The Egyptian Journal of Hospital Medicine (April 2025) Vol. 99, Page 1597-1603

Clinical And Echocardiography Parameters Differentiating Left Ventricular Hypertrophy Among Athletes And Hypertensive Patients

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ABSTRACT

Background: Left ventricular hypertrophy (LVH) is a common adaptive response in both athletes and hypertensive patients. However, distinguishing physiological from pathological LVH is critical to avoid misdiagnosis and unnecessary intervention.

Objective: This study aimed to identify clinical and echocardiographic parameters that differentiate LVH in athletes from that in hypertensive individuals.

Subjects and methods: In this observational cross-sectional study, 100 male subjects with echocardiographically confirmed LVH were enrolled: 50 competitive endurance / strength-training athletes and 50 hypertensive patients. All participants underwent comprehensive clinical evaluation and standard transthoracic echocardiography, including speckle-tracking analysis for global longitudinal strain (GLS). LV mass index, wall thickness, diastolic function, and strain parameters were compared between groups.

Results: Athletes were significantly younger (35.8 ± 6 vs. 44 ± 6.5 years, $p = 0.004$) and taller (177.7 ± 5.8 vs. 174.6 ± 5.3 cm, $p = 0.007$). Resting heart rate and blood pressures were lower in athletes ($p < 0.001$). Athletes exhibited larger LVEDD (53.9 ± 3.6 vs. 50.3 ± 3.2 mm, $p < 0.001$) and higher LV mass (259.4 ± 57.9 vs. 247.6 ± 34.7 g, $p = 0.001$), while hypertensive patients showed increased septal thickness (12.93 ± 0.70 vs. 11.70 ± 1.67 mm, $p < 0.001$) and relative wall thickness. Diastolic function was better preserved in athletes, as evidenced by higher e′ velocity (13.98 ± 1.74 vs. 9.31 ± 2.08 cm/s, $p < 0.001$) and lower E/e′ ratio (5.51 ± 1.34 vs. 9.00 ± 2.27, $p < 0.001$). GLS was significantly reduced in hypertensive patients ($–17.5 \pm 1.2$ vs. $–18.3 \pm 1.8\%$, $p = 0.036$).

Conclusion: A combination of clinical features, diastolic function parameters, and myocardial strain imaging effectively differentiates athlete's heart from hypertensive LVH. GLS and diastolic indices offer incremental diagnostic value in distinguishing physiological adaptation from early hypertensive remodelling.

Keywords: Left ventricular hypertrophy, Athlete's heart, Hypertension, Speckle-tracking echocardiography, Global longitudinal strain.

INTRODUCTION

Endurance exercise elicits a range of cardiovascular adaptations, with the most prominent being an increase in maximal cardiac output. This physiological enhancement is primarily attributed to cardiac enlargement, left ventricular hypertrophy (LVH), and improved myocardial contractile function [1]. Conversely, hypertension—one of the leading modifiable risk factors for cardiovascular morbidity—places chronic pressure overload on the left ventricle, which also culminates in LVH [2]. Notably, the structural cardiac changes induced by athletic training, such as increased left ventricular mass (LVM), augmented wall thickness, and dilation of the cardiac chambers, may closely mimic the remodeling patterns observed in hypertensive heart disease, creating diagnostic challenges in clinical practice [3].

It is important to recognize that elevated blood pressure can occasionally be observed in athletes, and in fact, it remains the most frequently encountered abnormality during pre-participation cardiovascular evaluations (PPE). This occurs despite the general blood pressure-lowering effects of habitual exercise and training [4].

Although debates persist regarding the predictive value of PPE, comprehensive cardiovascular screening remains a crucial step in safeguarding athletes' health and performance trajectories.

Misclassification, however, can result in unjustified disqualification from sports or, conversely, missed detection of underlying pathology, both of which may carry serious implications, including the risk of sudden cardiac death (SCD) [5]. While both conditions—athlete's heart and hypertensive heart disease—result in LVH, the underlying mechanisms and patterns of adaptation diverge significantly. Athlete's heart represents a physiological response to increased workload, whereas hypertension leads to pathological remodeling [6].

