Cardiology in the Young

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Left ventricle hypertrophy and re-modeling in children with essential hypertension: does the race matter?

Daiji Takajo[1], Thomas S. Przybycien[2], Preetha L. Balakrishnan[2], Girija Natarajan[3], Gautam K. Singh[2] and Sanjeev Aggarwal[2]

[1]Department of Pediatrics, Children's Hospital of Michigan, Detroit, MI, USA; [2]Division of Pediatric Cardiology, Department of Pediatrics, Children's Hospital of Michigan, Detroit, MI, USA and [3]Division of Neonatal & Perinatal Medicine, Department of Pediatrics, Children's Hospital of Michigan, Detroit, MI, USA

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Corresponding author:
D. Takajo; Email: daiji.takajo@cchmc.org

Abstract

Background: This is the first study to report on the impact of race on differences in the prevalence of echocardiographic left ventricular hypertrophy and left ventricular adaptation at the time of diagnosis of essential hypertension in children. *Methods:* This cross-sectional, single-centre study included patients aged 3–18 years who had newly diagnosed essential hypertension. Echocardiography was used to assess left ventricular mass index and left ventricular relative wall thickness. An left ventricular mass index > the 95th percentile for age and gender, and an left ventricular relative wall thickness > 0.42, were used to diagnose left ventricular hypertrophy and concentric adaptation. Various echocardiographic parameters were compared between African Americans and Caucasians. *Results:* The study included 422 patients (289 African Americans and 133 Caucasians) diagnosed with essential hypertension at a median age of 14.6 (interquartile range; 12.1–16.3) years. Eighty-eight patients (20.9%) had left ventricular hypertrophy. There was no statistically significant difference in the prevalence of left ventricular hypertrophy between African Americans and Caucasians (22.5% versus 17.3%, p=0.22). The median left ventricular relative wall thickness was 0.35 (0.29–0.43), and 114 patients (27.0%) had an left ventricular relative wall thickness > 0.42. The presence of an left ventricular relative wall thickness > 0.42 was significantly higher among African Americans compared to Caucasians (30.1% versus 20.3%, p = 0.04). The African American race was a strong predictor for an left ventricular relative wall thickness > 0.42 (odds ratio 1.7, p = 0.04), but not for left ventricular mass index > the 95th percentile (p = 0.22). Overweight/obesity was a strong predictor for an left ventricular mass index > the 95th percentile. *Conclusions:* There was no difference in the prevalence of left ventricular hypertrophy in children with essential hypertension of different races. Obesity, rather than being African American, is associated with left ventricular hypertrophy.

The prevalence of hypertension in children and adolescents under 19 years of age has been reported to be 4.0% and has been increasing over the past two decades.[1] This secular increase in the prevalence of hypertension has coincided with the concurrent obesity epidemic in children in the United States.[2] Along with obesity, hypertension in children is reported to be more prevalent in African American and Hispanic populations. A national survey in the United States from 1963 to 2002 revealed that the prevalence of hypertension in children was the highest in Mexican American (4.6%), followed by in African American (4.2%), and in Caucasian (3.3%) populations.[3] Similar observations were reported in a Texas school-based screening programme from 2000 to 2015 revealing a prevalence of hypertension of 3.1% in Hispanic, 2.7% in African American, 2.6% in Caucasian, and 1.7% in Asian adolescents (aged between 10 and 19 years).[4]

Left ventricle hypertrophy occurs in systemic hypertension as a remodelling adaptation to maintain contractile forces and counteract increased ventricular wall stress due to altered loading conditions.[5] However, increased wall stress may cause activation of cytokines and other molecular pathways[6] leading to apoptosis, myocardial fibrosis, and architectural changes. Therefore, left ventricular hypertrophy could have counterintuitive effects and may result in a decline in systolic reserve capacity and diastolic function.[7] Therefore, according to the American College of Cardiology/American Heart Association heart failure classification, left ventricular hypertrophy constitutes stage A (at high risk for developing heart failure without structural heart disease) of heart failure with potential to progress to stage B (structural heart disease but without symptoms of heart failure) and stage C (structural heart disease with prior or current symptoms of heart failure).[8]

It has been increasingly recognised that children with hypertension can develop left ventricular hypertrophy in their youth. As the presence of left ventricular hypertrophy in

children with hypertension suggests end-organ damage, echocardiograms are regularly performed to monitor the development of left ventricular hypertrophy. The reported prevalence of left ventricular hypertrophy among children with hypertension varies from 14 to 41%.[9–13] However, data on racial differences in the prevalence of left ventricular hypertrophy in children with essential hypertension is lacking. Since blood pressure in childhood is highly predictive of BP in adulthood (the tracking of BP),[14,15] the presence of hypertension in childhood could persist in adulthood. Therefore, knowledge of racial prevalence of left ventricular hypertrophy in children constitutes an important public health need.

The primary aim of this study is to evaluate the impact of race on the prevalence of left ventricular hypertrophy among children with essential hypertension. The secondary aim is to evaluate if there are any racial differences in left ventricular remodelling in children with essential hypertension. The further secondary aim is to evaluate the impact of confounding factor of obesity on the development of left ventricular hypertrophy and left ventricular adaptation.

