

Left Ventricular Measurements in Black Sub-Saharan Africans and White Maghreb

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Abstract

Background: Evidence that blacks have greater left ventricular mass (LVM) than whites has been demonstrated by large population-based American studies. However, to our knowledge, there is no study to date comparing LVM in Black Sub-Saharan Africans (BSSA) and the Maghreb white population. We compared LVM measured echocardiographically in asymptomatic BSSA and Maghreb. **Methods and Results:** A total of 100 asymptomatic BSSA and 189 Maghreb, (18 to 55 years old), underwent resting two-dimensional transthoracic echocardiography. LVM and geometry were assessed according to the 2015 American Society of Echocardiography and the European Association of Cardiovascular Imaging updated guidelines for cardiac chamber quantification. Crude or indexed LVM to body surface area or height^{2.7} was similar in BSSA and in Maghreb (132.7 ± 37.0 vs. 134.2 ± 35.7 g; 73.1 ± 17.8 vs. 72.9 ± 16.2 g/m²; 32.1 ± 9.8 vs. 33.6 ± 9.5 g/m^{2.7}). However, the left ventricular posterior wall was thicker in BSSA. Patterns of left ventricular geometry (normal, concentric remodeling, or concentric or eccentric hypertrophy) were equally distributed among the two ethnic groups. **Conclusions:** Left ventricular posterior wall thickness but not LVM is greater in BSSA than in Maghreb.

Keywords

Left Ventricular Mass, Ethnicity, White Maghreb, Black Sub-Saharan Africa

1. Introduction

Blacks are generally more susceptible to cardiovascular disease than whites [1]. Major efforts are underway to reduce disparities in health outcomes between

blacks and whites [2]. Left ventricular mass (LVM) reflects hemodynamic, neurohormonal, humoral, metabolic, and genetic disorders [3] [4] [5]. Environmental factors also influence LVM [6] [7]. Left ventricular hypertrophy (LVH) is an adaptive response allowing a normal ejection fraction despite abnormal pressure and/or volume load [4] [8] [9]. However, recent studies over many years [10] [11] [12] [13] have shown the importance of LVH as a subclinical marker of cardiovascular disease. Indeed, LVH is considered a cardiovascular risk factor (CVRF) [13] [14]. Its value in predicting cardiovascular events has been widely demonstrated in subjects with [10] [13] and without coronary artery disease [15] or heart failure [16]. LVM correlates closely with the incidence of heart failure, stroke, coronary disease [17], and overall mortality [18]. A reduced incidence of cardiovascular events is also noted after lifestyle modification and/or pharmacological treatment to reduce LVM [16] [19].

LVM is determined by heritable genetic factors [20] as well as environmental factors, the latter including diet and physical activity. It was recently demonstrated that a diet low in animal fat and high in vegetables, fruit, and monounsaturated fatty acids is associated with a low LVM [21] [22] and low cardiovascular risk. Physical activity reduces LVM [23], while physical inactivity is associated with increased LVM in whites but not in blacks [7].

The evidence that blacks have a greater LVM than whites do is almost indisputable [24]. LVH is more prevalent in blacks than whites [25] and is also more closely linked to the risk of cardiovascular mortality in African Americans than it is in whites [26]. Disparities in LVM and LVH are likely to explain a large part of the increased cardiovascular mortality in blacks [25].

Differences in LVM between African Americans and white Americans are widely described in the literature. However, to our knowledge, there is no study to date comparing LVM in black sub-Saharan Africans (BSSA) with that of the Maghreb, a white-skinned North African people of Arab-Berber origin. BSSA living in Africa certainly have common ancestry with African Americans, but this relationship is very remote and their lifestyles are very different. Similarly, the Maghreb, although white skinned, have historical and cultural roots completely different from the primarily European ethnic heritage of white Americans. Thus, the results from studies comparing African Americans with white Americans cannot be extrapolated to these two African ethnic groups. The MAG-SALVAGES (MAGhreb and Sub-Saharan African Left-Ventricular ArGeo-metry Study) aimed to compare the LVM of Maghreb with that of BSSA.

2. Methods

2.1. Study Design

2.1.1. Study Population and Settings

The MAG-SALVAGES is a population-based survey conducted in Marrakech, Morocco, from November 2015 to January 2016. The Kingdom of Morocco is a Northwest African country where the quality of university education has improved rapidly in recent years. This makes it a preferred destination for sub-Sa-

haran students, especially Francophone sub-Saharanans. This study compared a BSSA student population living in Marrakech with a Maghreb student population living the same city.

