Research Article

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Racial differences in patients referred for right heart catheterization and risk of pulmonary hypertension

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Abstract

African Americans (AA) have a higher incidence of pulmonary hypertension (PH) risk factors. Few studies have examined the racial differences in the prevalence and etiology of PH and direct comparison of invasive hemodynamics between AAs and Caucasians has rarely been reported. In this study, we examined whether racial differences exist in patients referred for right heart catheterization (RHC) and hypothesized that AA race is an independent risk factor for PH and is associated with increased adjusted mortality. We extracted data for AA and Caucasian patients who underwent RHC at Vanderbilt between 1998 and 2014. Clinical information was obtained from Vanderbilt's Synthetic Derivative, a de-identified mirror of our Electronic Medical Record. A total of 4576 patients were analyzed, including 586 (13%) AAs and 3990 (87%) Caucasians. AAs were younger than Caucasians by an average of eight years, but had more prevalent heart failure, features of metabolic syndrome, and higher creatinine. AAs also had higher mean pulmonary artery pressure and pulmonary vascular resistance. After adjusting for relevant co-morbidities, the AA race is associated with 41% increased risk of PH (odds ratio [OR] = 1.41, 95% confidence interval [CI] = 1.12–1.79). Among patients with PH, AA race is associated with 24% increased adjusted mortality (hazard ratio [HR] = 1.24, 95% CI = 1.09–1.45). AAs were younger but had more prevalent cardiometabolic and renal disease and worse pulmonary hemodynamics. The AA race is an independent risk factor for PH. Among patients with PH, the AA race is associated with increased adjusted mortality. Future studies should focus on delineating whether genetic or environmental factors contribute to PH risk in AAs.

Keywords

pulmonary hypertension, racial, ethnic, or social disparities in lung disease and treatment, health disparities

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Pulmonary hypertension (PH) is common, especially among women and the elderly, and is associated with increased morbidity and mortality.[1–7] PH can develop in isolation or as sequelae to a heterogeneous group of chronic conditions.[8–11]

Despite growing knowledge about the epidemiology and pathogenesis of PH, few studies have examined racial differences in the prevalence and etiology of PH. African Americans (AAs) have a higher prevalence of risk factors for PH such as heart failure, hypertension, and diabetes and develop these co-morbidities at a younger age.[12–15] In addition, AAs experience worse cardiovascular and pulmonary clinical outcomes.[1,16–19] It is unknown whether these risk factors translate into a higher prevalence of PH or associated mortality among AAs, as most epidemiologic studies of PH have been performed in racially homogenous populations, precluding direct racial comparisons.[20–25]

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We recently reported higher systolic pulmonary artery pressure by transthoracic echocardiography (TTE) in middle-aged AA participants of the Coronary Artery Risk Development in Young Adults Study (CARDIA) compared to Caucasians.[26] However, direct comparison of invasive hemodynamics between AAs and Caucasians referred for right heart catheterization (RHC) has rarely been reported.

In this study, we sought to examine if there were racial differences in PH prevalence, etiology, and prognosis in a large referral population at a tertiary academic medical center. We hypothesized that race is an independent risk factor for PH and that the AA race is associated with more adverse invasive hemodynamics and mortality.

Methods

Study population

The Vanderbilt University Institutional Review Board (no. 140544) approved this study, which consisted of adult patients referred for RHC in both inpatient and outpatient settings at Vanderbilt University Medical Center from 1998 to 2014. The indications for RHC are presented in Suppl. Table 1. All data were extracted from Vanderbilt's Synthetic Derivative, a de-identified mirror image of the Vanderbilt Electronic Medical Record that contains detailed patient information for over 2.5 million unique individuals.[27,28] RHC hemodynamic data were extracted from procedural reports and manually validated for accuracy.[29–31] For patients who had more than one RHC at Vanderbilt, only data from their first RHC were analyzed. Exclusion criteria and data cleaning procedures for this dataset have been described previously.[30,31] In brief, we excluded individuals with shock, hypertensive crisis, or complex congenital heart disease. In addition, patients with insufficient clinical data (absent pulmonary artery or pulmonary artery wedge pressure [PAWP]), previous cardiac (*International Classification of Diseases, Ninth Revision* [ICD-9] code V42.1) or lung (ICD-9 code V42.6) transplantation, acute myocardial infarction (ICD-9 code 410.*), or chronic pulmonary embolism (ICD-9 code 416.2) were excluded. Non-physiologic values (e.g. negative cardiac output) were systematically deleted and missing data were imputed for the purposes of regression analyses.[29–32]