Methods

Study design

This study was a retrospective, single-center study approved by the Central Michigan University Institutional Review Board. The study included patients aged 3–18 years who were diagnosed with essential hypertension, according to the defined criteria (given under Definition section), in an outpatient setting between 2010 and 2020.

Patients were identified by searching diagnostic codes (ICD-9; 401.1 or 401.9, ICD-10; I-10) in our hospital medical records. Patients who had been discharged from the emergency department or inpatient settings with these codes were excluded, as patients with hypertension in the setting of acute illness might be due to secondary hypertension. Following the screening with diagnostic codes, individual medical records were reviewed. Our exclusion criteria included patients diagnosed with (1) secondary hypertension, (2) any congenital heart diseases, (3) congenital genitourinary anomalies, (4) renal diseases and chronic renal insufficiency, (5) unknown self-reported race, (6) lack of availability of echocardiogram shortly before or after the diagnosis of essential hypertension, and (7) patients < 3 years of age or > 18 years at the time of diagnosis of essential hypertension.

The demographic data collected included age at diagnosis, age at echocardiogram, gender, height and weight, and self-reported race. The first echocardiogram at the time of diagnosis was evaluated for various left ventricular measurements to calculate the left ventricle mass and left ventricular hypertrophy. Screening laboratory findings were also collected if available.

Definition

Blood pressure measurements were taken using a DINAMAP PROCARE 400 monitor (GE Healthcare, UK) after the patients rested for three to five minutes in a sitting position, using an appropriate cuff size that covered 80–100% of the arm's circumference and a width that covered at least 40%.[16] The patients' blood pressure was categorised according to the 2017 American Academy of Pediatrics guideline, with normal blood pressure defined as < 90th percentile for children aged < 13 years and < 120/80 mmHg for children aged ≥ 13 years.[16] Hypertension was defined as any elevation in blood pressure more than 90th percentile for age, gender, and height for children aged < 13 years and ≥ 120/80 mmHg for children aged ≥ 13 years. Essential hypertension was defined as hypertension in which secondary causes such as renovascular disease, renal failure, or other causes of secondary hypertension were excluded.[17] In our institution, screening laboratory workup, including chemistry panel (electrolytes, blood urea nitrogen, and creatinine), renal ultrasound, urinalysis, and lipid profile, was performed in all patients with hypertension to exclude secondary hypertension. For other potential causes of hypertension, such as endocrine-related factors, further laboratory tests are conducted on a case-by-case basis. The age at diagnosis of hypertension was determined as the age when the patients were first presented to our nephrology or cardiology clinic after being referred by their primary care physician following confirmation of multiple abnormal blood pressure measurements at their office.

Body mass index was calculated as kg/m^2, where kg represents the patient's weight and m^2 is height in meters squared. The body mass index percentile was determined based on the Centers for Disease Control and Prevention growth charts for children and teens ages 2 through 19 years.[18]

Self-reported race was collected by asking the patients about their race during their first hospital visit, with options including African American, Asian, Caucasian, Middle Eastern, Hispanic/Latino, interracial, or not known. The answer was recorded in the patient's medical chart as personal information.

Echocardiogram

A single reader (DT) who was blinded to the clinical and demographic data, evaluated all eligible patients' echocardiograms at the time of their hypertension diagnosis. The echocardiograms were performed by certified and experienced cardiac sonographers using phase-arrayed appropriate probe for the patient's age and body habitus interfaced with Philips Sono IE 33 echocardiographic equipment (Philips, Andover MA), and stored for offline analysis. Echocardiograms at our centre are performed in accordance with the American Society of Echocardiography guidelines.[19] To assess of left ventricular mass and relative wall thickness, left ventricular end-diastolic linear dimensions (left ventricular end-diastolic dimension, left ventricular wall thickness, and interventricular septal thickness) were measured using M-mode echocardiography from the parasternal short-axis view below the mitral valve leaflets. The measurements were performed using leading edge-to-leading edge method and were averaged from three measurements. To assess the intra- and inter-observer variability, 20 echocardiograms were re-analyzed by the same investigator and a second investigator (SA).

Left ventricular mass

The left ventricular mass was calculated using the standard formula: left ventricular mass (g) = 0.8 * 1.04 (IVSD + LVIDD + PWTD)^3-LVIDD^3] + 0.6g, where IVSD is interventricular septum, LVIDD is left ventricular internal dimensions, and PWTD is posterior wall thickness. All measurements were performed at end-diastole.[20] Left ventricular mass index was calculated as left ventricular mass/height (m)$^{2.7}$. Left ventricular hypertrophy was defined as left ventricular mass index > 95th percentile for gender and age. For children ≥ 9 years old, the 95th percentile for left ventricular mass index was 40 g/m$^{2.7}$ for females and 45 g/m$^{2.7}$ for males.[21] For children

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< 9 years old, age and gender-specific 95th percentile cut-off values were used, as reported by Khoury et al.[21]

