recognizing their limitation in identifying cause of death), we observed a similar trend to previous reports; namely that 58%, 44% and 26% of deaths in those without HF, with HFpEF, and HFrEF, respectively, were due to non-cardiovascular causes (Figure 2). Together these findings emphasize the importance of treating comorbidities, particularly among those with HFpEF, as a potential approach to improving outcomes (37,49,52,53).

While we specifically evaluated an entirely African-American cohort over a long follow up period, limitations should be noted. The cross sectional design precludes assessment of causality between clinical characteristics, cardiac structure and function, and HF. However, differences between HFpEF, HFrEF, and those without HF may provide insight into targets for future investigation. The definition for prevalent HF was based upon Gothenburg criteria and unadjudicated hospitalization ICD-9 codes, although these methods have been validated

in ARIC (3,54). Importantly, this approach captures participants with prior or current symptoms of HF, as recommended by ACC/AHA staging of HF (55). LVEF was not assessed at the time of incident HF and it is possible that LVEF may have recovered between the incident HF event and our assessment. However, LVEF has previously been demonstrated to be similar between acute and chronic HF (56). Moreover, our findings suggest LVEF measured after incident HF still imparts prognostic information. Teichholz' method was used to calculate LVEF as volumetric measurements were not available. Diastolic function was assessed with transmitral Doppler E/A ratio as estimation of left atrial volumes, tissue Doppler imaging, transmitral E wave deceleration time, pulmonary venous flow, and isovolumic relaxation time were not obtained at echocardiography. While diastolic dysfunction is frequently reported in HFpEF, we found it to be present in 25% of participants with HFpEF, which did not differ between HFpEF and those without HF, likely reflecting limitations in diastolic assessment. There may have been selection bias with regard to participants who presented for echocardiography or had interpretable images, such that the sickest individuals, including more severe HF, may be underrepresented. Inherent to ARIC's design, this study includes a selected age range and consisted entirely of African-Americans from Jackson. Therefore, our results may not be generalizable to younger, more elderly, or all African-Americans. However, the mean age of African-Americans in most HF registries was 63–64 years old, falling within the range of our cohort (4–6,25). The observed mortality rates may not be directly applicable to a more contemporary time period due to temporal changes in HF management. Finally, the relatively low numbers of participants with prevalent HF may limit statistical power.

In summary, we found in a community based sample of middle-aged African-Americans, that demographic and clinical characteristics, as well as cardiac structure and function, significantly differed between HFpEF, HFrEF, and those without HF. HFpEF was more common than HFrEF and portended a worse prognosis than those without HF, but not as severe as HFrEF. As this population bears a disproportionate burden of HF, focused investigation on African-Americans is an important step to understanding HF and developing strategies for prevention, detection, and treatment.

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Abbreviations

HF	heart failure
HFpEF	heart failure with preserved ejection fraction
HFrEF	heart failure with reduced ejection fraction
ARIC	Atherosclerosis Risk in Communities
LV	Left ventricular
LVEF	Left ventricular ejection fraction
LVEDD	Left ventricular end diastolic diameter

LVMI	Left ventricular mass index
CHD	Coronary heart disease
CKD	Chronic kidney disease
LVH	Left ventricular hypertrophy

