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Differences in left ventricular geometry in hypertensive African-Europeans and Caucasian patients

Laurence Ejiofor[a], Concetta Di Nora[b,*], Eugenio Cervesato[a], Iulian Cosei[c], Andreea Ravasel[c], Bogdan A. Popescu[c,d], Francesco Antonini-Canterin[e]

[a] Cardiology Department, ARC Ospedale di Pordenone, Italy
[b] Cardiology Department, Azienda Sanitaria Universitaria Integrata di Trieste, Italy
[c] Emergency Institute of Cardiovascular Diseases 'Prof. Dr. C. C. Iliescu', Bucharest, Romania
[d] University of Medicine and Pharmacy "Carol Davila" Bucharest, Romania
[e] Ospedale Riabilitativo di Alta Specializzazione, Cardiologia Riabilitativa, Motta Di Livenza, Italy

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ABSTRACT

Background: There are data showing race-related differences regarding left ventricular (LV) geometry in hypertensive patients. Several authors reported that concentric remodeling is the most common remodeling pattern in hypertensive African-Americans, and this pattern may be related to prognosis. There is little information about the LV remodeling patterns in hypertensive Africans that migrated to Europe, which might have different distributions from those seen in African-Americans. The aim our study was to describe the prevalence of LV remodeling patterns in hypertensive African-Europeans and to compare it with that of hypertensive Caucasians.
Methods: This is a descriptive study that included 135 consecutive treated hypertensive African-Europeans and 128 hypertensive Caucasians. Patients were examined by transthoracic echocardiography and categorized into the four classic geometric patterns according to LV mass index (LVMi) and relative wall thickness (RWT).
Results: The mean age and gender distribution were similar in the 2 groups. Caucasians had significantly higher body mass index, LV diastolic and systolic diameters, while African-Europeans had higher RWT and interventricular septum thickness. No differences in LVMi was found. In the African-European group, the prevalence of normal pattern, concentric remodeling, concentric hypertrophy and eccentric hypertrophy were 13%, 36%, 45% and 6% respectively, while in Caucasians they were 21%, 33%, 34% and 12%, respectively. African-Europeans had a higher prevalence of concentric remodeling and hypertrophy compared to Caucasians (81% vs. 67%, $p = .005$).
Conclusions: This study shows important differences in LV geometry between treated African-European and Caucasian hypertensive patients. Also, African-Europeans may have slightly different characteristics compared to African-Americans.

1. Introduction

Arterial hypertension is a major health concern, with a prevalence in the adult population of 30–45% [1]. Hypertension generally leads to left ventricular hypertrophy (LVH), which is an important preclinical condition with incremental adverse prognostic value beyond that provided by traditional risk factors [2]. The attributable risk of LVH for all-cause mortality might be greater than that of single or multivessel coronary artery disease [3]. LVH has a dual role in patients with systemic hypertension, being both a necessary adaptation for maintaining a normal stroke volume against the increased pressure load and a pathologic manifestation of the hypertensive cardiovascular disease. Cardiac adaptation to arterial hypertension may result in three different left ventricular (LV) geometric patterns that reflect the different response to hemodynamic overload [4]. Increased LV mass may appear from predominant wall thickening or predominant chamber enlargement leading to concentric hypertrophy or eccentric hypertrophy, respectively. Relative LV wall thickening in the absence of an increase in LV mass is defined as concentric remodeling.

It is well known that African-Americans have a disproportionate burden of the cardiovascular morbidity and mortality compared with Caucasians. The reasons for this differences are not fully understood, but the increase of LV wall thickness, highly prevalent in the African-American population, is considered a major contributing factor [5].

* Corresponding author.
E-mail address: concetta.dinora@gmail.com (C. Di Nora).

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Although African-Americans have a higher predisposition to hypertension, previous studies have suggested that the higher prevalence of LV hypertrophy cannot be attributed exclusively to hypertension itself, but race factors might have an important contribution [6,7].

There is very little information available in the literature about the distribution of LV remodeling patterns in hypertensive Africans that migrated to Europe or European descendants of Africans (further reported as African-Europeans). Since African-Europeans and African-Americans have known different genetic characteristics, influenced by geographical and historical factors, the adaptive response to LV pressure overload could be different in the two populations [8,9]. In this context, it could be misleading simply to extrapolate data from African-Americans to describe biological adaptations in African-Europeans.

The aim of our study was to describe the prevalence of the remodeling patterns in hypertensive African-Europeans and to compare it with the distribution of different remodeling patterns in hypertensive Caucasians.