This study aimed to delineate the structural and functional cardiac changes associated with endurance / strength training and hypertension to better differentiate physiological adaptation from disease.

SUBJECTS AND METHODS

Design and population: This observational cross-sectional study was conducted at Cardiology Department, Ain Shams University Hospital. It was carried out on 100 subjects with LVH through the period from July 2024 to January 2025.

Participants with LVH were identified based on either LVM index exceeding 115 g/m² or the presence of symmetric hypertrophy of the interventricular septum and posterior wall measuring greater than 11 mm [7]. Eligible subjects were categorized into two groups:

Received: 04/11/2024
Accepted: 05/01/2025

Group 1 consisted of athletic individuals, while **group 2** included patients diagnosed with hypertension.

Inclusion criteria:

Group 1 (Athletes): Males aged 18–65 years who had consistently participated in combined endurance and strength training for more than 6 hours per week over the past 12 months, with no prior clinical diagnosis of hypertension.

Group 2 (Hypertensive patients): Males aged 18–65 years with a documented history of hypertension, defined by systolic blood pressure ≥ 140 mmHg and/or diastolic blood pressure ≥ 90 mmHg on repeated office measurements, in accordance with established guidelines [8]. These patients were also required to have ischemic heart disease excluded via a non-invasive diagnostic modality within the preceding six months.

Exclusion criteria: Subjects who had moderate or severe valvular heart disease, a history of CAD or MI, DM, LVEF below 50% as assessed by echocardiography, any form of cardiomyopathy, CHD and prior open-heart surgery, or current arrhythmia.

All patients were subjected to:

History and clinical examination: A thorough history was obtained, including demographic data, smoking status, and detailed risk profiles. Additional information regarding physical activity, exercise training, heart rate and both systolic and diastolic blood pressures.

Echocardiographic Assessment: Standard transthoracic 2D Echocardiographic examination: All participants underwent a comprehensive transthoracic echocardiographic assessment with simultaneous ECG monitoring. The examinations were primarily performed with patients in the left lateral decubitus position using a Vivid E95 ultrasound system equipped with an M4S matrix sector array transducer operating at a frequency of 2.5 MHz

Image acquisition was standardized at a depth of **16 cm**, encompassing parasternal long- and short-axis views, as well as apical two-, three-, and four-chamber windows. Two-dimensional (2D) and color Doppler datasets, synchronized with the QRS complex, were recorded and stored in cine-loop format. A full echocardiographic protocol—including M-mode, 2D imaging, tissue Doppler imaging (TDI), and both pulsed-wave and continuous-wave Doppler interrogation across cardiac valves—was performed in accordance with the guidelines established by the American Society of Echocardiography [7].

Conventional left ventricle echocardiographic parameters [7]:

1. **LV end diastolic diameter (EDD) and LV end systolic diameter (ESD):** Using M-mode at long axis parasternal window at the level of papillary muscles.
2. **LVEF:** Was calculated using biplane modified Simpson's method.
3. **LV mass and LV mass index:**

The following equation was used to determine LV mass in grams:

LV mass= 0.8x [1.04x "(PWT+ SWT+ LVIDd) ^{3}- (LVIDd) ^{3}"] + 0.6

Where LVIDd is the internal dimension at end diastole, PWT is the posterior wall thickness, SWT is inter ventricular septal wall thickness, 1.04 is the specific gravity of the myocardium, and 0.8 is the correction factor. All measurements were made at end-diastole (at the peak of the R-wave) in centimeters.

LV mass index=LV mass/ BSA (g/m^2).

4. Left atrial diameter (LAD): The anterior–posterior dimension of the left atrium was assessed using either M-mode or two-dimensional echocardiography, acquired from the parasternal long-axis view at end-ventricular systole.

5) Doppler measurements including: Mitral inflow parameters were evaluated using pulsed-wave Doppler to measure peak early (E) and late (A) diastolic filling velocities, and the E/A ratio was subsequently calculated. In addition, early diastolic mitral annular velocity (e′) was obtained using TDI, and the E/e′ ratio was derived to estimate LV filling pressures [9].