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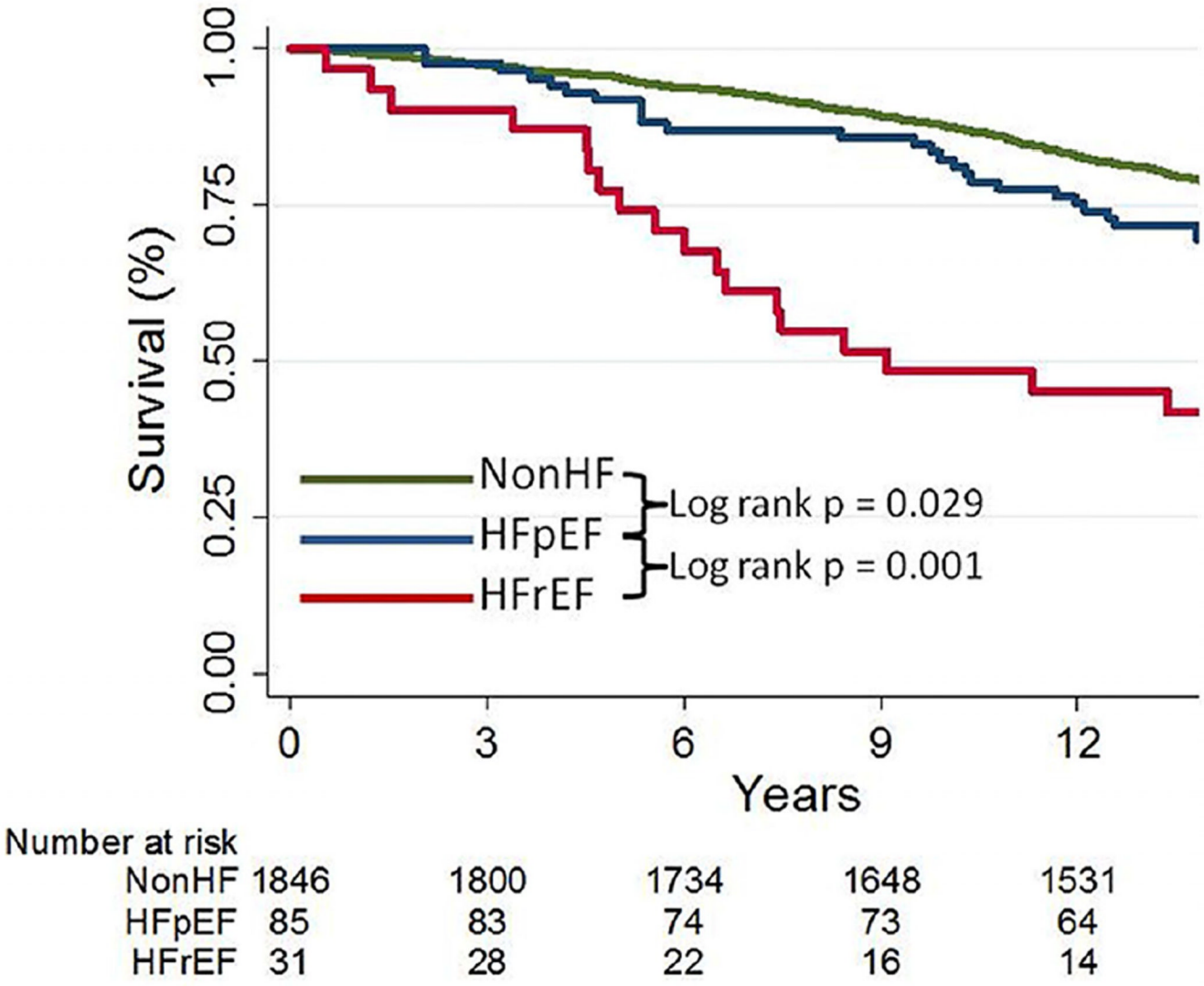

Figure 1. Survival in African-Americans according to heart failure status

Kaplan-Meier survival analysis in those without heart failure (nonHF), heart failure with preserved ejection fraction (HFpEF), and those with heart failure and reduced ejection fraction (HFrEF).

