

Prevalence and Characterization of the Hypertrophic Cardiomyopathy Phenotype Assessed by Echocardiography in an Adult Brazilian Population: The ELSA-Brasil Study

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Abstract

Background: Hypertrophic cardiomyopathy (HCM) is a genetically inherited cardiac disease with significant clinical impact. Although its epidemiology is well described in developed countries, the prevalence of HCM in Latin American populations remains poorly defined.

Objective: To estimate the prevalence of the HCM phenotype and describe its clinical and echocardiographic characteristics in a large adult Brazilian cohort.

Methods: The ELSA-Brasil study is a multicenter cohort including 15,105 active and retired public servants aged 35-74 years at baseline. Comprehensive clinical data collection was conducted at each investigation center. Baseline echocardiographic examinations were reviewed, and participants were classified as having Definitive HCM if established diagnostic criteria were met. The prevalence of HCM and its 95% CI were calculated. Individuals meeting borderline criteria were classified as Possible HCM. A p-value < 0.05 was considered statistically significant.

Results: Among 3,267 participants who underwent echocardiography at baseline (mean age 64 ± 9 years; 53% women), five individuals met diagnostic criteria for HCM, of whom 80% were women. The estimated prevalence of HCM was 0.15% (95% CI, 0.05%-0.36%). Three participants exhibited a septal hypertrophy phenotype, and all cases of Definitive HCM showed abnormal electrocardiographic findings. A total of 19 additional participants were classified as Possible HCM, with no statistically significant differences compared with the Definitive HCM group.

Conclusion: The prevalence of HCM in this Brazilian cohort was approximately 1.5 per 1,000 individuals, consistent with rates reported in other populations. Most cases of HCM occurred in women and were frequently associated with metabolic conditions. These findings provide novel data on the epidemiology and clinical profile of HCM in Brazil and highlight the need for further research on genetic cardiomyopathies in Latin America.

Keywords: Hypertrophic Cardiomyopathy; Cross-Sectional Studies; Echocardiography; Heart Failure.

Introduction

Hypertrophic cardiomyopathy (HCM) is the most common genetically inherited heart disease. It is associated with an increased risk of sudden cardiac death, arrhythmias, and heart failure.¹ The diagnosis of HCM is primarily based on imaging evidence of left ventricular (LV) hypertrophy

that is typically asymmetric in distribution and cannot be attributed to other causes.² Transthoracic echocardiography (TTE) is the main diagnostic modality for identifying LV hypertrophy and other structural and functional features associated with HCM.³

The prevalence of HCM has traditionally been estimated at approximately 1 in 500 individuals, with a slightly higher occurrence in men.⁴ However, advances in genetic testing and cardiac imaging, particularly among relatives of affected individuals, have suggested higher prevalence rates, reaching up to 1 in 200.⁵ Most epidemiological studies have relied on TTE to identify asymmetric LV hypertrophy in population-based cohorts.⁶⁻⁸ Some investigations incorporated electrocardiogram (ECG) or clinical screening before echocardiography and reported similar prevalence estimates.⁸⁻¹⁰ Although the absolute number of cases