Longitudinal strain parameters:

GLS of the LV was assessed using speckle-tracking echocardiography. For each subject, cine loops of at least three consecutive, ECG-gated cardiac cycles were obtained from apical four-, three-, and two-chamber views. Upon image acquisition, the software automatically selected the end-systolic frame; manual correction was applied when necessary to optimize accuracy. A region of interest (ROI) encompassing the full thickness of the myocardium was then defined by the software to compute GLS values [10].

Ethical considerations: The study was done after being accepted by the Research Ethics Committee, Ain Shams University. All patients provided written informed consents prior to their enrolment. The consent form explicitly outlined their agreement to participate in the study and for the publication of data, ensuring protection of their confidentiality and privacy. This work has been carried out in accordance with The Code of Ethics of the World Medical Association (Declaration of Helsinki) for studies involving humans.

Data management and statistical analysis

Data analysis was conducted using RStudio version 2.3.2. Prior to analysis, all data were collected,

coded, reviewed for accuracy, and entered into the software. The normality of quantitative variables was evaluated using the Shapiro–Wilk test. Continuous variables with a normal distribution were summarized as mean, standard deviation, and range, whereas non-normally distributed variables were reported as median and interquartile range (IQR).

Categorical variables were expressed as frequencies and percentages. For inferential statistics, the Chi-square test was employed to compare proportions between groups, Fisher's exact test was used in cases where the expected cell count was less than 5. Comparisons of paired quantitative data were performed using the paired t-test for normally distributed variables and the Wilcoxon signed-rank test for non-parametric data. Statistical significance was determined at a 95% confidence level, with a p-value ≤ 0.05 considered significant.

RESULTS

Demographics: The athletic group were younger than the hypertensive group (35.8 ± 6 vs 44 ± 6.5, p = 0.004) and had lower wight than hypertensive group (76.3 ± 7.5 vs. 80.1 ± 7.1.kg, p = 0.012) and taller (177.7 ± 5.8 vs 174.6 ± 5.3 vs. cm, p =0.007), with slightly lower BMI and higher BSA (Table 1).

Clinical parameters: Athletic group had statistically significant lower heart rate than hypertensive group (65.7 ± 9.6 vs.79.9 ± 7 bpm, p < 0.001), lower systolic blood pressure (115.6 ± 7.9 vs. 139 ± 10.5 mmHg, p < 0.001), and lower diastolic blood pressure (68.9 ± 7.7 vs. 84.4 ± 7.1 mmHg, p < 0.001) (Table 2).

Conventional Echo parameters: Regarding LV dimensions, athletic group had a larger LV LVEDD and LVESDD compared to hypertensive group (53.9 ± 3.6 vs 50.3 ± 3.2 mm, p < 0.001, 30.6 ± 3.5 vs. 33.9 ± 3.4 mm, p < 0.001 respectively).

LV wall thickness & LV mass: The hypertensive group had increased interventricular septum thickness than athletes' group (12.93 ± 0.70 vs. 11.70 ± 1.67 mm, p < 0.001) and higher relative wall thickness (0.47 ± 0.05 vs. 0.43 ± 0.05, p < 0.001). However, LV mass was higher in athletic group than in hypertensive group (259.4 ± 57.9 vs. 247.6 ± 34.7 g, p = 0.001).LV mass index were comparable in both groups.

Diastolic function: The hypertensive group had lower e' wave velocity than athletic group (9.31 ± 2.08 vs. 13.98 ± 1.74 cm/s, p < 0.001) and a higher E/e' ratio (9.00 ± 2.27 vs. 5.51 ± 1.34, p < 0.001), while the athletic group had significantly lower A wave velocity (55± 5.1 vs 74.6 ± 10.7, p < 0.001).

Left atrial diameter: The hypertensive group had slightly largely LA diameter than athletic group although no statistically significant correlation found.

LV global longitudinal strain (GLS): Slightly reduced in hypertensive patients than athletes' group (-17.5 ± 1.2 vs. -18.3 ± 1.8, p = 0.036) (Table 1 and figures 1-3).