PH classification

PH etiology was classified according to contemporary guidelines (Suppl. Table 2).[10] Briefly, PH was defined by mean pulmonary artery pressure (mPAP) ≥ 25 mmHg at rest. Pulmonary arterial hypertension (PAH) was defined as PH with mean PAWP ≤ 15 mmHg and pulmonary vascular resistance (PVR) > 3 Wood Units (WU). Individuals who met hemodynamic criteria for PAH underwent manual chart review to confirm or exclude the clinical diagnosis of PAH. PH due to left heart disease included isolated post-capillary PH (Ipc-PH) and combined pre- and post-capillary PH (Cpc-PH), defined as PH with PAWP > 15 mmHg and diastolic pressure gradient of <7 mmHg and ≥ 7 mmHg, respectively.[9] World Health Organization (WHO) group 3 PH was defined as PH with PAWP ≤ 15 mmHg, prevalent chronic obstructive pulmonary disease (COPD) (ICD-9 codes 491.* and 492.*) or interstitial lung disease (ICD-9 codes 516.*), and absence of another etiology on manual review.[11]

Clinical data

From the Synthetic Derivative, we extracted patient demographics, co-morbidities, medication exposure, laboratory values, and TTE data. Patient demographics such as age, gender, and race were obtained from administrative records. Co-morbidities were defined by a combination of ICD-9 coding and laboratory values or previously validated algorithms and included relevant cardiac, pulmonary, metabolic, and renal disease.[33] Medications were restricted to those on an individual's medication list in the six months before RHC to avoid including those prescribed after hemodynamics were obtained. Echocardiographic data were extracted from the TTE performed closest in date to RHC; the median elapsed time between RHC and TTE was one day (interquartile range [IQR] = 2–19 days).[34] TTE parameters such as left atrial enlargement, defined as anterior–posterior left atrial diameter > 40 mm, and left ventricular hypertrophy, defined as left ventricular posterior wall thickness ≥ 12 mm, were determined per established guidelines.[35,36] Co-morbidities, laboratory values, and echocardiographic data were restricted to within six months before or after RHC.

Outcomes

The primary outcome was the independent association between race and PH and the secondary outcomes were the independent association of race with PH survival and phenotype. Variables were selected a priori based on clinical knowledge of established or suspected risk factors for PH. For the survival analysis, the Synthetic Derivative is linked to the Social Security Death Index, which is continuously updated and used to determine vital status. Follow-up time was calculated from date of RHC to either date of death or 1 June 2016 (last date of censor).

Statistical analysis

Categorical variables were expressed as absolute values and percentages. Continuous variables were expressed as mean $\pm$ standard deviation and effect size of continuous variables were reported based on an increment from the 25th to 75th percentile value to provide more clinical insight than a standard deviation. Baseline characteristics were compared using Chi-squared test for categorical variables

and Wilcoxon rank-sum test for continuous variables. To assess the association between race and PH in patients referred for RHC, we used a multivariate logistic regression model, adjusting for age, gender, heart failure, hypertension, diabetes, COPD, interstitial lung disease, body mass index (BMI), creatinine, left ventricular ejection fraction (LVEF), and left ventricular hypertrophy. To determine survival, a Cox proportional hazards model was built. Because heart failure, hypertension, and diabetes violated the proportional hazards assumption, a stratified Cox model was fit with respect to these variables. Prevalent interstitial lung disease, LVEF, and left ventricular hypertrophy were removed in the survival analysis to avoid overfitting based on priority of likely confounders.