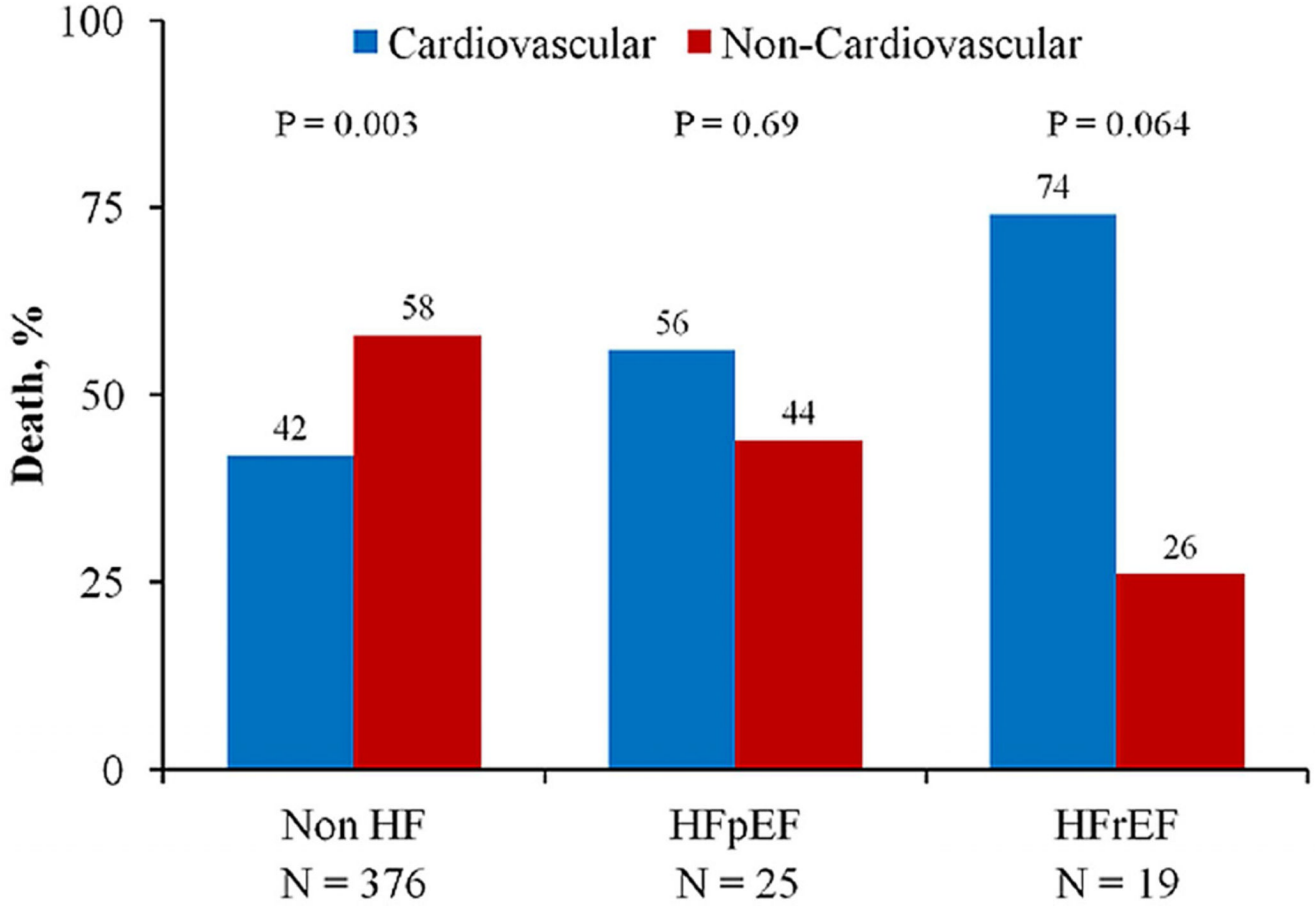

Figure 2. Proportion of deaths due to cardiovascular and non-cardiovascular causes according to heart failure status in African-Americans

Cause of death ascertained from ICD-9 codes. HFpEF = heart failure with preserved ejection fraction, HFrEF = heart failure with reduced ejection fraction, nonHF = no heart failure.

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Table 1

Characteristics of the Jackson participants of the ARIC cohort who underwent echocardiography stratified according to heart failure status.