Table (1): Comparison between the two studied groups according to demographic characteristics

	Athletes group (N = 50)	**Hypertensive group (N = 50)**	**p-value**
Age in years			**<0.004**
Mean ± SD	35.8 ± 6	44 ± 6.5	
Weight in Kg			**0.012**
Mean ± SD	76.3 ± 7.5	80.1 ± 7.1	
Height in cm			**0.007*[t]**
Mean ± SD	177.7 ± 5.8	174.6 ± 5.3	
BMI (KG/m^2)			0.394[t]
Mean ± SD	23.02 ± 2.16	25.38 ± 2.11	
Body surface area (m^2)			**0.004*[t]**
Mean ± SD	1.95 ± 0.11	1.92 ± 0.11	

n: number, SD: Standard Deviation, Kg: Kilogram, cm: centimeter, BMI: Body Mass Index, m²: square meter,
*: Significant P-value, t: Student's t-test.

Table (2): Comparison between the two studied groups regarding to clinical and echocardiographic features:

	Athletes group (N = 50)	Hypertensive group (N = 50)	p-value
HR (min)			**<0.001*[w]**
Mean ± SD	65.7 ± 9.6	79.9 ± 7	
SBP (mmHg)			**<0.001*[w]**
Mean ± SD	115.9 ± 7.9	139 ± 10.5	
DBP (mmHg)			**<0.001*[w]**
Mean ± SD	68.9 ± 7.7	84.4 ± 7.1	
LV end diastolic diameter (mm)			**<0.001*[w]**
Mean ± SD	53.9 ± 3.6	50.3 ± 3.2	
LV end systolic diameter (mm)			**<0.001*[t]**
Mean ± SD	30.6 ± 3.5	33.9 ± 3.4	
LVEF			0.11[t]
Mean ± SD	68.6 ± 6.2	66.7 ± 5.4	
PWT (mm)			0.71[w]
Mean ± SD	11.71 ± 1.53	11.84 ± 1.17	
IVS (mm)			**<0.001*[w]**
Mean ± SD	11.70 ± 1.67	12.93 ± 0.70	
Relative wall thickness			**<0.001*[w]**
Mean ± SD	0.43 ± 0.05	0.47 ± 0.05	
LV mass index			0.10[w]
Mean ± SD	131.3 ± 30.5	129.6 ± 22.3	
LV mass			**0.001*[w]**
Mean ± SD	259.4 ± 57.9	247.6 ± 34.7	
E wave			0.066
Mean ± SD	75.2 ± 12.4	81.8 ± 22	
A wave			**<0.001**
Mean ± SD	55± 5.1	74.6 ± 10.7	
E/A ratio			0.58[t]
Mean ± SD	1.10 ± 0.16	1.13 ± 0.35	
e' wave (cm /sec.)			**<0.001*[w]**
Mean ± SD	13.98 ± 1.74	9.31 ± 2.08	
E/e' Ratio			**<0.001*[w]**
Mean ± SD	5.51 ± 1.34	9.00 ± 2.27	
LV GLS			**0.03**
Mean ± SD	-18.3 ± 1.8	-17.5 ± 1.2	
Left atrial diameter (mm.)		39.0 ± 3.0	0.48
Mean ± SD	37.5 ± 7.0	39.0 ± 3.0	

n: number, HR: Heart Rate, bpm: beats per minute, SBP: Systolic Blood Pressure, DBP: Diastolic Blood Pressure, mmHg: millimeters of mercury, LV: Left Ventricle, LVEF: Left Ventricular Ejection Fraction, PWT: Posterior Wall Thickness, IVS: Interventricular Septum, mm: millimeter, LVEDD: Left Ventricular End-Diastolic Diameter, LVESD: Left Ventricular End-Systolic Diameter, E wave: Early diastolic mitral inflow velocity, A wave: Late diastolic mitral inflow velocity, E/A ratio: Ratio of early to late mitral inflow velocity, e′: Early diastolic mitral annular velocity, E/e′ ratio: Ratio of mitral inflow E wave to tissue Doppler e′ wave, LV GLS: Left Ventricular Global Longitudinal Strain, *: Significant P-value, t: Student's t-test, w: Mann–Whitney U test.