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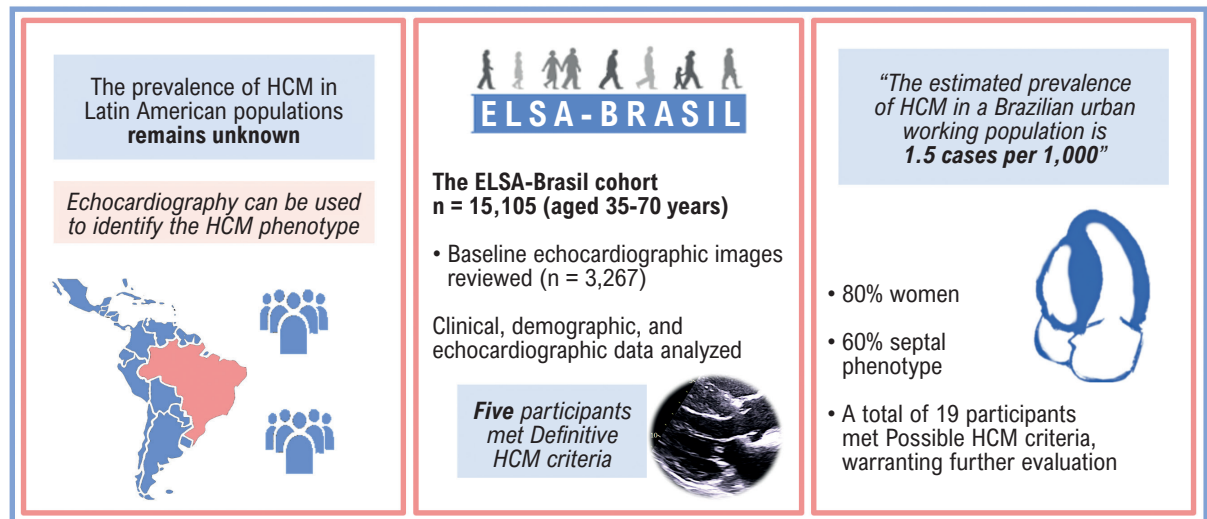
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Central Illustration: Prevalence and Characterization of the Hypertrophic Cardiomyopathy Phenotype Assessed by Echocardiography in an Adult Brazilian Population: The ELSA-Brasil Study



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HCM: hypertrophic cardiomyopathy.

identified in TTE-based studies has generally been small, often fewer than 25 individuals, these investigations provided foundational insights into the prevalence of HCM. More recently, large-scale analyses based on International Classification of Diseases, 10th Revision coding from health care and administrative databases have reported lower prevalence estimates of about 1 in 2,000 individuals.¹¹⁻¹⁴

In contrast, epidemiological data on the prevalence of HCM in Brazil and Latin America remain limited.¹⁵ Available studies from these regions have largely focused on descriptive analyses derived from HCM patient registries.¹⁶⁻¹⁹ Latin America and the Caribbean have a population of more than 650 million inhabitants and are characterized by substantial genetic heterogeneity, resulting from admixture among Indigenous populations and European and African immigrants.²⁰ Research on genetic diseases in this region is still in its early stages.²¹

The present study aimed to estimate the prevalence of the HCM phenotype identified by TTE among participants in the Brazilian Longitudinal Study of Adult Health (ELSA-Brasil). A descriptive, cross-sectional analysis was conducted to assess the prevalence of HCM in a community-dwelling, adult Brazilian population and characterize the associated phenotypic features.

Methods

Study population

The ELSA-Brasil cohort consists of public servants from six public educational and research institutions located in

Brazilian capital cities. ELSA-Brasil was designed to study chronic noncommunicable diseases. A total of 15,105 participants aged 35-74 years were enrolled, stratified by sex, age, and occupational category. Comprehensive baseline assessments were conducted at each investigation center between 2008 and 2010. Such assessments included demographic, clinical, laboratory, and ancillary examinations as well as follow-up visits. Previously published detailed descriptions of the study design, evaluations, and follow-up procedures are available.^{22,23} ELSA-Brasil complies with the latest version of the Declaration of Helsinki and was approved by the institutional research ethics committees at each participating center. All participants provided written informed consent.

We included participants who underwent complete TTE at baseline in the present analysis. This subgroup consisted of individuals aged 60 years or older (n = 2,933) and a randomly selected subsample of 10% of the entire cohort (n = 1,543).

Clinical and demographic parameters

Demographic and clinical data were obtained from the ELSA-Brasil database. Included variables were sex, age, anthropometric measurements, self-reported race (White, Black, Brown, or other), and family history of sudden death. Cardiometabolic risk profile was defined using data collected at the initial visit. Hypertension was defined as having a systolic blood pressure (SBP) ≥ 140 mmHg, a diastolic blood pressure ≥ 90 mmHg, or having used antihypertensive medication within the previous 2 weeks. Diabetes was defined as self-reported diabetes or the use of hypoglycemic

medication. It was also defined as having a fasting plasma glucose ≥ 126 mg/dL, a glycated hemoglobin level $\geq 6.5\%$, or 2-hour plasma glucose level ≥ 200 mg/dL after a 75-g oral glucose load. Obesity was defined as a body mass index ≥ 30 kg/m².