Results

Baseline characteristics

After pre-specified exclusions (n = 1221), the final cohort for analysis included 4576 patients, of which 3990 (87%) were Caucasians and 586 (13%) were AAs (Fig. 1). At the date of first RHC, AAs were younger than Caucasians by an average of eight years (53 ± 14 vs. 61 ± 15, $P < 0.001$). AAs also had higher rates of heart failure (62% vs. 45%), more features of metabolic syndrome such as hypertension (88% vs. 77%), diabetes (46% vs. 36%), and obesity (39% vs. 33%), and higher creatinine (1.8 ± 2.1 mg/dL vs. 1.2 ± 0.9 mg/dL; Table 1). In addition, AAs were more likely to be prescribed antihypertensive or diuretic medication. AAs and Caucasians did not differ in the prevalence of COPD, interstitial lung disease, or obstructive sleep apnea. There was also no difference in administration of PH-specific mediations between AAs and Caucasians.

Pulmonary hypertension distribution

In our referral population, AAs were more likely to have PH than Caucasians (73% vs. 57%, $P < 0.001$). The distribution of PH by etiology is shown in Fig. 2. Compared to Caucasians, AA patients had increased prevalence of Cpc-PH (13% vs. 7%, $P < 0.001$), PAH (16% vs. 11%, $P < 0.001$), and WHO group 3 PH (6% vs. 4%, $P = 0.02$), whereas there was no difference in the prevalence of Ipc-PH between groups ($P = 0.21$).

Invasive and hemodynamics and echocardiographic data

AAs had more severe pulmonary hemodynamics compared with Caucasians. AAs had higher right atrial pressure (10 ± 7 mmHg vs. 8 ± 6 mmHg), mPAP (34 ± 14 mmHg vs. 29 ± 14 mmHg), mean PAWP (17 ± 9 mmHg vs. 15 ± 8 mmHg), and PVR (4.0 ± 3.3 WU vs. 3.2 ± 3.3 WU, all $P < 0.001$; Table 2). AAs also had lower cardiac index (2.8 ± 1.0 L/min/m^2 vs. 2.9 ± 1.0 L/min/m^2) and mixed venous oxygen saturation ($63\% \pm 11\%$ vs. $68\% \pm 10\%$). On TTE, AAs had lower LVEF ($40\% \pm 19\%$ vs. $47\% \pm 16\%$) and higher estimated right ventricular systolic pressure (52 ± 23 mmHg vs. 46 ± 21 mmHg), with similar rates of left atrial enlargement and left ventricular hypertrophy.

Associations with prevalent PH

To investigate the association between race and PH diagnosis, we built a logistic regression model incorporating the following clinically relevant variables a priori: age, gender, heart failure, hypertension, diabetes, COPD, interstitial lung disease, BMI, creatinine, LVEF, and left ventricular hypertrophy. In our fully adjusted model, the AA race was independently associated with prevalent PH (odds ratio

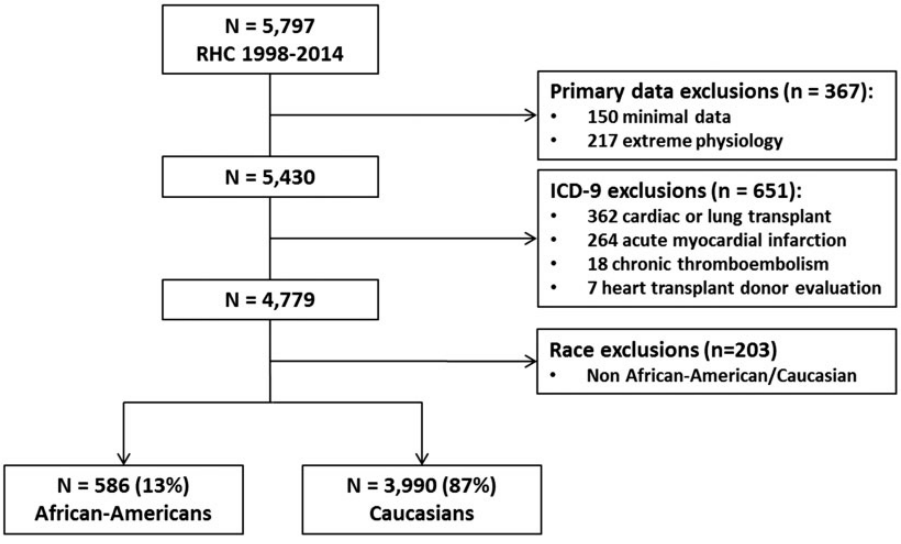

Fig. 1. Flow chart of patients referred for right heart catheterization. A schematic depiction of our study cohort. RHC, right heart catheterization; ICD-9, *International Classification of Diseases, 9th Revision*.