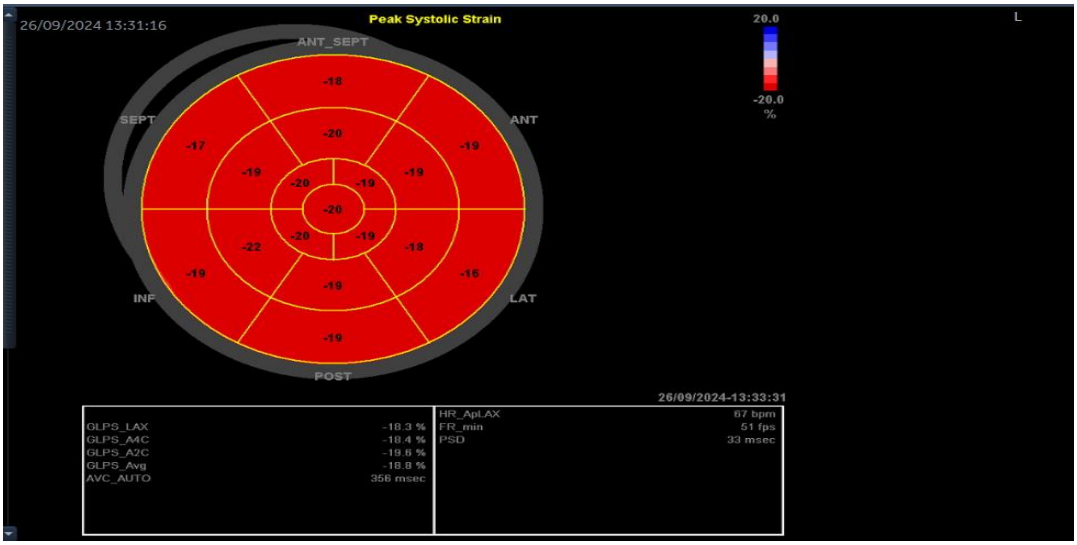

Figure (1): GLS among athletic group.

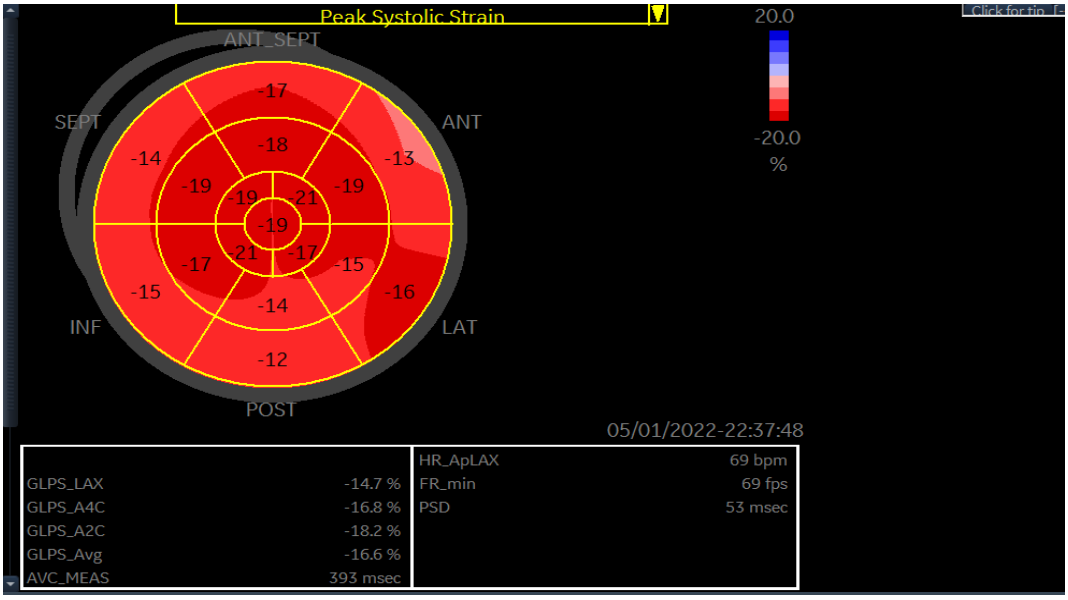

Figure (2): GLS among hypertensive group.