2. Methods

2.1. Subjects

The study population included 135 consecutive treated hypertensive African-Europeans (Africans living in Italy) and 128 treated hypertensive Caucasians that were referred for cardiologic evaluation in our hypertension clinic. Anthropometric parameters and blood pressure were measured for all the patients (pts). Medical history was obtained through direct questioning and by reviewing the medical records, with special interest about the history of hypertension, diabetes, smoking, presence of known coronary artery disease or angina and the medication used in the last month. Body mass index was calculated as weight (kilograms) divided by height (meters) squared. Body surface area (BSA) was calculated using DuBois formula: 0.20247 x height $(m)^{0.725}$ x weight $(kg)^{0.425}$. Hypertension was defined as systolic blood pressure (BP) $\geq$140 mmHg, diastolic BP $\geq$ 90 mmHg (at two separate measurements) or use of chronic treatment for systemic arterial hypertension.

All the patients were examined using transthoracic echocardiography. The exclusion criteria were more than mild valvular heart disease and known coronary artery disease, factors that have an important impact on LV remodeling.

Fasting blood samples were collected and plasma glucose, serum creatinine, total cholesterol and triglycerides levels were determined. Diabetes was defined as fasting glucose $\geq$126 mg/dl or the use of hypoglycemic medication. Estimated glomerular filtration rate (eGFR) was calculated using Cockroft-Gault formula. Reduced eGFR was defined as < 60 ml/min/1.73 m^2. Patients with total cholesterol and triglycerides levels above laboratory upper limit of normal were reported as having dyslipidemia.

2.2. Echocardiography

All measurements were performed according to American Society of Echocardiography and the European Association of Cardiovascular Imaging recommendations [10]. LV internal dimensions and wall thickness were obtained from parasternal long-axis view as 2D-guided linear measurements, perpendicular to the LV long axis, at end-diastole. LV volumes and LVEF were assessed using Simpson biplane method. LV mass (LVM) was determined using cube formula: $LVM = 0.8 \times 1.04 \times [(IVS + LVID + PWT)^3\text{-}LVID^3] + 0.6$ g, where IVS is interventricular septum, LVID is LV internal diameter and PWT is infero-lateral wall thickness. To take the body size into account, the LV mass was indexed for BSA (LVMi). LV hypertrophy was defined as LVMi > 115 g/m^2 in men and > 95 g/m^2 in women. Relative wall thickness (RWT) was calculated with the formula (IVS + PWT)/LVID. The upper limit of normal RWT was considered 0.42.

The patients were categorized into the four classic geometric patterns according to LV mass index (LVMi) and relative wall thickness (RWT): normal geometry (non-LV hypertrophy and normal RWT), concentric hypertrophy (LV hypertrophy and RWT > 0.42), eccentric hypertrophy (LV hypertrophy and normal RWT) and concentric remodeling (non-LV hypertrophy and RWT > 0.42) [4].

2.3. Statistical analysis

Descriptive statistics were displayed using means and standard deviation for continuous variables, while categorical variables were presented as numbers and percentages. To compare the distribution of different parameters between African-Europeans and Caucasians we used unpaired Student *t*-tests for parametric variables with a normal distribution (after testing for normality using Kolmogorov–Smirnoff test) and chi-square tests for non-parametric variables, as appropriate. For all the tests, p values < .05 were considered to indicate statistical significance. For statistical analysis, a dedicated software was used (IBM SPSS Statistics Version 20, IBM Corporation).

Written informed consent was obtained for all the participants.

3. Results

African-Europeans and Caucasians had similar gender distribution (65% vs. 64% men, p = .84). There was no difference regarding the mean age in the two groups (mean age of African-Europeans and Caucasians was 40 ± 10 years and 42 ± 10 years, respectively; p = .10). The height was similar in the two groups (170 ± 8 cm vs. 171 ± 10 cm, p = .41), but Caucasians had significantly increased weight compared to African-Europeans (89 ± 21 kg vs. 77 ± 12 kg) which lead to a higher BMI (30 ± 7 kg/m^2 vs. 27 ± 5 kg/m^2) and BSA (2 ± 0.2 m^2 vs. 1.9 ± 0.2 m^2). When pts. where classified as obese/non-obese (BMI cut-off 30 kg/m^2), Caucasians had a significantly increased prevalence of obesity (44.5% vs 20%, p < .001).