Laboratory data included measures of kidney function, total cholesterol, high-density lipoprotein cholesterol, and triglycerides. Cardiovascular risk was estimated using the pooled cohort equations outlined in the 2013 American College of Cardiology/American Heart Association Guideline on the Assessment of Cardiovascular Risk.²⁴

Electrocardiogram

A standard 12-lead ECG was performed at each investigation center during the initial visit by using Burdick Atria 6100 equipment (Davis Medical Electronics, California, USA). ECG tracings were then digitally transferred to a central reading center and stored in an electronic database. Automated analysis was performed using the Glasgow ECG Interpretation Algorithm. Abnormal recordings were then manually reviewed according to Minnesota Code criteria.^{25,26}

ECG findings were categorized as i) conduction abnormalities, namely atrioventricular block, complete or incomplete bundle branch block, axis deviation, delta waves, or pathological Q waves; ii) repolarization abnormalities, namely ST-segment deviations, enlarged and/or inverted T waves, or QT interval abnormalities; and iii) LV hypertrophy, which was defined according to Minnesota criteria.

Transthoracic echocardiography

Between 2008 and 2010, trained echocardiographers performed all baseline TTE examinations at the investigation centers using standardized ultrasound systems (Aplio XG; Toshiba Corporation, Tochigi, Japan). Preliminary interpretations were recorded locally and forwarded to the ELSA-Brasil echocardiography reading center together with the image datasets. Final analyses were conducted using a dedicated workstation (ComPACS 10.5, Medimatic Srl, Italy).

Left atrial diameter, LV wall thickness, and chamber dimensions were measured from the parasternal long-axis view. Atrial volumes were calculated using Simpson's method from apical views. Color and spectral Doppler recordings of the mitral, aortic, and tricuspid flows, as well as the mitral annular velocities, were obtained. Readers also qualitatively assessed the presence of LV hypertrophy, abnormal intracardiac gradients, wall motion abnormalities, valvular disease, and other relevant findings. Image acquisition and interpretation protocols were developed in accordance with contemporary echocardiographic guidelines.²⁷

Definition of the hypertrophic phenotype

A Definitive HCM phenotype was defined according to the following echocardiographic criteria: i) asymmetric LV wall thickness ≥ 15 mm; or ii) unequivocal apical hypertrophy with wall thickness exceeding that of the basal segments.²⁸

Supporting features included a contralateral wall thickness ratio greater than 1.5 and the presence of systolic anterior motion (SAM) of the mitral valve, which is defined as systolic

displacement of the mitral valve leaflet or chordal structures toward the LV outflow tract.

A Possible HCM phenotype was defined as asymmetric LV wall thickness of 13-15 mm; symmetric LV wall thickness ≥ 15 mm; or the presence of secondary features suggestive of a primary cardiomyopathy, including SAM, myocardial crypts, or papillary muscle displacement.

In order to identify cases of HCM, two experienced echocardiographers (A.B. and M.F.) independently reviewed all echocardiographic studies with a reported LV wall thickness ≥ 13 mm at the central reading center ($n = 170$). Discrepancies were resolved by consensus. Participants with an alternative cause of LV hypertrophy, such as stage 3 hypertension or moderate-to-severe left-sided valvular disease (excluding SAM), were not classified as having an HCM phenotype. Studies with inadequate image quality or foreshortened views were excluded.