Table 1. Baseline characteristics of patients referred for RHC.

Characteristic	African Americans (n = 587)	Caucasians (n = 3990)	*P* value
Age (years)	53 ± 14	61 ± 15	<0.001
Male gender (n (%))	272 (46)	2063 (52)	0.02
Hypertension (n (%))	514 (88)	3069 (77)	<0.001
Coronary artery disease (n (%))	388 (66)	2894 (73)	0.002
Heart failure (n (%))	366 (62)	1778 (45)	<0.001
Atrial fibrillation (n (%))	122 (21)	1240 (31)	<0.001
Chronic obstructive pulmonary disease (n (%))	73 (12)	487 (12)	0.86
Interstitial lung disease (n (%))	39 (7)	247 (6)	0.66
Obstructive sleep apnea (n (%))	64 (11)	406 (10)	0.58
Systemic lupus erythematous (n (%))	27 (5)	41 (1)	<0.001
Scleroderma (n (%))	2 (2)	109 (3)	0.34
Diabetes mellitus (n (%))	267 (46)	1452 (36)	<0.001
Obesity (n (%))	229 (39)	1304 (33)	0.02
Brain natriuretic peptide (pg/mL)	948 ± 1254	562 ± 861	<0.001
Creatinine (mg/dL)	1.8 ± 2.1	1.2 ± 0.9	<0.001
Hemoglobin A1c (%)	6.4 ± 1.5	6.1 ± 1.2	<0.001
Body mass index (kg/m^2)	31 ± 8	30 ± 7	0.007
Low density lipoprotein (mg/dL)	92 ± 35	92 ± 39	0.72
High density lipoprotein (mg/dL)	46 ± 19	43 ± 17	0.003
Triglycerides (mg/dL)	119 ± 100	161 ± 162	<0.001
Medications (n (%))			
Any antihypertensive	459 (78)	2844 (71)	<0.001
Any diuretic	398 (68)	2165 (54)	<0.001
Any anticoagulant	196 (33)	1243 (31)	0.26
Any lipid-lowering agent	238 (41)	1998 (50)	<0.001
*Pulmonary hypertension medications**			
Prostacyclins	12 (2)	95 (2)	0.62
Endothelin receptor antagonists	14 (2)	84 (2)	0.66
Phosphodiesterase inhibitors	22 (4)	107 (3)	0.14
Any	36 (6)	225 (6)	0.62

*Medications limited to six months before date of RHC.

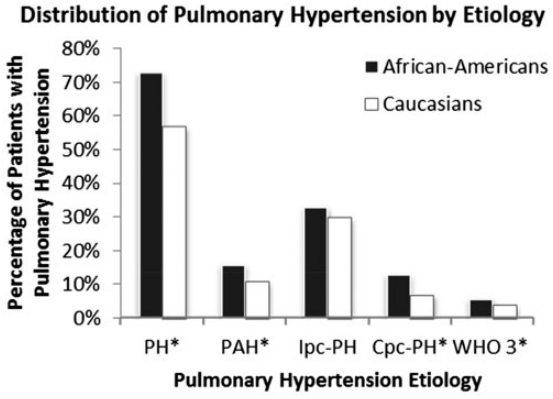

Fig. 2. Bar graph showing distribution of PH by etiology, expressed as percentage of total number of African Americans (AAs, n = 586) and Caucasians (n = 3990) in our cohort. AAs had higher prevalence of PH (73% vs. 56%), PAH (16% vs. 11%), Cpc-PH (13% vs. 7%), and WHO group 3 PH (6% vs. 4%), but not Ipc-PH (33% vs. 30%). *$P < 0.05$.

[OR] = 1.41, 95% confidence interval [CI] = 1.12–1.79), along with heart failure (OR = 2.45, 95% CI = 2.10–2.89), diabetes (OR = 1.18, 95% CI = 1.01–1.38), COPD (OR = 1.42, 95% CI = 1.13–1.77), and interstitial lung disease (OR = 1.66, 95% CI = 1.23–2.22, Fig. 3). In subgroup analysis, the AA race was independently associated with Cpc-PH ($P = 0.007$), PAH ($P = 0.02$), and WHO group 3 PH ($P = 0.02$), but not Ipc-PH ($P = 0.11$; Table 3). Full multivariable analysis results are reported in Suppl. Table 3.