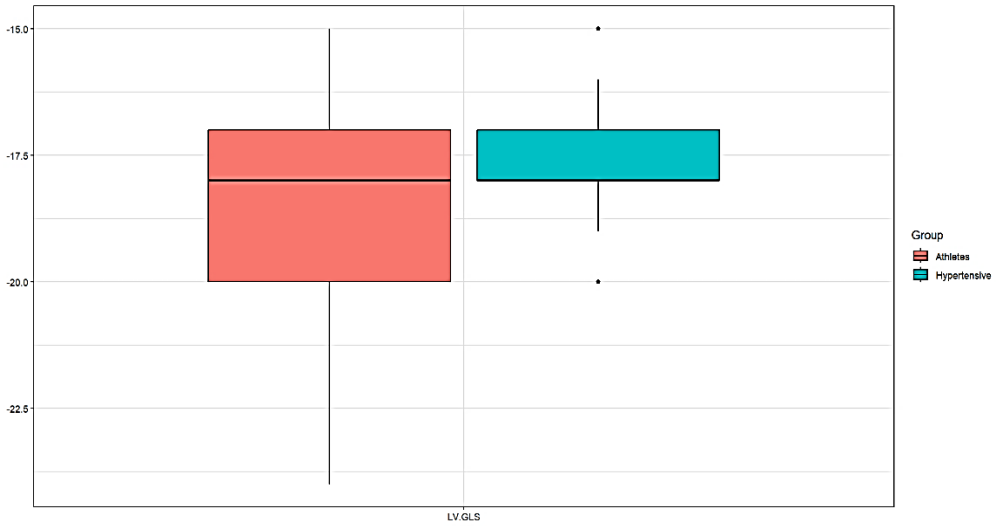

Figure (3): Boxplot showing the difference between Athletes and hypertensive groups regarding LV GLS.

DISCUSSION

LVH may develop as a normal physiological response to intense physical training, commonly seen in athletes, or as a pathological consequence of genetic conditions or sustained pressure overload. Conventional echocardiography remains a cornerstone in the evaluation of LVH, playing a crucial role in distinguishing between adaptive and maladaptive cardiac remodeling [11]. Technological advancements in echocardiographic imaging have significantly enhanced our capacity to assess structural and functional cardiac changes in athletes. These modifications are considered compensatory mechanisms to meet the increased circulatory demands imposed by rigorous training. Nonetheless, accurate differentiation between physiological hypertrophy and pathology remains essential [12]. The current study aimed to distinguish LVH in athletes from that in hypertensive patients using a combination of clinical evaluation, standard echocardiography, and speckle-tracking strain imaging.

Clinically, notable differences were observed between the two groups. Athletes exhibited significantly lower resting heart rates ($p = 0.001$), a finding attributed to enhanced parasympathetic tone and cardiovascular efficiency associated with training. Additionally, systolic and diastolic blood pressure values were significantly lower in the athletic cohort compared to hypertensive subjects ($p < 0.001$), further supporting the role of autonomic modulation in trained individuals [13].

According to standard echocardiographic measurements, the athlete group demonstrated a significantly larger LVEDD compared to the hypertensive group ($p < 0.001$). Although, the hypertensive group showed a slightly increased left atrial diameter, the difference was not statistically significant. Interventricular septal (IVS) thickness and relative wall thickness (RWT) were notably greater in hypertensive individuals, while total left ventricular mass was higher in athletes. These findings are consistent with those reported by **Cappelli *et al***. [14] who observed increased LVEDD and LV mass among 50 endurance-trained athletes compared to 22 hypertensive patients with LVH, with no significant difference in ejection fraction between groups. Similar results were echoed by **Dores *et al***. [15], who noted comparable adaptations across different levels of athletic training.