Definitive and Possible cases of HCM were further classified into septal, apical, or concentric subtypes according to the predominant region of LV hypertrophy.³ The concentric subtype was defined by hypertrophy involving two opposing LV walls. SAM was identified by anterior displacement of the mitral valve leaflet toward the LV outflow tract during systole. Resting subvalvular obstruction was quantified in mmHg using continuous-wave Doppler ultrasound. Quantitative measurements of cardiac chambers and diastolic function parameters were obtained from the final ELSA-Brasil echocardiography database.

Statistical analysis

The prevalence of HCM was calculated as the proportion of individuals who met the Definitive diagnostic criteria, relative to the total number of participants included in the analysis. 95% CIs were estimated by using the Wilson score method.²⁹

Descriptive statistics were used to characterize the overall study population and the HCM subgroups (Definitive and Possible). Categorical variables were presented as absolute frequencies and percentages, which were then compared using the chi-square test. Normality of continuous variables was assessed using the Shapiro-Wilk test, evaluation of skewness and kurtosis, and visual inspection of histograms. Normally distributed variables are presented as mean $\pm$ standard deviation and were compared using an unpaired Student's *t* test. Non-normally distributed variables are presented as median (interquartile range) and were compared using the Mann-Whitney *U* test. All tests were two-tailed, and *p*-values < 0.05 were considered statistically significant. Statistical analyses were performed using Stata version 14.0 (StataCorp LP, College Station, TX, USA).

Results

Of the 4,476 participants who were initially eligible for the present analysis, 3,267 (mean age 64 ± 9 years; 53% women) completed baseline TTE with interpretable archived images (Figure 1). No significant differences in demographic or clinical characteristics were observed between participants included in the analysis and those excluded (Supplemental Table 1). Table 1 presents the overall characteristics of the study population. Table 2 presents detailed echocardiographic findings.

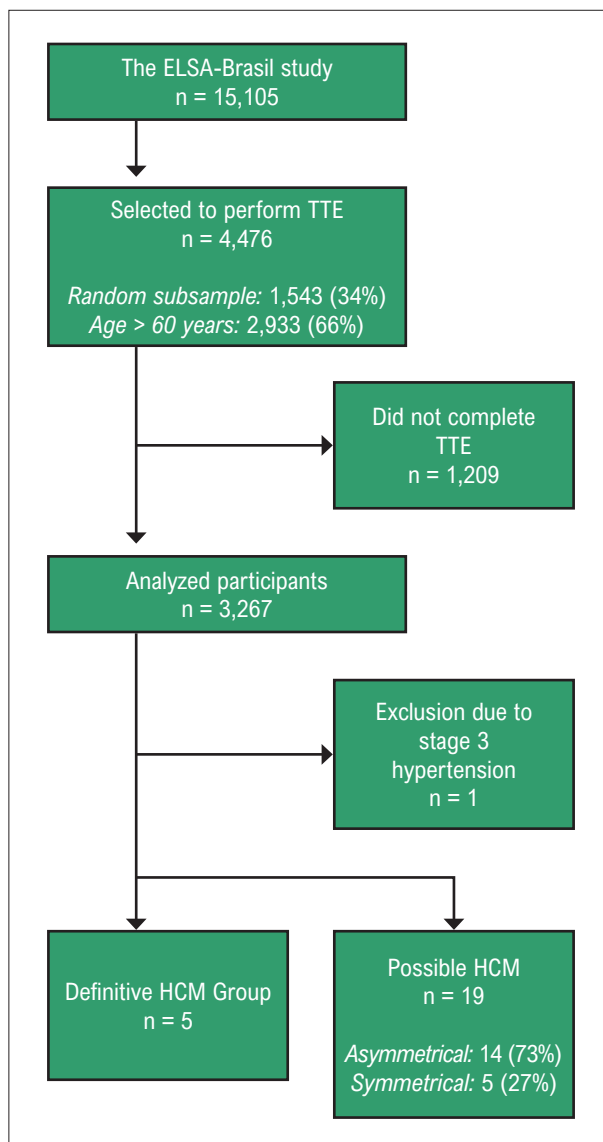


Figure 1 – Study flowchart. HCM: hypertrophic cardiomyopathy; TTE: transthoracic echocardiographic examination.