Left ventricle relative wall thickness

Left ventricular relative wall thickness is a measure used to assess various geometric adaptations or remodelling of the left ventricle in response to hypertension. It was calculated from the standard formula: LVRWT = 2 * PWTD / LVIDD. An left ventricular relative wall thickness > 0.42 was considered abnormal.[20] Different left ventricular geometry responses to hypertension were defined as follows: concentric hypertrophy (left ventricular mass index > 95th percentile and left ventricular relative wall thickness > 0.42), concentric re-modeling (left ventricular mass index ≤ 95th percentile and left ventricular relative wall thickness > 0.42), eccentric re-modeling (left ventricular mass index > 95th and left ventricular relative wall thickness ≤ 0.42), and normal left ventricular geometry when both left ventricular mass index and left ventricular relative wall thickness were normal (Supplemental Figure 1).

Statistical analysis

The statistical analysis was performed using SPSS version 28 (IBM SPSS Inc., Chicago, IL). Based on normality tests (Kolmogorov-Smirnov and Shapiro-Wilk), it was determined that there were no normally distributed continuous variables. Therefore, continuous variables were reported as median and interquartile range, while categorical variables were denoted by number and percentage. Due to the small sample size of other races, only African American and Caucasian races were included in the analysis. All pre-specified demographic and echocardiographic parameters between the groups were analysed using the Mann-Whitney U test and Chi-Square test, as appropriate. Univariate and multivariate logistic regression analysis were used to identify the significant parameters to predict left ventricular mass index > 95th percentile and left ventricular relative wall thickness > 0.42. A $p < 0.05$ was considered statistically significant. Inter-rater and intra-rater reliability were assessed by means of the Shrout-Fleiss intra-class correlation coefficient to determine consistency among raters.[22]

Results

Patient population

A total of 422 patients, including 297 males (70.4%), were enrolled in the study, comprising 289 African American (68.5%) and 133 Caucasians (31.5%) (Supplemental Figure 2). The demographics and clinical data of the entire cohort are represented in Table 1. For the entire cohort, the median body mass index and body mass index percentile were 29.4 (22.7–37.2) kg/m^2 and 98th (85–99) percentile, respectively. Laboratory tests were performed on 322 patients (76.3%) at the time of diagnosis, and the median values of serum haemoglobin A1c, total cholesterol, triglyceride, high-density lipoprotein, and low-density lipoprotein were all within normal limits (Table 1).

When comparing demographic data between two races, height (170 versus 166, p = 0.02), weight (86 versus 76, p = 0.002), and body mass index (31.2 versus 27.9, p = 0.026) were significantly higher among African American than Caucasian patients. Serum creatinine level and haemoglobin A1c were significantly higher in African American patients than Caucasians (creatinine; 0.77 versus 0.67, p < 0.001, haemoglobin A1c; 5.8 versus 5.4, p < 0.001).

Diagnosis of hypertension

The median age at the diagnosis of hypertension in the study population was 14.6 (12.1–16.3) years. The median systolic blood pressure and diastolic blood pressure for the entire cohort were 140 (131–149) mmHg and 76 (68–83) mmHg, respectively. Of the total patients, 54 (12.8%) had elevated blood pressure, 135 (32.0%) had Stage 1 hypertension, and 233 (55.2%) stage 2 (Supplemental Table 1). The systolic and diastolic blood pressure were not significantly different between African American and Caucasian patients (systolic blood pressure; 140 versus 140, p = 0.485, diastolic blood pressure; 76 versus 74, p = 0.290) (Table 1). The relationship between blood pressure and age, gender, race, and body mass index is shown in Figure 1. Most patients had an echocardiogram performed before starting anti-hypertensive medication, but 10 patients (2.4%) were already on medication at the time of diagnosis by their primary care physician. The median age at echocardiogram was 14.8 (12.5–16.4) years, and the median interval between the diagnosis of hypertension and the initial echocardiogram was 18 (4–48) days.

Left ventricular mass index, left ventricular relative wall thickness (Table 2)

The median left ventricular mass for the entire group was 138 (107–181) g, and the median left ventricular mass index was 35.2 (29.1–41.9) g/m$^{2.7}$. Left ventricular hypertrophy, as defined by left ventricular mass index > 95th percentile, was present in 88 patients (20.9%). The median left ventricular relative wall thickness was 0.35 (0.29–0.43), and 114 patients (27.0%) had left ventricular relative wall thickness > 0.42. The left ventricular adaptation to essential hypertension was characterised as normal, concentric hypertrophy, concentric remodelling, and eccentric remodelling patterns in 259 (61.4%), 39 (9.2%), 75 (17.8%), 49 (11.6%) patients, respectively.

Although there was no statistically significant difference in left ventricular mass index (g/m$^{2.7}$) between the African American and Caucasian groups (35.4 versus 34.6, p = 0.24), African American patients had a higher left ventricular relative wall thickness (0.37 versus 0.32, p < 0.001) and a higher percentage presence of left ventricular relative wall thickness > 0.42 (30.1% versus 20.3%, p = 0.035) than Caucasians. Therefore, concentric adaptation (concentric remodelling and concentric hypertrophy) was more prevalent among African Americans than Caucasians. The presence of left ventricular mass index > 95th percentile was not significantly different between the two groups (22.5% versus 17.3%, p = 0.222).