Physiological hypertrophy in athletes is typically characterized by an increase in myocardial mass while preserving normal myocardial architecture, reflecting the heart's adaptation to sustained volume and pressure loads during intense physical activity. This remodeling is considered a benign and functional response tailored to the demands of exercise [14]. The extent of this adaptation may vary based on several factors including the type of sport, intensity and duration of training, as well as body composition. In contrast, the pathological remodeling observed in hypertension—driven by chronic pressure overload—is often associated with increased myocardial wall thickness and concentric LVH, particularly in individuals with poorly controlled blood pressure, whether in clinical or ambulatory settings [2, 3].

With respect to diastolic function, the athletic group exhibited normal LA diameters, although these were slightly smaller than those observed in hypertensive patients. Additionally, athletes demonstrated significantly lower A-wave velocities and E/e′ ratios, along with higher e′ velocities, indicating more favorable diastolic performance. In contrast, the hypertensive group showed impaired diastolic indices, consistent with elevated filling pressures. These findings align with those reported by **Saghiri *et al***. [16] who examined 108 participants—including hypertensive individuals, strength-trained athletes with LVH, and healthy controls—and found that hypertensive patients had significantly higher E/e′ ratios compared to athletes. Similarly, **Dores *et al***. [15] noted lower A-wave peak velocities and reduced E/e′ ratios in highly trained athletes, underscoring the enhanced diastolic function commonly seen in this population. Evaluating diastolic function is a critical component in distinguishing physiological from pathological LVH, especially as diastolic dysfunction may precede structural changes in hypertensive heart disease. In many cases, it represented the earliest manifestation of hypertension-related cardiac involvement, even in the absence of overt hypertrophy. Conversely, athletes often develop superior diastolic function as an adaptive mechanism to preserve stroke volume at elevated heart rates [12]. The reduction in A-wave velocity observed in athletes is reflective of a shift in ventricular filling toward early diastole, a hallmark of efficient diastolic compliance [3, 12].

GLS has emerged as a valuable echocardiographic tool for differentiating patterns of LVH, particularly in distinguishing physiological adaptation from pathological remodeling. In this study, GLS measurements revealed a statistically significant difference between the athlete and hypertensive groups ($p < 0.001$), with the hypertensive cohort demonstrating notably reduced strain values. This reduction reflects subclinical myocardial dysfunction not always evident through conventional echocardiographic parameters. Similar results were reported by **Lo Iudice *et al***. [17], who evaluated GLS in 36 endurance athletes versus sedentary matched controls using standard and real-time 3D echocardiography; athletes displayed significantly higher GLS values. Consistent with our findings, **Cappelli *et al***. [14] also noted lower GLS values among hypertensive patients compared to trained athletes, reinforcing the utility of strain imaging in this context. Furthermore, **Lazzeroni *et al***. [18] proposed that GLS is sensitive to variations in afterload, with higher strain values under increased afterload suggesting preserved contractile function. Conversely, lower GLS readings may reflect impaired myocardial mechanics, potentially signaling an increased risk for adverse outcomes such

as symptom development, irreversible myocardial injury, and myocardial fibrosis. These insights underscore the clinical relevance of GLS in early detection of myocardial dysfunction, especially when structural indices alone may not fully differentiate between physiological and pathological LVH.

LIMITATIONS

This study was limited by its cross-sectional design, which precludes assessment of longitudinal changes in cardiac remodeling over time. The sample included only male participants, which may limit the generalizability of findings to female athletes or hypertensive patients. Additionally, blood pressure measurements were taken in a clinical setting, potentially subject to white coat effect, and ambulatory BP monitoring was not performed. Lastly, although speckle-tracking echocardiography was utilized, cardiac MRI—the gold standard for tissue characterization—was not employed for further differentiation of LVH etiology.

CONCLUSION

While endurance and strength training can lead to increases in LVM, wall thickness, and chamber dimensions, these adaptations may resemble the pathological remodelling seen in hypertension. However, certain clinical parameters can help differentiate between athlete's heart and hypertensive left ventricular hypertrophy. The presence of high blood pressure, an increase in LV mass, elevated or upper-normal LV dimensions, along with impaired diastolic function and reduced GLS, supported a diagnosis of hypertension and are findings that were not typically observed in athletes.

Financial support and sponsorship: Nil.
Conflict of Interest: Nil.

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