Five participants met the echocardiographic criteria for a Definitive HCM phenotype, yielding an estimated prevalence of 0.15% (95% CI, 0.05%-0.36%), corresponding to approximately 1 case per 667 individuals (Central Illustration). One participant with a concentric hypertrophy pattern was not classified as having HCM because of concomitant stage 3 hypertension. Participants classified as having Definitive HCM had a mean age of 64 ± 3 years, and four were women (80%). Table 3 details individual-level clinical and echocardiographic characteristics.

Among Definitive cases of HCM, septal hypertrophy was the most frequent subtype, observed in three participants. None of these individuals exhibited resting LV outflow tract obstruction; however, one participant showed typical SAM of the mitral valve with mild mitral regurgitation. One participant

presented with an apical phenotype, characterized by a wall thickness of 11 mm, systolic obliteration of the apical LV segments, and absence of the expected basal-to-apical tapering of wall thickness. The remaining case exhibited hypertrophy involving the apical, septal, and inferior LV regions, without wall motion abnormalities, and was classified as a concentric HCM phenotype. Compared with the overall study population, Definitive cases of HCM had smaller LV cavity dimensions, a high prevalence of LV hypertrophy, and evidence of concentric remodeling. Features of diastolic dysfunction were also observed, including increased left atrial volumes, reduced early diastolic mitral annular velocity (e'), and elevated E/e' ratios.

All participants with a Definitive HCM phenotype were in sinus rhythm and reported no history of atrial fibrillation or flutter. Repolarization abnormalities were evident in all cases on ECG, though none met the Minnesota criteria for LV hypertrophy. Conduction abnormalities were identified in four of the five individuals.

A total of 19 participants were classified as having a Possible HCM phenotype. This group exhibited a high prevalence of hypertension (94%; mean SBP 149 ± 26 mmHg), diabetes (50%), obesity (41%), and chronic kidney disease stage 3 or greater (37%). A total of 14 of these individuals (73%) had asymmetric thickening of the anterior interventricular septum that did not exceed the 15-mm diagnostic threshold. The remaining five participants exhibited a concentric pattern with symmetric involvement of the LV walls.

Tables 1 and 2 present comparisons between the Definitive and Possible HCM groups. Both groups showed high rates of hypertension and excess body weight, with no statistically significant differences. Similar patterns were observed for family history of sudden death and ECG findings. Participants in the Definitive HCM group tended to have smaller LV cavity dimensions and more pronounced concentric remodeling. However, the small number of Definitive cases of HCM resulted in wide confidence intervals and limited statistical power for between-group comparisons.

Discussion

In this echocardiography-based analysis of a large cohort of active and retired public employees from major Brazilian cities, we found that the prevalence of HCM was 1.5 per 1,000 individuals. This estimate aligns with findings from significant major population-based HCM studies conducted in the Northern Hemisphere.^{6,10}

Epidemiological data on the prevalence of HCM in Latin America remain scarce. Nevertheless, the echocardiographic patterns observed in the present study are broadly consistent with registry-based reports from the region. Espinosa-Zabaleta et al.¹⁵ described a predominance of asymmetric, nonobstructive septal hypertrophy among Mexican patients. Two Brazilian studies similarly reported similar findings: a predominance of women accounting for 52% of cases in the study by Arteaga et al.¹⁶ and 60% in the study by Mattos et al.,¹⁷ with septal phenotypes being most frequent. In contrast, de Leon et al.³⁰ described a Colombian cohort characterized by a predominance of men, a higher frequency of obstructive

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Table 1 – Demographic, clinical, and ECG characteristics of the study population and comparison between Definitive and Possible HCM groups in the ELSA-Brasil study