	HFpEF N = 85	**Non HF N = 1846**	***NonHF vs. HFpEF p-value***	**HFrEF N = 31**	***HFrEF vs. HFpEF p-value***
Age, years	61.1 (57.1,66.5)	58.5 (54.6,63.8)	0.001	62.0 (54.9,65.7)	0.89
Gender (Female)	72 (85)	1156 (63)	<0.001	20 (65)	0.036
Orthopnea	27 (32)	175 (9)	<0.001	13 (42)	0.38
PND	18 (21)	116 (6)	<0.001	12 (39)	0.09
Self reported LE edema	55 (65)	588 (32)	<0.001	16 (52)	0.28
Hypertension	72 (85)	1088 (59)	<0.001	26 (84)	1.00
Obesity	60 (71)	831 (45)	<0.001	19 (61)	0.37
Diabetes mellitus	36 (42)	409 (22)	<0.001	21 (68)	0.021
Pulmonary Disease	23 (27)	129 (7)	<0.001	5 (16)	0.33
Peripheral Arterial Dis.	8 (14)	62 (5)	0.013	2 (10)	1.00
Coronary Heart Dis.	11 (13)	69 (4)	0.001	10 (32)	0.027
Chronic Kidney Dis.	1 (1)	55 (3)	0.72	8 (27)	<0.001
Stroke	4 (5)	42 (2)	0.14	5 (16)	0.056
Current Tobacco Use	10 (12)	371 (20)	0.07	4 (13)	1.00
Current Alcohol Use	17 (20)	579 (31)	0.03	5 (16)	0.79
Aspirin	50 (59)	852 (46)	0.026	13 (42)	0.14
Lipid Lowering Med	4 (5)	74 (4)	0.77	4 (13)	0.21
Anti Hypertensive Med	74 (87)	910 (49)	<0.001	28 (90)	0.76
Beta blockers	12 (14)	163 (9)	0.10	4 (13)	0.87
ACE Inhibitor	17 (20)	165 (9)	0.002	14 (45)	0.009
Diuretic	49 (58)	412 (22)	<0.001	21 (68)	0.39
Digoxin	8 (9)	20 (1)	<0.001	11 (36)	0.002
Heart Rate, bpm	68 (64,74)	68 (64,72)	0.32	72 (66,78)	0.20
Systolic BP, mmHg	130 (120,141)	128 (117,142)	0.42	135 (122,154)	0.14
Diastolic BP, mmHg	74 (68,81)	76 (70,83)	0.13	72 (64,92)	0.76
Pulse Pressure, mmHg	52 (45,65)	52 (43,63)	0.15	60 (51,72)	0.06
Body mass index, kg/m^2	32 (29,37)	29 (26,33)	<0.001	34 (27,40)	0.97

	HFpEF N = 85	**Non HF N = 1846**	***NonHF vs. HFpEF p-value***	**HFrEF N = 31**	***HFrEF vs. HFpEF p-value***
Total cholesterol, mg/dL	206 (182,227)	204 (180,231)	0.88	210 (184,245)	0.24
LDL cholesterol, mg/dL	124 (105,154)	126 (102,151)	0.85	124 (104,156)	0.88
HDL cholesterol, mg/dL	53 (41,64)	54 (43,66)	0.41	54 (42,66)	0.55
Triglycerides, mg/dL	110 (85,150)	98 (74,134)	0.007	117 (84,157)	0.85
WBC	5.3 (4.5,6.3)	5.1 (4.2,6.3)	0.25	6.4 (5.2,7.2)	0.019
Hemoglobin, g/dL	12.6 (11.9,13.3)	13.1 (12.2,13.9)	0.003	12.5 (11.7,13.4)	0.69
Creatinine, mg/dL	0.86 (0.76,0.96)	0.86 (0.76,1.06)	0.003	0.96 (0.86,1.26)	<0.001
eGFR, ml/min/1.73m^2	93 (79,114)	90 (79,103)	0.66	78 (59,100)	0.006
Glucose, mg/dL	109 (96,156)	102 (94,118)	0.004	124 (97,203)	0.30
LVH on ECG (Cornell),%	3 (4)	125 (7)	0.37	8 (26)	0.001
QRS duration, msec	94 (87,103)	93 (86,101)	0.39	102 (92,109)	0.042
Advanced retinopathy,%	17 (20)	222 (12)	0.037	12 (39)	0.07
Ankle Brachial Index	1.05 (0.97,1.13)	1.10 (1.03,.1.17)	0.002	1.03 (0.99,1.14)	0.99

Data presented as counts (percentages) and medians (interquartile ranges) for categorical and continuous variables, respectively. BMI = body mass index, BP = blood pressure, bpm = beats per minute, Dis = disease, ECG = electrocardiogram, eGFR = estimated glomerular filtration rate, HDL = high density lipoprotein, LDL = low density lipoprotein, LE = lower extremity, LVH = left ventricular hypertrophy, Med = medication, PND = paroxysmal nocturnal dyspnea, WBC = white blood cell. Obesity defined as BMI ≥ 30 kg/m^2.

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Table 2

Cardiac structure and function according to heart failure status and left ventricular ejection fraction among Jackson participants of the ARIC study.