2.1.2. Recruitment Strategy

Investigators: The investigation team consisted of senior residents of the Cardiology Unit of Cady Ayyad University. The principal investigators (BKP and CA) introduced the study, explained the goals, described how the study was to be performed, and described the responsibilities of each investigator. The principal investigators attended staff meetings to discuss and answer any questions the investigator team had. A pre-test session was conducted asking a dozen students to evaluate the questionnaire, to test the appropriateness of the data collection sheet, and to assess the ability of the investigators to apply the protocol correctly.

Study participants: The CESAM (Confédération d'Etudiants et Stagiaires Africains de Marrakech) that is a cultural association encompassing all BSSA students in Marrakech, provided us with the names and addresses of BSSA students. Recruitment of Maghreb students was facilitated by the Secretary General of the Cady Ayyad University Students Office. Students were randomly chosen and invited to participate in the study.

Eligibility criteria: The subjects recruited for the MAG-SALVAGES were asymptomatic students aged 18 years or older, regardless of their level of study (undergraduate, graduate, post-graduate, or doctoral). Participants with significant valvulopathy, who had poor echogenicity to the extent that echocardiographic parameters could not be measured, or who did not complete the planned investigations were excluded.

2.2. Data Collection

Left ventricular measurements were taken according to the 2015 American Society of Echocardiography and the European Association of Cardiovascular Imaging updated guidelines for cardiac chamber quantification [27], using an HD 15 Sonos 5500 (Phillips Medical Systems, Andover, MA, USA) ultrasound system equipped with 2.5, 3.5, and 5.0 MHz transducers. Three measurements were taken for each variable, with the mean value used in analysis. Images were stored for subsequent validation by a team of three skilled training specialists of the CHU Mohammed VI echocardiography laboratory. Two-dimensionally guided M-mode echocardiography was performed on a parasternal long-axis view. Interventricular septum (IVS) thickness in diastole (IVSd) in mm, left ventricular posterior wall (PW) thickness in diastole (LVPWd) in mm, and left ventricular end-diastolic diameter (LVEDd) in mm were measured at end-diastole at a level just below the mitral valve leaflets. Simultaneous ECG was used to correlate measurements with the cardiac cycle. Diastolic wall thickness was measured at the onset of the QRS wave. LVM was calculated according to the American Society of Echocardiography simplified cubed equation linear method using the following equation: $LVM \text{ (grams)} = 0.8 \times 1.04 \times [(LVEDd + IVSd + LVPWd)^3 -$

(LVEDd)3] + 0.6 g. LVM was indexed by BSA and by height^{2.7}. LVM was considered normal when $\leq 115 \text{ g/m}^2$ or $\leq 48 \text{ g/m}^{2.7}$ in males and $\leq 95 \text{ g/m}^2$ or $\leq 44 \text{ g/m}^{2.7}$ in females. LVH was defined as LVM exceeding those values. The relative wall thickness (RWT) of the left ventricle (LV) was calculated as $(2 \times \text{LVPWd})/\text{LVEDd}$. LV geometric patterns were defined as follows: normal geometry (normal LVM and $\text{RWT} \leq 0.42$), concentric remodeling (normal LVM and $\text{RWT} > 0.42$), concentric hypertrophy (LVH and $\text{RWT} > 0.42$), and eccentric hypertrophy (LVH and $\text{RWT} \leq 0.42$).

To analyze LV systolic function, the ejection fraction (stroke volume/diastolic volume $\times 100$), percentage of LV systolic shortening (%) ($[(\text{diastolic diameter} - \text{systolic diameter})/\text{diastolic diameter} \times 100]$) and cardiac output (stroke volume \times heart rate) were calculated. Stroke volume was calculated as $2\pi D/4 \times$ velocity time integral, where D is the diameter of the aortic annulus measured in the left parasternal long-axis view at mid-systole from inner edge to inner edge.

The presence or absence of valve disease was detected by visual assessment in 2D and color Doppler in the left parasternal long-axis and apical four-chamber views.

Statistics

Details about the other data collection strategy (demographic characteristics, lifestyle, personal medical history, physical measurements, Vital signs, and ECG Measurements and interpretation) have been described elsewhere [28] [29].