Characteristic	Study population (n = 3,267)	Definitive HCM (n = 5)	Possible HCM (n = 19)	p-value*
Age, years	62 [57-67]	64 [61-67]	64 [62-71]	0.45
Female sex, n (%)	1,728 (53)	4 (80)	6 (32)	0.051
Race				0.34
Black	497 (15)	2 (40)	6 (32)	
Brown	801 (25)	2 (40)	3 (16)	
White	1,798 (56)	1 (20)	10 (53)	
Family history of sudden death	972 (30)	3 (60)	7 (37)	0.35
Hypertension	1,667 (51)	4 (80)	18 (95)	0.29
Diabetes mellitus	764 (23)	2 (40)	11 (58)	0.47
Obesity	724 (22)	3 (60)	8 (41)	0.46
Overweight	1,399 (43)	2 (40)	7 (37)	0.89
BMI, kg/m ²	27.0 ± 4.5	32.5 ± 9.1	29.8 ± 5.9	0.42
CKD [†]	364 (11)	0	7 (37)	0.11
eGFR, mL/min/1.73 m ²	79 [68-90]	67 [62-78]	69 [58-84]	0.97
SBP, mmHg	126 ± 18	139 ± 11	149 ± 27	0.47
DBP, mmHg	76 ± 10	86 ± 9	84 ± 15	0.75
10-year ASCVD risk, %	8.4 [3.5-16.8]	8.4 [7.6-26.9]	23.5 [14.6-41.0]	0.12
ECG				
HR, bpm	65 ± 10	73 ± 14	67 ± 8	0.21
Atrial fibrillation or flutter	29 (0.9)	0	0	—
Conduction abnormalities	1,107 (34)	4 (80)	13 (68)	0.61
Repolarization abnormalities	945 (29)	5 (100)	10 (53)	0.05
LV hypertrophy	22 (0.7)	0	0	—

ASCVD: atherosclerotic cardiovascular disease; BMI: body mass index; CKD: chronic kidney disease; DBP: diastolic blood pressure; ECG: electrocardiogram; eGFR: estimated glomerular filtration rate; HR: heart rate; LV: left ventricle; SBP: systolic blood pressure. Data are presented as n (%), mean ± standard deviation, or median [interquartile range]. *p-value for comparison between Definitive HCM and Possible HCM groups. [†]Defined as estimated glomerular filtration rate < 60 mL/min/1.73 m².

septal phenotypes, and a low prevalence of overweight and obesity (8%).

In our study, most individuals with Definitive HCM were women in late adulthood. Although HCM has traditionally been considered a disease of younger individuals,³¹ accumulating evidence indicates an increasing incidence and prevalence in older populations, which is consistent with our findings. Moon et al.¹² demonstrated a strong association between age and *de novo* HCM diagnosis in a Korean population. Similarly, Bai et al.¹³ reported a progressive increase in both incidence and prevalence over a 10-year period in a stable Chinese cohort. Age-related penetrance is a recognized feature of HCM; genotype-positive, phenotype-negative individuals may develop overt disease later in life.^{32,33} While the older age distribution of the ELSA-Brasil echocardiography sample

may partially explain the observed prevalence, our findings may also reflect a relatively benign clinical course among individuals diagnosed later in life.

Participants in the ELSA-Brasil study exhibited a high overall burden of metabolic disorders.²² Consistent with this profile, more than half of individuals with Definitive HCM and nearly all those classified as Possible HCM had arterial hypertension. Hypertension is common in older patients with HCM and may influence phenotypic expression and clinical presentation.³⁴ A Spanish HCM cohort reported a lower prevalence of sarcomeric mutations among hypertensive individuals, suggesting potential phenotypic modulation.³⁵ In addition, most cases of HCM in our study were overweight or obese. Obesity has become increasingly prevalent worldwide³⁶ and has been reported in 25%-37%

Table 2 – Echocardiographic parameters of the study population and comparison between Definitive and Possible HCM groups in the ELSA-Brasil study