	HFpEF N = 85	**Non HF N = 1846**	***NonHF vs. HFpEF p-value***	**HFrEF N = 31**	***HFrEF vs. HFpEF p-value***
LVEF, %	67 (59,75)	64 (56,71)	0.002	39 (28,44)	<0.001
FS 2D, %	37 (31,44)	34 (29,40)	0.002	19 (13,22)	<0.001
Mitral E vel., cm/s	76 (66,89)	76 (65,87)	0.50	79 (63,92)	0.52
Mitral A vel., cm/s	80 (67,91)	75 (64,88)	0.13	96 (72,106)	0.001
Mitral E/A	0.94 (0.79,1.12)	1.00 (0.84,1.19)	0.14	0.75 (0.66,1.10)	0.054
Diastolic Dysfunction,%	23 (27)	460 (25)	0.70	22 (71)	<0.001
SWT, cm	1.22 (1.09,1.32)	1.16 (1.04,1.29)	0.028	1.27 (1.12,1.34)	0.35
PWT, cm	1.21 (1.09,1.36)	1.17 (1.06,1.31)	0.057	1.27 (1.20,1.41)	0.07
LVEDD, cm	4.35 (4.07,4.67)	4.34 (3.98,4.72)	0.76	5.43 (4.73,6.08)	<0.001
LVESD, cm	2.60 (2.42,2.99)	2.82 (2.45,3.20)	0.014	4.32 (3.59,5.32)	<0.001
LV Mass, g	239 (202,285)	230 (188,276)	0.10	366 (304,436)	<0.001
LVMI, $g/m^{2.7}$	63 (51,74)	56 (47,68)	0.002	92 (75,101)	<0.001
LVH (LVMI ≥$51 g/m^{2.7}$)	64 (75)	1183 (64)	0.037	29 (94)	0.035
Relative Wall Thickness	0.57 (0.51,0.62)	0.54 (0.47,0.62)	0.057	0.48 (0.42,0.56)	<0.001
RWT ≥ 0.42	79 (93)	1668 (90)	0.57	23 (74)	0.01
LA Diameter, cm	3.4 (3.1,3.8)	3.4 (3.0,3.7)	0.12	3.8 (3.3,4.3)	0.008
LA enlargement (>4cm)	16 (19)	240 (13)	0.14	12 (39)	0.048
Aortic Root Diameter, cm	3.1 (2.8,3.3)	3.0 (2.8,3.2)	0.08	3.0 (2.8,3.3)	0.85
≥ Mod Regurgitation					
Mitral	0 (0)	13 (1)	1.00	3 (10)	0.02
Aortic	1 (1)	9 (1)	0.35	0 (0)	1.00
Tricuspid	2 (2)	29 (2)	0.65	1 (3)	1.00
LV Geometry					
Normal	4 (5)	80 (4)	0.79	1 (3)	1.00
Concentric Remodeling	17 (20)	583 (32)	0.023	1 (3)	0.039
Concentric Hypertrophy	62 (73)	1085 (59)	0.009	22 (71)	0.82
Eccentric Hypertrophy	2 (2)	98 (5)	0.32	7 (23)	0.001

Data presented as counts (percentages) and medians (interquartile ranges) for categorical and continuous variables, respectively. FS = fractional shortening, LA = left atrium, LV = left ventricular, LVEDD = left ventricular end diastolic diameter, LVEF = left ventricular ejection fraction, LVESD = left ventricular end systolic diameter, LVMI = left ventricular mass index, Mod = moderate PWT = posterior wall thickness, RWT = relative wall thickness, SWT = septal wall thickness.

Table 3

Mortality rates and risk of death according to heart failure status and left ventricular ejection fraction in African-Americans.

	Non HF N=1846	**HFpEF N=85**	**HFrEF N=31**
Deaths and mortality rates			
Deaths, n (%)	393 (21)	26 (31)	19 (61)
Person-Time (yrs)	23,707	1,032	291
Deaths / 100 person yrs * (95% CI)	1.66 (1.50,1.83)	2.52 (1.72,3.70)	6.52 (4.16,10.22)
Hazard ratios (95% CI)		*HFpEF vs. Non HF*	*HFrEF vs. HFpEF*
Unadjusted		1.55 (1.04,2.30)	2.62 (1.45,4.75)
Age, gender adjusted		1.51 (1.01,2.25)	2.50 (1.37,4.58)
Age, gender, LVEF † adjusted		1.61 (1.08–2.41)	n/a
Age, gender, LVEF †, Propensity score ‡ adjusted		1.35 (0.87–2.09)	2.29 (1.19–4.42)

* Death rates standardized to median age (58.7 years) of cohort.

† LVEF not included in comparison of HFrEF vs. HFpEF

‡ Propensity score calculated utilizing baseline characteristics that significantly differed between groups.

LVEF = Left ventricular ejection fraction