Comparison between two races with stratification according to body mass index (Supplemental Table 2, Figure 2, Supplemental Figure 3)

The study further examined the potential impact of overweight and obesity on left ventricular mass index and left ventricular relative wall thickness by dividing the entire cohort into two groups: a group with normal body mass index and a group with overweight/obesity. The analysis of left ventricular mass, left ventricular mass index, and left ventricular relative wall thickness for subgroups of African American and Caucasian patients is presented in *Supplemental Table 2*. The results showed no significant difference in left ventricular mass index between African American and Caucasian patients with normal body mass index (31.2 versus 29.3, p = 0.617) or with overweight/obesity (37.1 versus 36.1, p = 0.243). However, left ventricular relative wall

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Table 1. Demographics and comparison between two groups of African American race vs. Caucasian race.

Median (interquartile range) or number (%)	All (n = 422)	AA patients (n = 289)	Caucasian patients (n = 133)	p value
Age at echocardiogram (years)	14.8 (12.5–16.4)	14.8 (12.9–16.4)	14.8 (11.8–16.2)	0.288
Age at diagnosis of essential HTN (years)	14.6 (12.1–16.3)	14.6 (12.7–16.3)	14.6 (11.5–16.0)	0.141
Gender (male)	297 (70.4)	203 (70.2)	94 (70.7)	0.93
Height (cm)	**169 (156–177)**	**170 (159–178)**	**166 (151–176)**	**0.020**
Weight (kg)	**80 (60–110)**	**86 (62–116)**	**76 (59–95)**	**0.002**
BMI (kg/m^2)	**29.4 (22.7–37.2)**	**31.2 (22.8–38.8)**	**27.9 (22.5–33.7)**	**0.026**
BMI percentile	98 (85–99)	98 (85–99)	97 (86–99)	0.449
Normal	98 (23.2)	67 (23.2)	31 (23.3)	0.193
Overweight	55 (13.0)	31 (10.7)	24 (18.0)	
Obesity	264 (62.6)	187 (64.7)	77 (57.9)	
Underweight	5 (1.2)	4 (1.4)	1 (0.8)	
Severe obesity[¶]	192 (45.5)	139 (48.1)	53 (39.8)	0.114
Systolic blood pressure	140 (131–149)	140 (131–149)	140 (133–151)	0.485
Diastolic blood pressure	76 (68–83)	76 (69–84)	74 (67–82)	0.290
Blood pressure category				
Elevated blood pressure	54 (12.8)	35 (12.1)	19 (14.3)	0.329
Stage 1 HTN	135 (32.0)	99 (34.3)	36 (27.1)	
Stage 2 HTN	233 (55.2)	155 (53.6)	78 (58.6)	
Serum creatinine	**0.70 (0.59–0.86)**	**0.77 (0.60–0.90)**	**0.67 (0.49–0.79)**	**<0.001**
HbA1c level*	**5.7 (5.4–5.9)**	**5.8 (5.6–6.1)**	**5.4 (5.2–5.6)**	**<0.001**
Prediabetes (5.7-6.4)	**40 (9.5)**	**37 (12.8)**	**3 (2.3)**	**0.002**
Diabetes (≥6.5)	4 (0.9)	4 (1.4)	0 (0.0)	0.284
Total cholesterol level	156 (137–177)	155 (137–177)	162 (137–177)	0.713
Triglyceride level	86 (65–139)	78 (62–108)	114 (84–166)	0.006
HDL	46 (39–52)	46 (38–53)	45 (39–49)	0.580
LDL	93 (75–108)	92 (72–103)	95 (77–111)	0.295

AA = African American; BMI = body mass index; HbA1c = haemoglobin A1c; HDL = high-density lipoprotein; HTN = hypertension; LDL = low-density lipoprotein; LV = left ventricular, LVRWT = left ventricle relative wall thickness.
*HbA1c level was available in 75 out of 422 patients only.
[¶]Severe obesity was defined by body mass index≥120% of 95th percentile or body mass index≥35.

thickness was significantly higher among African American patients than Caucasian patients for both with normal body mass index (0.37 versus 0.29, $p < 0.01$) and the overweight/obesity group (0.37 versus 0.33, $p = 0.006$). No significant difference was observed in left ventricular geometry response to essential hypertension between African American and Caucasian patients in either the normal body mass index or overweight/obesity groups. In addition, the study performed gender stratification in combination with body mass index, and the results remained consistent in all the echocardiographic parameters except for male patients in the overweight/obesity group (Supplemental Table 3).