Survival analysis

The median follow-up was 4.7 years (IQR = 2.8–8.6 years). In total, 255 (44%) AAs and 1465 (37%) Caucasians died during follow-up. In unadjusted analysis, AA race was associated with decreased survival (log rank test, $P < 0.001$) in all-comers at eight-year follow-up (Fig. 4a) but not in

Table 2. Invasive hemodynamics and transthoracic echocardiography data.

	All patients (n = 4576)			Patients with PH (n = 2689)		
	AAs (n = 586)	Caucasians (n = 3990)	*P* value	AAs (n = 428)	Caucasians (n = 2261)	*P* value
Invasive hemodynamics						
RA pressure (mmHg)	10 ± 7	8 ± 6	<0.001	12 ± 7	11 ± 6	0.002
RV systolic pressure (mmHg)	52 ± 21	45 ± 20	<0.001	60 ± 20	57 ± 19	0.003
Mean PAP (mmHg)	34 ± 14	29 ± 14	<0.001	40 ± 11	38 ± 11	<0.001
Mean PAWP (mmHg)	17 ± 9	15 ± 8	<0.001	19 ± 9	19 ± 8	0.79
DPG (mmHg)	6 ± 9	4 ± 9	<0.001	8 ± 10	6 ± 11	<0.001
PVR (Woods units)	4.0 ± 3.3	3.2 ± 3.3	<0.001	4.8 ± 3.5	4.3 ± 3.9	<0.001
Cardiac index* (L/min/m^2)	2.8 ± 1.0	2.9 ± 1.0	<0.001	2.6 ± 0.9	2.7 ± 0.9	<0.001
PA oxygen saturation (%)	63 ± 11	68 ± 10	<0.001	61 ± 11	64 ± 10	<0.001
Non-invasive hemodynamics						
RV systolic pressure (mmHg)	52 ± 23	46 ± 21	<0.001	56 ± 24	51 ± 21	0.03
TRV (m/s)	3.3 ± 1.4	3.1 ± 0.8	<0.001	3.5 ± 1.5	3.3 ± 0.8	0.22
LA enlargement† (n (%))	299 (58)	1911 (58)	0.85	229 (61)	1263 (65)	0.15
LV hypertrophy‡ (n (%))	168 (33)	1041 (31)	0.58	126 (33)	634 (32)	0.72
LV ejection fraction§ (%)	40 ± 19	47 ± 16	<0.001	40 ± 19	45 ± 17	0.001

*By Fick's method.
†Anterior-posterior LA diameter > 40 mm.
‡LV posterior wall thickness ≥ 12 mm.
§By biplane Simpson's method.
RA, right atrial; RV, right ventricular; PAP, pulmonary arterial pressure; PAWP, pulmonary arterial wedge pressure; DPG, diastolic pressure gradient; PVR, pulmonary vascular resistance; AA, African American; PH, pulmonary hypertension; TRV, tricuspid regurgitant velocity; LA, left atrial; LV, left ventricular.