Characteristic	Study population (n = 3,267)	Definitive HCM (n = 5)	Possible HCM (n = 19)	p-value*
LV septal thickness, mm	9.9 ± 1.7	17.0 ± 2.9	15.7 ± 2.0	0.25
LV posterior wall thickness, mm	9.1 ± 1.4	13.2 ± 3.3	12.0 ± 2.1	0.35
LV end-diastolic diameter, mm	44.9 ± 4.9	39.0 ± 5.3	44.1 ± 5.6	0.10
LV end-systolic diameter, mm	28.2 ± 4.7	24.1 ± 7.3	29.7 ± 5.1	0.07
LV ejection fraction, % (Teichholz)	66 ± 9	68 ± 8	62 ± 11	0.26
RWT	0.41 ± 0.08	0.69 ± 0.21	0.55 ± 0.13	0.09
LAVI, mL/m ²	27.2 ± 8.5	33.3 ± 11.2	29.5 ± 8.7	0.43
LV mass index, g/m ²	82 ± 21	130 ± 50	130 ± 31	0.98
E-wave velocity, cm/s	67 ± 16	63 ± 16.5	63 ± 16	0.96
E/e' ratio	8.3 ± 2.5	13.1 ± 6.0	12.1 ± 3.1	0.64

HCM: hypertrophic cardiomyopathy; LAVI: left atrial volume index; LV: left ventricle; RWT: relative wall thickness. Data are presented as mean ± standard deviation. *p-value for comparison between Definitive HCM and Possible HCM groups.

Table 3 – Clinical and echocardiographic findings of the Definitive HCM phenotype individuals in the ELSA-Brasil cohort

Participant	Age, years	Sex	Race	Maximum wall thickness, mm	Septal wall thickness, mm	Posterior wall thickness, mm	LV diameter, mm	Phenotype	LV outflow tract gradient, mmHg	BMI, kg/m ²	SBP, mmHg
I	62	Female	Black	17	17	11	43	Septal	26	47	138
II	70	Female	Black	15	15	11	31	Septal	18	31	158
III	64	Female	White	20	18	19	44	Septal	< 15	25	138
IV	65	Female	Brown	15	15	12	38	Concentric	< 15	26	135
V	60	Male	Brown	13	10	9	38	Apical	< 15	32	128

BMI: body mass index; HCM: hypertrophic cardiomyopathy; LV: left ventricle; SBP: systolic blood pressure.

of patients with HCM.^{37,38} Excess body weight has been linked to increased LV wall thickness, chamber dilation, and poorer functional status.³⁹ It may also exacerbate LV outflow tract gradients and atrial fibrillation burden.³⁸ Park et al.⁴⁰ observed higher rates of HCM diagnosis among overweight and obese individuals. Recent studies have proposed that obesity may contribute to the development or expression of an HCM phenotype, even in genotype-negative individuals.^{37,41} For example, De Faria et al.⁴² reported higher obesity rates and later age at diagnosis among genotype-negative HCM patients compared with genotype-positive cases. Taken together, these findings suggest that altered metabolic and hemodynamic loading conditions associated with obesity and hypertension may influence the expression of the HCM phenotype.

The inclusion of individuals classified as Possible HCM was intended as an exploratory analysis. This group showed a high prevalence of hypertension, obesity, and abnormal

kidney function, conditions that are often associated with LV hypertrophy. Despite limited statistical power, these individuals appeared to exhibit a distinct pattern, with relatively more eccentric LV hypertrophy, which is consistent with the adaptive remodeling typically seen in hypertensive heart disease.⁴³ Nonetheless, approximately three-quarters of Possible cases of HCM showed asymmetric hypertrophy, which may be partly due to the presence of a septal bulge, a common finding in older adults.⁴⁴

An overlap between Definitive and Possible HCM phenotypes cannot be ruled out. It is not possible to determine from the present data whether some findings in the Possible HCM group represent early or milder forms of true HCM or instead reflect age-related or comorbidity-driven LV remodeling. This overlap underscores a frequent clinical challenge in distinguishing HCM from other causes of LV hypertrophy based solely on imaging findings.