The systolic and diastolic blood pressure was similar in the two groups. Significantly more Caucasians had dyslipidemia (33.6% vs. 5.9%, p < .001) and reported a history of smoking (33.6% vs. 8.1%, p < .001). Chronic kidney disease was present in < 10% of pts., with similar distribution across races. There was a trend towards increased prevalence of diabetes mellitus in Caucasians compared to African-Europeans (19.5% vs. 11.9%, p = .09). The clinical characteristics of pts. stratified by race are summarized in Table 1.

In Table 2 are summarized the main echocardiographic parameters evaluated in the two groups. Caucasians had significantly larger mean LV diastolic diameter and mean LV systolic diameter, findings probably explained by larger BSA. After indexing this diameters to BSA, they did

Table 1
Characteristics of study population stratified by race.

Characteristics	African-Europeans (n = 135)	Caucasians (n = 128)	P value
Gender – men	88 (65%)	82 (64%)	0.84
Age (years)	40 ± 10	42 ± 10	0.10
Height (cm)	170 ± 8	171 ± 10	0.41
Weight (kg)	77 ± 12	89 ± 21	< 0.001
Body mass index (kg/m^2)	27 ± 5	30 ± 7	< 0.001
Body surface area (m^2)	1.9 ± 0.2	2 ± 0.2	< 0.001
Systolic blood pressure (mmHg)	144 ± 25	145 ± 20	0.76
Diastolic blood pressure (mmHg)	90 ± 16	91 ± 13	0.66
Mean blood pressure (mmHg)	108 ± 18	109 ± 15	0.67
eGFR ≤60 ml/min/1.73m^2	11 (8.1%)	10 (7.8%)	0.617
Diabetes mellitus	16 (11.9%)	25 (19.5%)	0.09
Dyslipidemia	8 (5.9%)	43 (33.6%)	< 0.001
Obesity	27 (20%)	57 (44.5%)	< 0.001
History of smoking	11 (8.1%)	43 (33.6%)	< 0.001

Table 2
Echocardiographic characteristics of study population stratified by race.

Characteristics	African-Europeans (n = 135)	Caucasians (n = 128)	*P* value
LV diastolic diameter (mm)	47 ± 0.6	50 ± 0.5	< 0.001
LV systolic diameter (mm)	30 ± 0.6	33 ± 0.7	0.002
Interventricular septum (mm)	12.2 ± 0.3	11.5 ± 0.2	0.019
Infero-lateral wall (mm)	11.6 ± 0.2	11.2 ± 0.2	0.096
LV ejection fraction (%)	60 ± 7	59 ± 8	0.28
RWT	0.51 ± 0.12	0.45 ± 0.08	< 0.001
LVMi (g/m^2)	116 ± 40	114 ± 33	0.67
Left atrial anterior-posterior diameter (mm)	35 ± 6	38 ± 6	< 0.001

Abbreviations: LV - left ventricle; LVMi - left ventricular mass index; n - number; RWT - relative wall thickness.

not differ significantly between African-Europeans and Caucasians: mean values of indexed LV diastolic diameter were 25.2 mm ± 3.5/m^2 vs. 2.562 ± 3.4 mm/m^2 (p = .417) and mean values of indexed LV systolic diameter were 16.0 mm ± 3.6/m^2 vs. 16.4 ± 3.8 mm/m^2 (p = .375). Left ventricular ejection fraction was similar in the two groups, and Caucasians had higher left atrial anterior-posterior diameter.

African-Europeans had a significant thicker interventricular septum (p = .019), with a trend towards thicker LV infero-lateral wall (p = .096). This resulted in a significantly higher mean value of RWT in African-Europeans (0.51 ± 0.12 vs. 0.45 ± 0.08, p < .001). Even after we dichotomized the values of RWT in normal/abnormal using the cut-off of 0.42, the differences remained significant (p = .007). There were no significant differences regarding LVMi (116 ± 40 g/m^2 vs. 114 ± 33 g/m^2, p = .67).

The prevalence of remodeling patterns in each group is represented in Fig. 1. The majority of pts. in both groups had concentric remodeling and hypertrophy, while only 13% and 21% had a normal pattern in the African-Europeans and Caucasian groups, respectively. The African-Europeans had a significantly higher prevalence of concentric remodeling and hypertrophy compared to the Caucasians (81% vs. 67%, p < .01).