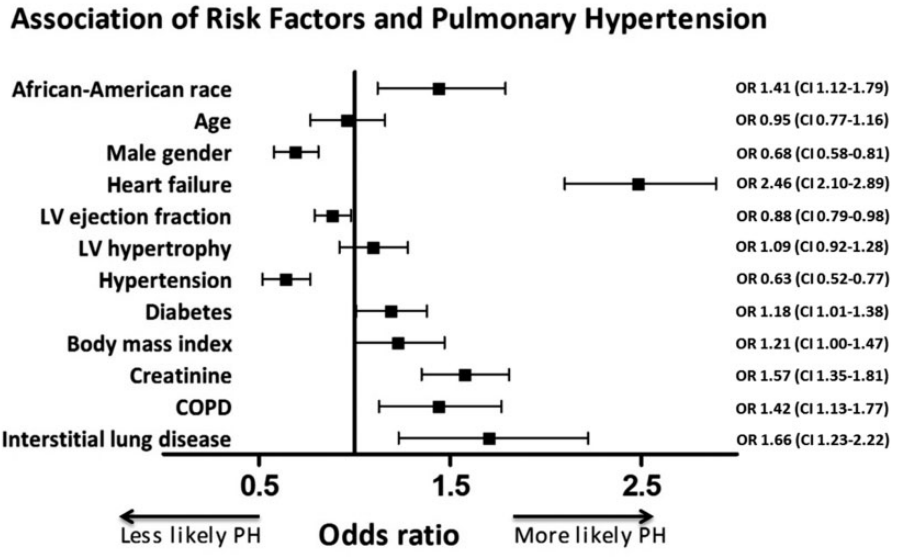

Fig. 3. Forest plot of association of risk factors and PH. All continuous variables were reported based on an increment from the 25th to 75th percentile value. AA race, heart failure, diabetes, COPD, interstitial lung disease, and higher BMI and creatinine were risk factors for PH. Male gender and higher LVEF were protective against PH. OR, odds ratio; 95% CI, confidence interval.

Table 3. Adjusted association of African American race and pulmonary hypertension.

	All PH (n = 2689)	PAH (n = 564)	Ipc-PH (n = 1456)	Cpc-PH (n = 364)	WHO 3 PH (n = 214)
Odds ratio	1.41	1.47	0.83	1.54	2.01
Confidence interval	1.12–1.79	1.08–2.02	0.66–1.04	1.12–2.11	1.11–3.64

PH, pulmonary hypertension; PAH, pulmonary arterial hypertension; Ipc-PH, isolated post-capillary PH; Cpc-PH, combined pre- and post-capillary PH; WHO 3, World Health Organization group 3.

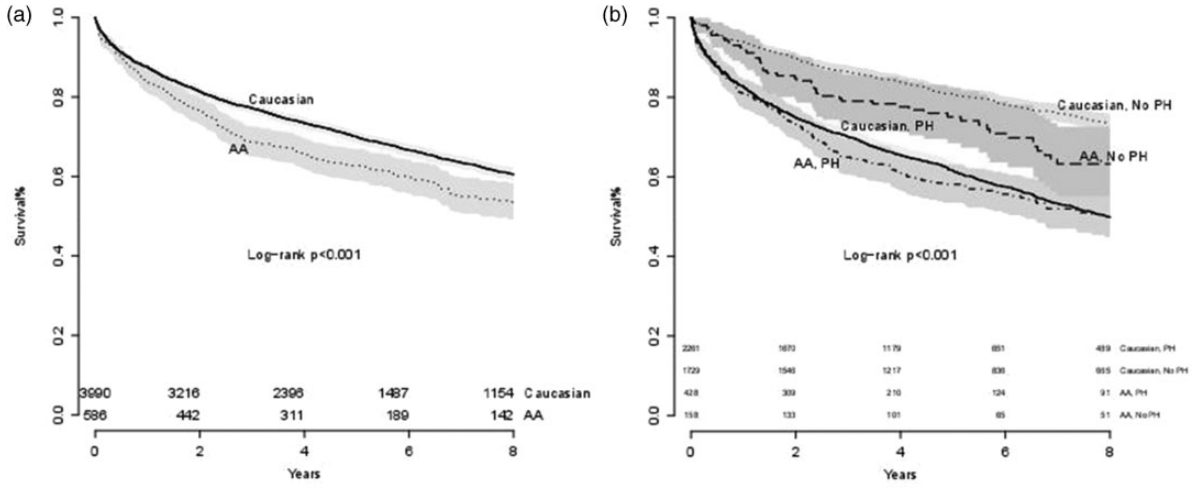

Fig. 4. Kaplan-Meier curves for all patients referred for RHC (a) and in patients with and without PH (b). At eight-year follow-up, the AA race was associated with decreased survival (log rank test, $P < 0.001$) in all-comers but not in patients with PH.