2.3. Statistics

After encoding and validation, the data were entered in a computer using Epi-InfoTM statistical software, version 7.1.2.0 (Atlanta, USA) (<https://www.cdc.gov/epiinfo/index.html>). Results for each BSSA were compared with two Maghreb subjects matched for age (± 1 year) and sex in order to overcome the influence of these variables on LVM. Continuous variables are expressed as means and categorical variables as percentages. In order to compare means in independent samples, Student's t test or the Mann-Whitney test was used. Comparison of the means of three or more groups was performed with analysis of variance. The Tukey test for multiple comparisons was used to distinguish the different groups. For variables not fitting a normal distribution, the Kruskal-Wallis analysis of variance and the corresponding test of multiple comparisons were used. Pearson's correlation coefficient (r) was used to assess any significant association between two continuous variables. To compare proportions, the chi-square test or Fisher exact test was used. Frequency distributions of LVM dichotomized by ethnicity were constructed. Comparisons were made between BSSA and Maghreb for all parameters. Similar analyses stratified by gender were performed. When differences were observed on univariate analysis, the effect of potential confounders was studied by adjustment with multivariate linear regression. A significance level of 5% was adopted. IBM SPSS version 21 (http://www.ibm.com/support/knowledgecenter/SSLVMB_21.0.0) was used for statistical calculations.

2.4. Ethics and Consent

This research was conducted in strict compliance with the recommendations of the Helsinki Declaration III. All respondents were briefed on the conclusions drawn from their investigations. Approval was obtained from the ethics committee of Cady Ayyad University. Each participant provided written informed consent.

3. Results

Table 1 displays comparatively the demographic and lifestyle characteristics of SSA and Maghreb. Overall, Maghreb were more active and consumed more fruits and vegetables than the BSSA did. The two different ethnic groups did not differ in age, gender, physical activity and cigarette smoking.

Table 2 displays the physical variables and cardiovascular risk factors of the study population as a whole and stratified according to the ethnicity. Maghreb had greater parameters of obesity, including BMI, WC, HC, WHR and fat mass.

Table 1. Characteristics of the study population as a whole and stratified according to ethnicity.

Variables	All participants n = 289	BSSA n = 100	Maghreb n = 189	P
Demographic characteristics				
Age, years	29.8 ± 11.2	28.5 ± 10.7	30.4 ± 11.5	0.171
Gender				0.121
Male, n (%)	173 (59.9)	65 (65.0)	108 (57.1)	
Female, n (%)	116 (40.1)	35 (35.0)	81 (42.9)	
Place of birth				<0,0001
SSA, n (%)	99 (34.4)	98 (98.0)	1 (0,5)	
Maghreb, n (%)	189 (65.6)	2 (2.0)	187 (99.5)	
Lifestyle characteristics				
Regular consumption of				
Fruits, n (%)	162 (56.1)	39 (39.0)	123 (65.1)	<0.0001
Vegetables, n (%)	225 (77.9)	68 (68.0)	157 (83.1)	0.005
Fats, n (%)	276 (95.3)	93 (93.0)	183 (96.8)	0.201
Smoking/Alcohol				
Current smoker, n (%)	18 (6.2)	6 (6.0)	12 (6.3)	0.564
Excessive drinker, n (%)	8 (2.8)	2 (2.0)	6 (3.2)	0.436
Physical activity				
Active, n (%)	203 (70.2)	61 (61.0)	142 (75.1)	0.015
Inactive, n (%)	86 (29.8)	39 (39.0)	47 (24.9)	0.018
sedentary, n (%)	87 (30.1)	19 (29.0)	58 (30.7)	0.801

BSSA: Black Sub-Saharan African, SSA: Sub-Saharan Africa.

Table 2. Physical variables and cardiovascular risk factors of the study population as a whole and stratified according to the ethnicity.