Univariate logistic regression model to predict left ventricular hypertrophy (Supplemental Table 4)

The study conducted univariate logistic regression analysis to predict left ventricular mass index > 95th percentile and left ventricular relative wall thickness > 0.42. For left ventricular mass index > 95th percentile, the analysis identified body mass index percentile as the only significant variable (Odds ratio 1.04, $p < 0.001$). For left ventricular relative wall thickness > 0.42, the analysis identified African American (Odds ratio 1.69, $p = 0.036$) and age at diagnosis of hypertension (Odds ratio 1.10, $p = 0.012$) as significant variables in univariate regression. The final multivariable model confirmed that the significant predictors for left ventricular relative wall thickness > 0.42 were African American (Odds ratio 1.614, $p = 0.059$) and age at diagnosis of hypertension (Odds ratio 1.098, $p = 0.016$).

Reproducibility

The inter-rater reliability between rater 1 (DT) and 2 (SA) was assessed using six variables in 20 randomly selected echocardiograms, resulting in a total of 120 measurements. The two-way random model analysis showed an average intra-class correlation coefficient of 0.970. Similarly, the intra-rater reliability of rater 1 (DT) was assessed using the same six variables in 20 randomly selected echocardiograms, also resulting in 120 measurements. The

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Table 2. Echocardiography parameters and comparison between African American and Caucasian patients.

Median (interquartile range) or number (%)	All (n = 422)	AA patients (n = 289)	Caucasian patients (n = 133)	p value
LV mass (g)	138 (107–181)	143 (111–188)	129 (97–157)	0.004
LVMI (g/m$^{2.7}$)	35.2 (29.1–41.9)	35.4 (29.0–43.2)	34.6 (29.3–39.6)	0.284
LVRWT	**0.35 (0.29–0.43)**	**0.37 (0.31–0.44)**	**0.32 (0.27–0.40)**	**<0.001**
LVMI>95th percentile (LVH)	88 (20.9)	65 (22.5)	23 (17.3)	0.222
LVRWT>0.42 (concentric adaptation)	**114 (27.0)**	**87 (30.1)**	**27 (20.3)**	**0.035**
LV geometry				
Normal	259 (61.4)	169 (58.5)	90 (67.7)	0.143
Concentric re-modeling	75 (17.8)	55 (19.0)	20 (15.0)	
Eccentric hypertrophy	49 (11.6)	33 (11.4)	16 (12.0)	
Concentric hypertrophy	39 (9.2)	32 (11.1)	7 (5.3)	

AA = African American; BMI = body mass index; LV = left ventricular; LVH = left ventricle hypertrophy; LVMI = LV mass index; LVRWT = left ventricle relative wall thickness.