A normal ECG is uncommon in HCM;⁴⁵ all individuals with Definitive HCM in our study had abnormal ECG findings, including both repolarization and conduction abnormalities. However, none of them met the Minnesota criteria for LV hypertrophy, which is in line with previous reports showing limited sensitivity of voltage-based criteria in HCM.⁴⁶ The high prevalence of overweight and obesity among cases of HCM may have further reduced ECG sensitivity, as increased body habitus can attenuate QRS voltages.

Several limitations merit consideration. The ELSA-Brasil cohort consists of active and retired public servants from major urban centers, so it may not be fully representative of the Brazilian population as a whole. Individuals with severe disability or limited work capacity may be underrepresented. Participants younger than 35 years were not included, which may have excluded cases of early-onset HCM, often associated with more severe disease.^{47,48} Echocardiographic sampling prioritized older individuals, who are more likely to exhibit cardiovascular abnormalities and LV hypertrophy. To reduce selection bias, a predefined random subsample of 10% of the cohort was also included. Finally, the study protocol was not designed to establish a Definitive diagnosis of HCM according to current standards because genetic testing, a detailed clinical evaluation, and advanced imaging modalities were unavailable. Therefore, our findings reflect the prevalence of an HCM echocardiographic phenotype rather than confirmed clinical HCM; however, this approach is comparable to that used in earlier population-based prevalence studies. The small number of Definitive cases limited subgroup analyses and resulted in wide confidence intervals around prevalence estimates.

Conclusions

This echocardiography-based analysis of a Brazilian adult population estimated the prevalence of an HCM phenotype at approximately 1.5 cases per 1,000 individuals (95% CI, 0.5-3.6 per 1,000). The identified cases were predominantly female and exhibited a high prevalence of hypertension and excess body weight. Additionally, we identified a subgroup of individuals with borderline echocardiographic features suggestive of HCM whose clinical significance warrants further investigation.

To our knowledge, this study provides the most robust population-based estimate of the prevalence of HCM in Latin America to date. Further research on genetic cardiomyopathies is necessary to better identify, characterize, and manage affected individuals, especially given the growing number of treatment options for HCM.

Author Contributions

Conception and design of the research: Bertuol Filho AA, Santos ABS, Duncan BB, Foppa M; Acquisition of data:

Bertuol Filho AA, Foppa M; Analysis and interpretation of the data: Bertuol Filho AA, Pereira AC, Santos ABS, Ribeiro ALP, Scolari FL, Foppa M; Statistical analysis: Bertuol Filho AA, Santos ABS, Foppa M; Writing of the manuscript: Bertuol Filho AA, Santos ABS, Duncan BB, Pianca EG, Scolari FL, Foppa M; Critical revision of the manuscript for content: Bertuol Filho AA, Pereira AC, Santos ABS, Ribeiro ALP, Duncan BB, Pianca EG, Scolari FL, Foppa M.

Potential conflict of interest

No potential conflict of interest relevant to this article was reported.

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Study association

This article is part of the thesis of master submitted by Amadeu Antonio Bertuol Filho, from Universidade Federal do Rio Grande do Sul.

Ethics approval and consent to participate

This study was approved by the Ethics Committees of the participating institutions: i) Public Health Institute, Universidade Federal da Bahia (protocol 027/06; May 26, 2006); ii) Fundação Oswaldo Cruz (protocol 343/06; September 18, 2006); iii) University Hospital, Universidade de São Paulo (protocol 659/06; May 19, 2006); iv) Universidade Federal de Minas Gerais (protocol 186; June 28, 2006); v) Center for Health Sciences, Universidade Federal do Espírito Santo (protocol 041/06; May 31 2006); vi) Hospital de Clínicas de Porto Alegre (protocol 194/06; May 15, 2006). All the procedures in this study were in accordance with the 1975 Helsinki Declaration, updated in 2013. Informed consent was obtained from all participants included in the study.

Use of Artificial Intelligence

The authors did not use any artificial intelligence tools in the development of this work.

Data Availability Statement

The underlying content of the research text is contained within the manuscript.

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