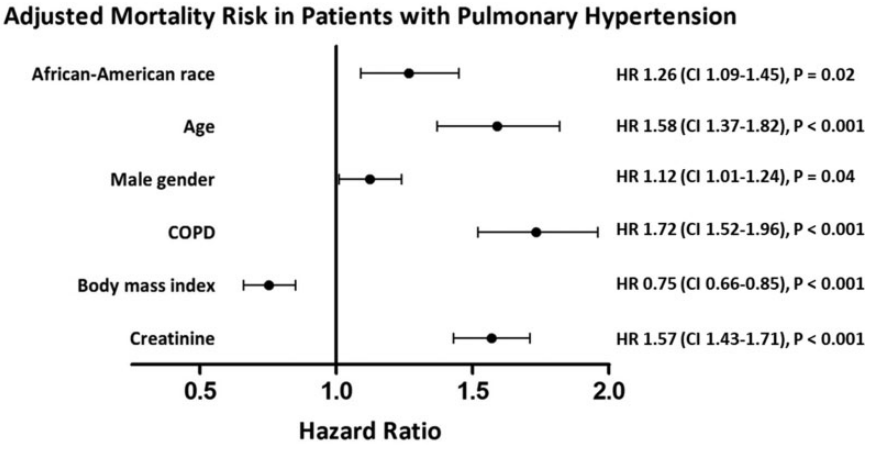

Fig. 5. Forest plot of association of co-morbidities and mortality in patients with PH using Cox proportional hazards model, stratified by hypertension, heart failure, and diabetes and adjusted for race, age, gender, COPD, BMI, and creatinine. Among patients with PH, the AA race was associated with 26% increased adjusted mortality. HR, hazard ratio; CI, confidence interval.

patients with hemodynamic PH (Fig. 4b). In the fully adjusted, multivariate stratified Cox model of patients with PH, AA race was associated with increased mortality (hazard ratio [HR] = 1.24, 95% CI = 1.09–1.45; Fig. 5). In exploratory analyses, race was not an independent predictor of mortality for any specific PH etiology (Suppl. Table 4).

Discussion

In this study of patients referred for RHC, we found that AAs were younger than Caucasians by an average of eight years but had more prevalent cardiometabolic and renal disease and more severe pulmonary hemodynamics. After adjusting for relevant co-morbidities, AA race was associated with 41% increased risk for PH and 24% increased mortality among patients with PH. Given the higher prevalence of PH and associated mortality among AAs, these findings are important to AAs with PH risk factors and their physicians.

There are limited published data examining racial differences in PH. Retrospective analysis of the National Hospital Discharge Survey showed differential mortality trends between Caucasians and AAs with PH during 2001–2010.[1,4] However, they relied on ICD-9 codes for all medical diagnoses which can be inaccurate and lacked invasive hemodynamic data. Contemporary registries tend to focus on patients with PAH, excluding other etiologies of PH.[20–23,25,37] The Registry to Evaluate Early and Long-term Pulmonary Arterial Hypertension Disease Management reported decreased survival for Caucasians compared to AAs at five-year follow-up; however, patients with WHO group 2 and 3 PH were not included and invasive hemodynamics between races were not directly compared.[38] Two recent studies have drawn attention to the importance

of echocardiographic estimates of systolic PAP in AAs. Chaudhry et al. found that 7% of patients in the Jackson Heart Study had echocardiographic evidence of PH, which was associated with increased heart failure hospitalizations.[22,39] In the biracial CARDIA study, we found that AAs had higher systolic PAP after adjusting for demographics and left ventricular structure and function.[26] The generalizability of the findings from CARDIA is unknown because it consists of relatively healthy individuals, not a clinical referral population. Our study aimed to address some of the current gaps in literature by examining PH prevalence, etiology, and prognosis in a large heterogeneous population referred for invasive hemodynamic evaluation.