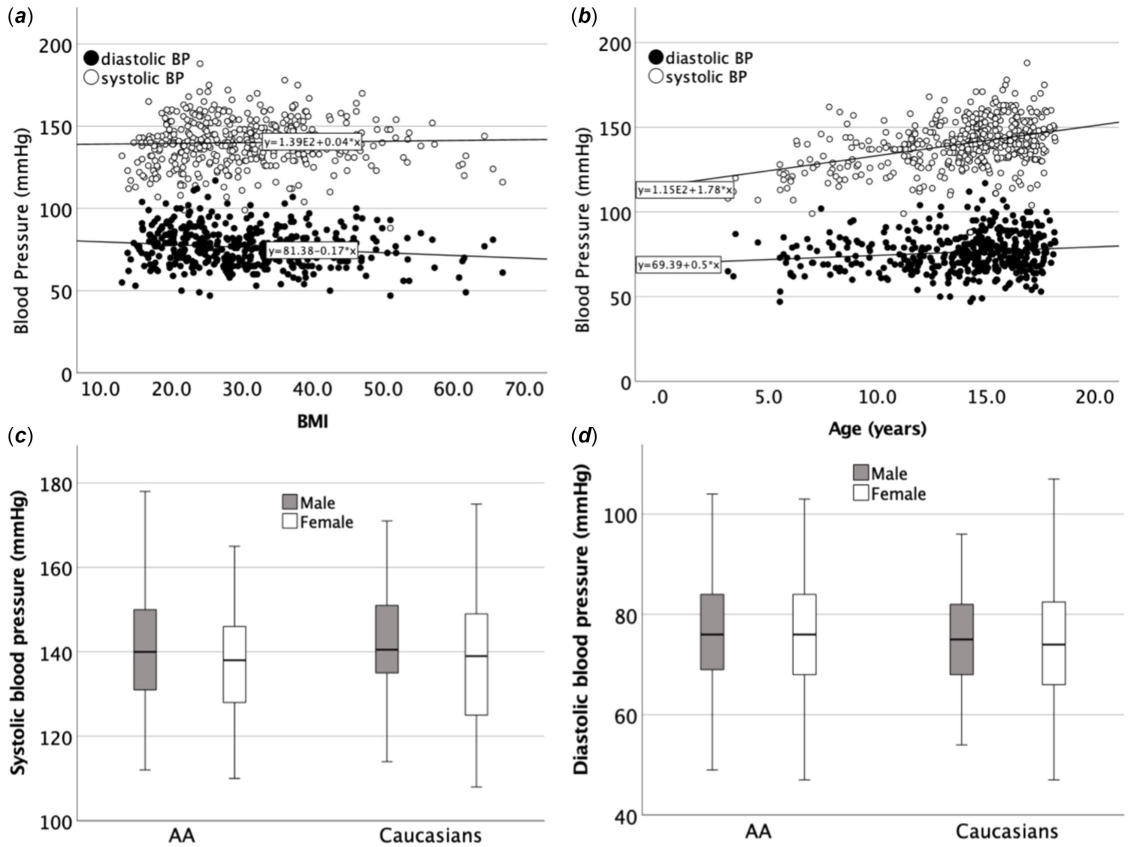

Figure 1. (***a***) Scatterplots showing the relationship between body mass index and systolic/diastolic pressure; (***b***) Scatterplots showing the relationship between age and systolic/diastolic pressure; (***c***) Boxplot showing the systolic blood pressure in African American and Caucasian patients; and (***d***) Boxplot showing the diastolic blood pressure in African American and Caucasian patients.

one-way random model analysis revealed an average intra-class correlation coefficient of 0.979.

Discussion

Our study did not find any differences in the prevalence of left ventricular hypertrophy at the time of essential hypertension diagnosis between African American and Caucasian paediatric populations. However, we did observe a significantly higher prevalence of concentric adaptation of left ventricle in African Americans compared to Caucasians with essential hypertension.

We found that nearly 20% of children with essential hypertension had left ventricular hypertrophy at the time of initial presentation, emphasising the importance of obtaining an echocardiogram to manage essential hypertension. The prevalence of left ventricular hypertrophy among children with hypertension

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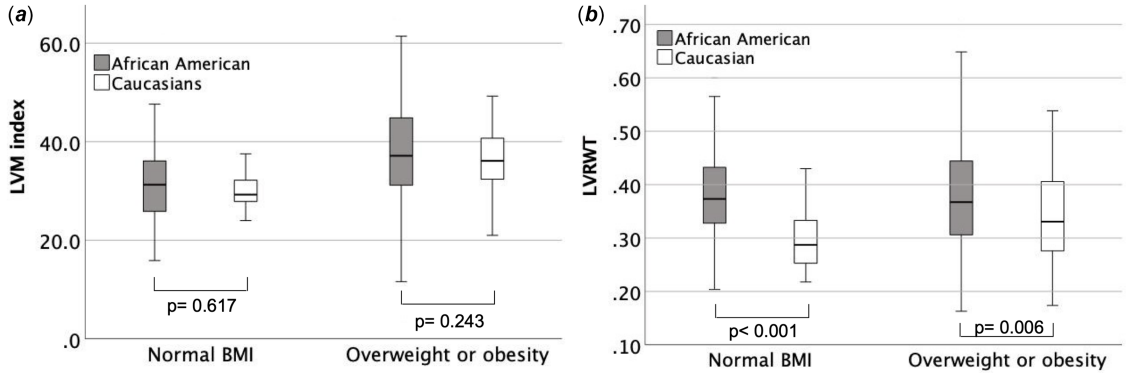

Figure 2. (*a*) Boxplots comparing left ventricular mass index between African Americans and Caucasians by stratification with body mass index. (*b*) Boxplots comparing left ventricular relative wall thickness between African American and Caucasians by stratification with body mass index.

ranges from 14 to 41%, depending on the method used to diagnose left ventricular hypertrophy.[9–13] Monitoring for left ventricular hypertrophy is crucial as patients with left ventricular hypertrophy are at a higher risk for developing heart failure.[8]

Previous studies have reported a higher prevalence of left ventricular hypertrophy in African Americans than in Caucasians,[23,24] with higher prevalence observed in paediatric patients with hypertension.[9,10,12,13,25–31] However, these studies included patients with various significant confounding factors for left ventricular hypertrophy such as obesity, renal diseases, congenital heart diseases, or other causes of secondary hypertension. Our study is the first to focus on patients with essential hypertension.

Although we found no difference in the prevalence of obesity and overweight in the two races in our cohort, we conducted a subgroup analysis to determine the impact of race on left ventricular mass index and left ventricular relative wall thickness by stratifications according to the presence of overweight and obesity. We found that being African American was not associated with an increased left ventricular mass index after stratification according to body mass index percentile, but obesity or overweight (body mass index percentile) was an independent predictor of increased left ventricular mass index regardless of race.

The question remains whether the African American population has a higher left ventricular relative wall thickness ("left ventricular concentric adaptation") compared to other races, regardless of the presence of hypertension. No previous studies compared left ventricular relative wall thickness among children with normal blood pressure between African Americans and other races. In adults, a study reported that concentric left ventricular hypertrophy and increased electrocardiographic voltage were associated with genetically determined African ancestry.[32] It is possible that African American patients need a different reference range for left ventricular relative wall thickness if they normally have higher left ventricular relative wall thickness compared with other races. However, another study comparing African American and Caucasian patients with hypertension found that African Americans had a higher prevalence of concentric adaptation compared to Caucasian patients, while there was no difference in left ventricular mass index.[33] This finding supports our observations, suggesting that the increased prevalence of concentric adaptation with a similar prevalence of left ventricular hypertrophy in response to an exaggerated burden of hypertension in African Americans compared to Caucasians in paediatric populations has a genetic underpinning. This genetic factor may play a significant role in cardiac remodelling in response to increased afterload.[34] Further studies are needed to determine if the African American population has "thicker posterior wall" regardless of the presence of hypertension.