4. Discussion

It is well known that African-Americans, compared with Caucasians, generally develop hypertension at a younger age with higher values of blood pressure. Hypertensive African-Americans have a greater progression to heart failure, coronary heart disease, end-stage renal disease and stroke than their white counterparts. Even more, they have a mortality rate related to stroke and coronary heart disease of 50% and

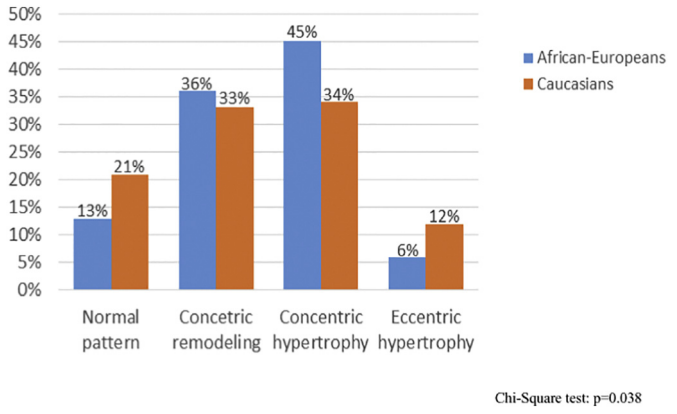

Fig. 1. Prevalence of remodeling patterns stratified by race.

80% higher, respectively, than those of white Americans [11].

There are contradictory data whether an inherent racial susceptibility or environmental factors are implicated in the higher predisposition of Africans to LV remodeling and adverse outcomes, but there is a growing body of evidence suggesting that both factors play an important role. The Multi-Ethnic Study of Atherosclerosis (MESA) that included 6814 pts. (38.5% Caucasians, 27.8% African American, 21.9% Hispanic) with a median follow-up of 4 years suggested that the increased risk of congestive heart failure seen in African-Americans is related to the higher prevalence of hypertension, diabetes mellitus and low socioeconomic status [12]. Another study which compared 34 hypertensive African-Americans and 39 hypertensive Caucasians with similar age, duration of hypertension, and weight showed that most Caucasians had a normal ventricular geometry, whereas concentric remodeling and concentric hypertrophy were common adaptive ventricular patterns in the African-Americans. However, Caucasians were taller, and African-Americans had a significantly higher BMI (obesity was present in 59% of African-Americans and 28% of Caucasians); these results led the investigators to conclude that the differences in LV geometry might be secondary to the different anthropometric characteristics [13].

On the other hand, data derived from the The Coronary Artery Risk Development in Young Adults Study (CARDIA) that included 3475 African Americans and Caucasians suggested that race-related differences in remodeling patterns are not fully explained by the exposure to cardiovascular risk factors [14]. Other studies reported similar results, with increased left ventricular mass and relative wall thickness in hypertensive African-Americans “out of proportion” relative to the differences in standard clinical and hemodynamic parameters when compared with Caucasians [7]. Further data supporting the hypothesis that race has an important impact of LV geometry came from several studies of athletes. In healthy adolescent amateur football players (age 11.8–16.9 years) it was reported that Africans exhibited a greater thickness of septum and LV posterior wall, higher LVM and RWT than Caucasians [15]. Another study of Norwegian professional football players reported similar LVMi between African-Europeans and Caucasians but with an increased RWT in African-Europeans athletes, which resulted in a significantly increased prevalence of LV concentric remodeling pattern than that seen in Caucasian athletes [16]. Similar results were reported by Basavarajaiah et al., showing that black athletes had increased LV wall thickness and LV mass compared with white athletes, without differences in the LV cavity size and blood pressure in this two groups. One-fifth of black athletes had LVH compared with just 4% of white athletes. No differences in LV wall thickness or LV cavity was shown between black and white sedentary subjects [17]. Data from the Framingham Heart Study and the Hypertension Genetic Epidemiology Network (HyperGEN) study showed important heritability in LV mass and geometry in African Americans and Caucasians, with the implication of inherited genes and familial environments [18,19]. Genes may influence LV mass regardless of the blood pressure values by influencing the production of cellular proteins or extracellular matrix. LV mass, as measured by echocardiography, is a strong predictor factor of cardiovascular events in the general population and in patients with hypertension [20,21]. LV mass predicts the risk of cardiovascular events independently of blood pressure, cigarette smoking, total cholesterol level or presence of coronary artery disease [22,23]. Furthermore, there are studies that showed the prognostic value of LV geometry pattern beyond that provided by LV mass in patients with essential hypertension or aortic valve disease [23,24]. Even when the left ventricular mass is normal, there are data suggesting that the concentric remodeling pattern might be associated with increased cardiovascular risk [25].