We found that AAs had higher PAPs and higher prevalence of PH. Although our study was not designed to uncover the mechanisms behind this observation, several factors may contribute. Jankowich et al. found that higher circulating endothelin-1, a potent vasoconstrictor, is associated with higher systolic PAP and mortality in AA patients of the Jackson Heart Study.[40] Compared with Caucasians, AAs with PAH also have reduced response to treatment by endothelin receptor antagonists[41] and AAs with systemic hypertension, a risk factor for PH, appear to release more endothelin-1 in response to stress, have almost threefold higher absolute circulating endothelin-1 levels, and have impaired endothelial function and vasodilation.[42–44] Therefore, increased endothelin-1 signaling may contribute to higher pulmonary pressure in AAs. Next, we recently have identified shared genetic variants between Caucasian patients with PAH and Cpc-PH.[31] Given the much higher prevalence of Cpc-PH among AAs compared to Caucasians in this study, we speculate that AAs may be genetically more predisposed to develop PAH in response to left heart disease. Moreover, lower lung volumes among AAs appear to contribute to higher estimated systolic PAP on echocardiography.[26] Finally, socioeconomic status modifies PH risk factors such as heart failure, hypertension, and diabetes and tends to be worse in AAs.[12–15] Recently, Parikh et al. reported that AAs had a twofold increased risk of death compared with Caucasians in a smaller cohort ($n = 45$ AAs) referred specifically for PH evaluation. After adjusting for health insurance status, mortality did not differ between AAs and Caucasians, suggesting that socioeconomic status may influence outcomes.[45] Our study built on this work by analyzing a larger sample size including all patients undergoing RHC, regardless of indication, to increase the generalizability of our findings. We also adjusted for additional PH risk factors and echocardiographic measures of LV structure and function. These observations suggest that molecular, genetic, and environmental factors may all predispose AAs to PH and warrant further study to shed insight on PH pathophysiology or to identify at-risk individuals.[46]

We found that race was not independently associated with Ipc-PH, whereas the prevalence of Cpc-PH among AAs was double that of Caucasians. While AAs had higher prevalence of heart failure and lower mean LVEF, other adverse cardiac remodeling parameters such as left ventricular hypertrophy and left atrial enlargement were similar between races. AAs with PH also had similar PAWP as Caucasians with PH (Table 2), suggesting that more prevalent PH and worse pulmonary hemodynamics seen in AAs are not solely explained by more severe cardiac disease. Consistent with other studies showing pulmonary vascular remodeling in patients with Cpc-PH,[47] we speculate that AAs may represent an at-risk population who are more susceptible to pulmonary arterial remodeling in response to left atrial hypertension. Therefore, AAs should be an important subgroup included in clinical trials targeting patients with Cpc-PH and future studies using biological samples are required to test this hypothesis.

Finally, we found that among patients with PH, AA race was independently associated with decreased survival after adjusting for relevant co-morbidities, building upon prior studies that showed increased mortality among AAs with idiopathic PAH.[18,19] PH is often diagnosed late in its disease course, due to under-recognition and overlapping symptoms with other conditions.[1,2] Studies show that AAs with heart failure have more co-morbidities and higher rates of hospitalization, which could be in part due to concurrent PH.[12,14,47–51] Therefore, our observations have important clinical implications regarding the frequency of PH surveillance and the degree of risk factor modification in at-risk AAs. Of note, we did not observe any differences in adjusted mortality in individual PH categories. However, a positive point estimate towards increased mortality was seen in the Ipc-PH and Cpc-PH groups (HR = 1.12, 95% CI = 0.88–1.43 and HR = 1.21, 95% CI = 0.80–1.81, respectively; Suppl. Table 4). In addition to validating these findings in a larger, external cohort, future work should include longitudinal studies to investigate disease trajectory and response to treatment.

Limitations

There are several limitations to this study. First, our cross-sectional, single-centered study involves a referral population, so we are unable to comment on the prevalence and etiology of PH by race in the general community. Second, our dataset does not allow us to comment on socioeconomic or health insurance status, but we acknowledge these are potentially important determinants of outcomes in patients with PH. Third, similar to other large Electronic Medical Record-based studies, co-morbidity information was largely obtained from ICD-9 coding, which can be inaccurate. However, we used algorithms for co-morbidity ascertainment that have high accuracy in our system or have been validated in the Electronic Medical Records and Genomics network.[33] Finally, we were unable to review RHC waveforms from the Vanderbilt Synthetic Derivative to confirm hemodynamic measurements, but we have previously shown strong agreement between manually reviewed values and

computer generated mean values in our catheterization laboratory.[30]

Conclusion

AAs referred for invasive hemodynamic evaluation at a large tertiary care center are younger than Caucasians and have a higher co-morbidity burden. The AA race is an independent risk factor for PH and is associated with a more severe pulmonary hemodynamic profile. Finally, among patients with PH, the AA race is associated with increased adjusted mortality. These findings warrant future studies to delineate whether genetic or environmental factors contribute to PH risk in AAs.

Conflict of interest

The author(s) declare that there is no conflict of interest.

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