Limitation

This single-centre, retrospective study has a few limitations. First, the actual duration of hypertension is not clear due to our study design. Although we attempted to identify patients who underwent an echocardiogram soon after being diagnosed with hypertension, some patients may have long-standing hypertension before being identified by their primary care physician's office. However, even in a prospective study, it would be difficult to determine the duration of hypertension. Second, we relied on self-reported race and were unable to collect data on genetically determined ancestry. Third, we assessed overweight and obesity using body mass index and were unable to use body fat percentage, which may be more effective in distinguishing patients with obesity from athletes. Fourth, as American Academy of Pediatrics 2017 guideline points out,[16] accurate blood pressure measurement can be challenging in individuals with obesity associated with appropriate cuff size selection. Moreover, obese or overweight children have been excluded from the most recent nomogram of blood pressure. These factors potentially influence accurate diagnosis of hypertension in this population. Lastly, 10 patients were taking anti-hypertensive medication when they underwent echocardiograms at our institution. Their primary care physicians had prescribed these medications upon diagnosing hypertension, prior to the evaluation at our facility. While the use of anti-hypertensive medication has the potential to affect left ventricular hypertrophy and left ventricular remodelling, we believe this had a relatively small impact on our results. These patients were referred to our centre shortly after receiving their hypertension diagnosis and starting their anti-hypertensive treatment at their primary care physician's office.

Conclusions

A significant (20%) of children with essential hypertension had left ventricular hypertrophy even at the time of initial diagnosis. There were no differences in the prevalence of left ventricular hypertrophy in children with essential hypertension between African American and Caucasian populations when the rates of obesity and

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overweight were similar. It is more likely that the presence of obesity and overweight, rather than race, is associated with left ventricular hypertrophy. The major finding of our study is that children with African American race with essential hypertension had a significantly higher prevalence of concentric adaptation compared to Caucasians. Further studies are warranted to investigate the impact of differences in cardiac remodelling among races in response to hypertension.

Supplementary material. The supplementary material for this article can be found at https://doi.org/10.1017/S1047951123003840.

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Author contributions. Dr Takajo, Mr. Przybycien, Dr Natarajan, and Dr Aggarwal conceptualised and designed the study, designed the data collected instruments, collected data, drafted the initial manuscript, and reviewed and revised the manuscript.

Dr Balakrishnan and Dr Singh critically reviewed the manuscript for important intellectual content.

All authors approved the final manuscript as submitted and agreed to be accountable for all aspects of the work.

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Competing interests. None.

Ethical approval. This retrospective chart review study involving human participants was in accordance with the ethical standards of the institutional and national research committee and with the 1964 Helsinki Declaration and its later amendments or comparable ethical standards. The Human Investigation Committee (IRB) of Central Michigan University approved this study.