Variables	All participants	BSSA	Maghreb	p
Weight, kg	73.3 ± 14.9	72.2 ± 13.2	73.9 ± 15.8	0.368
Height, cm	168.1 ± 9.8	169.7 ± 8.5	167.2 ± 10.3	0.039
BMI, kg/m ²	25.9 ± 5.5	24.5 ± 4.6	26.7 ± 5.7	0.001
WC, cm	86.3 ± 14.5	82.4 ± 15.2	88.4 ± 13.7	0.001
HC, cm	99.6 ± 10.6	96.3 ± 9.4	101.4 ± 10.7	<0.0001
WHR, cm	0.87 ± 0.12	0.86 ± 0.17	0.87 ± 0.08	0.399
WHeR, cm	0.45 ± 0.09	0.41 ± 0.06	0.50 ± 0.08	0.031
SBP, mmHg	119.4 ± 15.1	116.6 ± 14.6	120.9 ± 15.2	0.020
DBP, mmHg	77.2 ± 10.1	76.4 ± 10.4	77.6 ± 10.0	0.316
PP, mmHg	42.2 ± 11.7	40.2 ± 11.1	43.3 ± 11.9	0.035
MBP, mmHg	91.3 ± 10.7	89.8 ± 10.7	92.1 ± 10.6	0.084
HR, bpm	72.7 ± 12.9	71.5 ± 12.0	73.4 ± 13.4	0.227
Fat mass, %	20.9 ± 12.2	17.4 ± 9.2	22.8 ± 13.1	<0.0001
Lean mass, %	29.6 ± 4.5	29.6 ± 4.3	29.6 ± 4.7	0.972
CVRF				
HTN, n (%)	9 (3.1)	3 (3.0)	6 (3.2)	0.893
DM1, n (%)	1 (0.3)	0 (0.0)	1 (0.5)	0.654
DM2, n (%)	5 (1.7)	1 (1.0)	4 (2.1)	0.441
Total obesity, n (%)	63 (21.8)	15 (15.0)	48 (25.4)	<0.0001
Abdominal obesity, n (%)	64 (22.1)	14 (14.0)	50 (26.5)	0.013

BSSA: Black Sub-Saharan African, BMI: body mass index, WC: waist circumference, HC: hip circumference, WHC: waist-to-hip ratio, WHeR = waist-to-height ratio, SBP: systolic blood pressure, DBP: diastolic blood pressure, PP: pulse pressure, MBP: mean blood pressure, HR: heart rate, FM: fat mass, CVRF: cardiovascular risk factor, DM1: type 1 diabetes mellitus, DS2: type 2 diabetes mellitus.

They had also a higher prevalence of both total and abdominal obesity. When comparing the parameters of blood pressure, Maghreb had higher SBP, while the other parameters including DBP, MBP and PP did not differ between the two ethnic groups. The prevalence of hypertension and diabetes mellitus was similar in the two ethnic groups.

Electrocardiographic and echocardiographic characteristics of the two ethnic groups are shown in **Table 3**. QRS and QTc duration were the only ECG parameters that differed between the two populations, with a significantly longer average duration among Maghreb. The same table shows a significantly higher LVEDd among Maghreb than among BSSA, but this difference disappeared when indexed to BSA. LVPWd was significantly greater among BSSA than among Maghreb. This difference persisted after adjustment for confounding factors (BMI, WC, HR, and fat mass). The differences observed for all other echocardiographic parameters were not statistically significant, including LVM for which the means of the two groups were similar regardless of the mode of Indexing.

Table 3. Electrocardiographic and echocardiographic characteristics.

Variable	All participants	BSSA	Maghreb	p
PR, msec	145.9 ± 27.5	146.2 ± 23.2	145.8 ± 29.6	0.901
QRS, msec	94.7 ± 23.2	90.2 ± 20.5	97.1 ± 24.2	0.016
QTc duration, msec	401.5 ± 40.1	394.1 ± 33.6	405.4 ± 42.7	0.023
LVEDd, mm	48.2 ± 4.8	47.1 ± 5.1	48.9 ± 4.6	0.004
LVEDd/BS, mm/m ²	25.6 ± 6.0	26.2 ± 2.9	25.3 ± 7.2	0.218
LVEDs, mm	30.3 ± 4.1	29.9 ± 4.2	30.6 ± 4.1	0.185
LVPWd, mm	8.1 ± 1.3	8.3 ± 1.2	7.9 ± 1.3	0.011
IVSd, mm	8.2 ± 1.3	8.4 ± 1.3	8.2 ± 1.3	0.187
LVM, g	133.7 ± 36.1	132.7 ± 37.0	134.2 ± 35.7	0.726
LVM/height ^{2.7}	33.1 ± 9.6	32.1 ± 9.8	33.6 ± 9.5	0.185
LVM/BS, mg/cm ²	73.0 ± 16.8	73.1 ± 17.8	72.9 ± 16.2	0.922
LVEF, %	65.7 ± 7.7	65.3 ± 9.4	65.9 ± 6.6	0.526

BSSA: Black Sub-Saharan African. PR: PR interval. QRS: QRS duration. QTc: heart rate-corrected QT. LVEDd: left ventricular end-diastolic diameter. LVEDd/BS: left ventricular end-diastolic diameter indexed for body surface area. LVEDs: left ventricular end-systolic diameter. LVPWd: left ventricular posterior wall thickness in diastole. IVSd: Interventricular septum thickness in diastole. LVM: left ventricular mass crude. LVM/height^{2.7}: left ventricular mass indexed for height 2.7. LVM/BS: left ventricular mass indexed for body surface area. LVEF: left ventricular ejection fraction.