There is little information available about the LV remodeling patterns in hypertensive African-Europeans, and in the context of geographical and historic factors that might influence the genetic diversity, it is difficult to extrapolate the above-mentioned data about African-

Americans to describe African-Europeans [8,9]. It was reported that the most frequent abnormal LV geometric pattern in hypertensive African-Americans was concentric remodeling while the hypertensive African-Europeans from our population had most often concentric hypertrophy (45%), finding that is concordant with the results of a smaller study that included Africans that migrated to Europe [26–29]. Also, in our African-European population we observed a normal pattern in only 13% of cases vs 21% in Caucasians, while the hypertensive African-Americans from the Jackson Heart Study had a normal pattern in 74.2% of cases even though our patients were younger, finding which may be secondary to a referral bias in our population, taking into account that the Jackson Heart Study was a community-based study [27]. The relatively low prevalence of normal pattern observed in our population is similar to that reported in SABRE Study which included echocardiographic evaluation of 1356 pts. (642 Caucasians, 224 African-Caribbeans living in the United Kingdom and 490 Asians) and reported that only 16% of Africans and 32.7% of Caucasians had normal LV geometry [30].

There are contradictory data regarding the LVMi of African-Americans and African-Europeans compared with Caucasians. Both Dallas Heart Study and CARDIA reported that African-Americans had increased LVMi compared to Caucasians, while no differences were reported in other studies [14,31,32]. Regarding the African-Caribbeans from SABRE Study, it was reported that three-dimensional left ventricular mass was similar between Africans and Caucasians, while conventional two-dimensional LVMi was significantly higher in African-Caribbeans, concluding that three-dimensional echocardiography might be more accurate for assessing LVMi because of less geometric assumptions [30]. In our population, in which we excluded pts. with known coronary artery disease, using two-dimensional derived LVMi, we found similar LVMi between the African-Europeans and Caucasians.

Contrary to the reported data in literature about African-Americans, African-Europeans included in our study had less cardiovascular risk factors compared with Caucasians: the prevalence of obesity, dyslipidemia and history of smoking was significantly lower; there was a trend towards increased prevalence of diabetes mellitus in Caucasians and we didn't find significant differences regarding the prevalence of chronic renal disease [33]. Also, the systolic, diastolic and mean blood pressure were similar in this two groups of treated hypertensive pts.

African-Europeans in our study had higher RWT than Caucasians, difference that remained significant even after we dichotomized the values of RWT in normal/abnormal using the cut-off of 0.42. Even though we performed an unadjusted comparison of RWT in the two groups, it should be noted that the confounding factors commonly described to be associated with higher values of RWT (i.e. BMI, blood pressure, prevalence of dyslipidemia, diabetes mellitus, chronic renal disease, smoking) were either similar or less in the African-European group. Increased RWT resulted in a significantly higher prevalence of concentric remodeling and hypertrophy in the African-Europeans compared to Caucasians.

To our knowledge, our study included the largest number of hypertensive African-Europeans for assessing the influence of increased afterload on LV geometry comparative with Caucasians. Only two studies previously specifically addressed this issue: Abassade et all included a small number of pts. (20 African-Europeans and 20 Caucasians), while SABRE study, which had a larger number of pts., included African-Caribbeans which may have genetic and phenotypic characteristics more similar to African-Americans given the historic and geographic factors [29,30].

4.1. Study limitations

Given the cross-sectional nature of the study, we cannot establish a temporal sequence of the observed cardiovascular abnormalities. Also, we couldn't accurately determine the duration of hypertension, which might have an important influence over the LV geometric pattern. Another possible limitation is that ambulatory blood pressure wasn't assessed, while there are data suggesting that Africans might have higher blood pressure during nighttime, despite similar values with Caucasians during daytime [34]. Other unmeasured confounding variables might have an important role in explaining the differences observed between African-Europeans and Caucasians. No normotensive control participants were included in this study, which limited our ability to further analyze the influence of hypertension on LV remodeling patterns. Furthermore, participants were receiving different classes of antihypertensive medication which may be a confounding factor. Lastly, arterial hypertension was defined as $\geq$140/90 mmHg, regardless last debated results showed in the SPRINT trial [35,36].

5. Conclusions

This study shows important differences in LV geometry between treated African-European and Caucasian hypertensive patients. Also, African-Europeans may have slightly different characteristics compared to African-Americans. The need for improved control of hypertension and better screening for end-organ damage in Africans are supported by this results that show a higher prevalence of cardiac hypertrophy among hypertensive Africans.

The relative contribution of race in LV remodeling remains uncertain. Further longitudinal studies are necessary to investigate the ethnic differences in hypertension etiology, severity and prognosis, and their impact regarding LV remodeling. This evidence could help clarify the differences in adverse outcomes that currently describe the two ethnic groups regarding the natural history of hypertension, and may have an impact on the treatment strategy and goals.

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