References

1. Song P, Zhang Y, Yu J, et al. Global prevalence of hypertension in children: a systematic review and meta-analysis. JAMA Pediatr 2019; 173: 1–10.
2. McNiece KL, Poffenbarger TS, Turner JL, Franco KD, Sorof JM, Portman RJ. Prevalence of hypertension and pre-hypertension among adolescents. J Pediatr 2007; 150: 640–644.
3. Din-Dzietham R, Liu Y, Bielo MV, Shamsa F. High blood pressure trends in children and adolescents in national surveys, 1963 to 2002. Circulation 2007; 116: 1488–1496.
4. Cheung EL, Bell CS, Samuel JP, Poffenbarger T, Redwine KM, Samuels JA. Race and obesity in adolescent hypertension. Pediatrics 2017; 139: e20161433.
5. Nadruz W. Myocardial remodeling in hypertension. J Hum Hypertens 2015; 29: 1–6.
6. Jia G, Aroor AR, Hill MA, Sowers JR. Role of Renin-Angiotensin-aldosterone system activation in promoting cardiovascular fibrosis and stiffness. Hypertension 2018; 72: 537–548.
7. Marketou ME, Parthenakis F, Vardas PE. Pathological left ventricular hypertrophy and stem cells: current evidence and new perspectives. Stem Cells Int 2016; 2016: 2016–10.
8. Hunt SA, Abraham WT, Chin MH, et al. 2009 focused update incorporated into the ACC/AHA 2005 Guidelines for the diagnosis and management of heart failure in adults: a report of the American college of cardiology foundation/American heart association task force on practice guidelines: developed in collaboration with the international society for heart and lung transplantation. Circulation 2009; 119: e391–479.
9. Hanevold C, Waller J, Daniels S, Portman R, Sorof J, International Pediatric Hypertension A. The effects of obesity, gender, and ethnic group on left ventricular hypertrophy and geometry in hypertensive children: a collaborative study of the international pediatric hypertension association. Pediatrics 2004; 113: 328–333.
10. Brady TM, Fivush B, Flynn JT, Parekh R. Ability of blood pressure to predict left ventricular hypertrophy in children with primary hypertension. J Pediatr 2008; 152: 73–78, 78 e71.
11. Sorof JM, Alexandrov AV, Cardwell G, Portman RJ. Carotid artery intimal-medial thickness and left ventricular hypertrophy in children with elevated blood pressure. Pediatrics 2003; 111: 61–66.
12. Daniels SR, Loggie JM, Khoury P, Kimball TR. Left ventricular geometry and severe left ventricular hypertrophy in children and adolescents with essential hypertension. Circulation 1998; 97: 1907–1911.
13. Pruette CS, Fivush BA, Flynn JT, Brady TM. Effects of obesity and race on left ventricular geometry in hypertensive children. Pediatr Nephrol 2013; 28: 2015–2022.
14. Du T, Fernandez C, Barshop R, Chen W, Urbina EM, Bazzano LA. 2017 pediatric hypertension guidelines improve prediction of adult cardiovascular outcomes. Hypertension 2019; 73: 1217–1223.
15. Chen X, Wang Y. Tracking of blood pressure from childhood to adulthood: a systematic review and meta-regression analysis. Circulation 2008; 117: 3171–3180.
16. Flynn JT, Kaelber DC, Baker-Smith CM, Subcommittee On S, Management Of High Blood Pressure In C,et al. Clinical practice guideline for screening and management of high blood pressure in children and adolescents. Pediatrics 2017; 140: e20171904.
17. Carretero OA, Oparil S. Essential hypertension. Part I: definition and etiology. Circulation 2000; 101: 329–335.
18. Centers for Disease Control and Prevention. BMI Calculator Child and Teen. Centers for Disease Control and Prevention, 2020 https://www.cdc.gov/healthyweight/bmi/calculator.html
19. Lopez L, Colan SD, Frommelt PC, et al. Recommendations for quantification methods during the performance of a pediatric echocardiogram: a report from the pediatric measurements writing group of the American society of echocardiography pediatric and congenital heart disease council. J Am Soc Echocardiogr 2010; 23: 465–495.
20. Lang RM, Badano LP, Mor-Avi V, et al. Recommendations for cardiac chamber quantification by echocardiography in adults: an update from the American society of echocardiography and the European association of cardiovascular imaging. J Am Soc Echocardiogr 2015; 28: 1–39 e14.
21. Khoury PR, Mitsnefes M, Daniels SR, Kimball TR. Age-specific reference intervals for indexed left ventricular mass in children. J Am Soc Echocardiogr 2009; 22: 709–714.
22. Shrout PE, Fleiss JL. Intraclass correlations: uses in assessing rater reliability. Psychol Bull 1979; 86: 420–428.
23. Drazner MH, Dries DL, Peshock RM, et al. Left ventricular hypertrophy is more prevalent in blacks than whites in the general population: the dallas heart study. Hypertension 2005; 46: 124–129.
24. Levy D, Anderson KM, Savage DD, Kannel WB, Christiansen JC, Castelli WP. Echocardiographically detected left ventricular hypertrophy: prevalence and risk factors. The framingham heart study. Ann Intern Med 1988; 108: 7–13.
25. Mendizabal B, Khoury P, Woo JG, Urbina EM. Racial differences in the influence of risk factors in childhood on left ventricular mass in young adulthood. J Pediatr 2020; 217: 152–157.
26. Dhuper S, Abdullah RA, Weichbrod L, Mahdi E, Cohen HW. Association of obesity and hypertension with left ventricular geometry and function in children and adolescents. Obesity (Silver Spring) 2011; 19: 128–133.
27. Dekkers C, Treiber FA, Kapuku G, Van Den Oord EJ, Snieder H. Growth of left ventricular mass in African American and European American youth. Hypertension 2002; 39: 943–951.
28. de Simone G, Mureddu GF, Greco R, et al. Relations of left ventricular geometry and function to body composition in children with high casual blood pressure. Hypertension 1997; 30: 377–382.
29. Brady TM, Fivush B, Parekh RS, Flynn JT. Racial differences among children with primary hypertension. Pediatrics 2010; 126: 931–937.
30. Daniels SR. Hypertension-induced cardiac damage in children and adolescents. Blood Press Monit 1999; 4: 165–170.

31. Daniels SR, Meyer RA, Liang YC, Bove KE. Echocardiographically determined left ventricular mass index in normal children, adolescents and young adults. J Am Coll Cardiol 1988; 12: 703–708.
32. Alame AJ, Garg S, Kozlitina J, et al. Association of African ancestry with electrocardiographic voltage and concentric left ventricular hypertrophy: the dallas heart study. JAMA Cardiol 2018; 3: 1167–1173.
33. Ejiofor L, Di Nora C, Cervesato E, et al. Differences in left ventricular geometry in hypertensive African-Europeans and Caucasian patients. Eur J Intern Med 2019; 62: 43–47.
34. Kizer JR, Arnett DK, Bella JN,et al. Differences in left ventricular structure between black and white hypertensive adults: the hypertension genetic epidemiology network study. Hypertension 2004; 43: 1182–1188.

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