4. Discussion

This is, to the best of our knowledge, the first population-based study to evaluate ethnic disparities in LVM on the African continent. The main finding was the lack of difference in LVM between Maghreb and BSSA, two ethnic groups with quite different historical origins, dietary habits, physical activity, and physical habitus.

This comparison of LVM in BSSA and Maghreb ideally would comprise samples randomly selected from these ethnic groups. Unfortunately, it is difficult to obtain a representative sample of both populations in the strict epidemiologic sense for obvious reasons of feasibility and budget. For this study, we assumed that a BSSA community and a Maghreb community living in Morocco were representative of the populations from which they originated. CESAM includes BSSA students from the majority of sub-Saharan countries, including Mali, Democratic Republic of the Congo, Congo-Brazzaville, Central African Republic, Cameroon, Togo, Chad, Zimbabwe, Tanzania, Angola, Sudan, Somalia, Madagascar, Mauritius, Comoros, Senegal, Nigeria, and Mauritania [28] [29]. Random sampling in this community can be therefore considered representative of the sub-Saharan population.

LVM was evaluated using resting two-dimensional transthoracic echocardiography, long considered the gold standard for measurement of LVM. Magnetic resonance imaging is more accurate than echocardiography [30], so it might be expected to supplant echocardiography. Indeed, the recommended formula for echocardiographic estimation of LVM uses linear measurements based on the

assumption that the LV is a prolate ellipsoid of revolution, whereas cardiac magnetic resonance models of the LV free of geometric assumptions or acoustic window dependency, yielding better accuracy and reproducibility. However, echocardiography remains the most commonly used imaging modality due to its widespread availability and lower cost. Because it remains the clinical standard, this study used transthoracic echocardiography to measure LVM.

The results of this study contrast with the vast majority of studies that have investigated this issue and which have reported a greater LVM and prevalence of LVH in blacks than in whites [25] [31]-[37].

An old meta-analysis by Devereux *et al.* reviewed nine previous studies looking at ethnic disparities in LVM and authors reported that LV wall thickness but not LVM was consistently greater in blacks than in whites. That analysis recalls the often forgotten fact that thick walls are not the same as LVH, even LV wall thickness is commonly considered a surrogate for LVM, so that wall thickening is equated with LVH. In reality, this overlooks the relationship between LV volume and wall thickness. The 2015 ASE guidelines for chamber quantification [29] describe a normal LV wall thickness (IVS or PW) as 0.6 - 1.0 cm for males and 0.6 - 0.9 cm for females. An increased wall thickness may be suggestive of LVH, but as an isolated number it gives no information about LVM or LV modeling. Indeed, LVM is estimated by calculating the total heart volume (epicardial volume) minus the volume of blood in the cavity (endocardial volume), resulting in myocardial volume, and then multiplying by the specific gravity of tissue (1.05). In our study, BSSA had significantly thicker LVPWd than Maghreb, but the LVM did not differ between the two ethnic groups.

LVM is determined by heritable genetic [12] and environmental factors. The historical and ethnic origins of the participants of this study both suggest genetic differences between these two populations. The impact of genetics is often cited to explain ethnic disparities in LVM. But phenotypic manifestations are related to both genetic and environmental factors. In this study, the main environmental factors that may interact with genetics to affect LVM were unevenly distributed between the ethnic groups. The BSSA were less obese and more physically inactive than the Maghreb, while the opposite was observed in the American population, where blacks were more obese and had a higher LVM than whites. It has been postulated that the higher prevalence of obesity in African American populations explains the greater prevalence of LVH, but Mark *et al.* showed that this ethnic disparity persisted after adjustment for body composition [16].

5. Limitations of the Study

A limitation of this study is that BSSA participants were drawn from a migrant population, whose lifestyle therefore does not necessarily reflect that of their country of origin.

6. Conclusion

BSSA had thicker LVPWd than the Maghreb, while the two ethnic groups had

similar LVM. Genetic and environmental factors that influence LVM differ from one ethnic group to another and from one person to another both in nature and magnitude. This probably explains both the differences and similarities in LVM between two ethnic groups, as well as between two individuals.

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