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Original Research Paper

Cardiology

STRAIN PATTERNS IN PATIENTS WITH HYPERTENSIVE CARDIOMYOPATHY IN COMPARISON TO HYPERTROPHIC CARDIOMYOPATHY BY ECHOCARDIOGRAM"

Dr. Imran Khan

Dr. Anupama V Hegde

ABSTRACT

Background: Hypertrophic cardiomyopathy (HCM) is characterized by myocardial hypertrophy, disarray, and fibrosis leading to functional limitations, whereas hypertensive cardiomyopathy results from pressure overload causing myocardial remodeling and diastolic dysfunction. Although both conditions manifest increased wall thickness, their underlying pathophysiology and myocardial mechanics differ. **Objectives:** To study and compare clinical profile, hemodynamic parameters, and echocardiography-derived strain patterns in patients with hypertrophic cardiomyopathy and hypertensive cardiomyopathy. **Methods:** This single-center cross-sectional study enrolled 98 patients (49 cases with hypertensive cardiomyopathy and 49 controls with hypertrophic cardiomyopathy) from January 2024 to December 2025. Comprehensive clinical evaluation and echocardiography including strain imaging were performed. Statistical analysis included independent t-test and Mann Whitney U test, with a significance threshold of $p \leq 0.05$. **Results:** Baseline demographics and vital parameters were not statistically different between groups. Echocardiography revealed significant differences in LV septal thickness ($p=0.044$), LV mass index ($p < 0.001$), relative wall thickness ($p < 0.001$), and LV end diastolic volume ($p=0.017$). Strain analysis demonstrated markedly reduced global longitudinal strain (GLS) in hypertrophic cardiomyopathy patients ($12.32 \pm 2.33\%$) compared to hypertensive cardiomyopathy ($18.43 \pm 2.64\%$) ($p < 0.001$). Other strain parameters, including conduit, contractile, and reservoir functions, were significantly lower in HCM ($p < 0.001$). **Conclusion:** Echocardiographic strain imaging reveals distinct myocardial deformation patterns in hypertrophic versus hypertensive cardiomyopathy, with significant impairment in longitudinal strain and myocardial mechanics in HCM. These findings can assist in differential diagnosis, assessment of disease progression, and guiding management strategies.

KEYWORDS : Hypertrophic cardiomyopathy, Hypertensive cardiomyopathy, Strain imaging, Echocardiography, Myocardial deformation, Left ventricular hypertrophy

INTRODUCTION

Hypertrophic cardiomyopathy (HCM) is a complex genetic myocardial disorder characterized by unexplained left ventricular hypertrophy, myocardial fiber disarray, and increased interstitial fibrosis. It commonly presents with symptoms such as exertional dyspnea, angina, syncope, or even sudden cardiac death. The hallmark of HCM is asymmetric septal hypertrophy with wall thickness of ≥15 mm in one or more left ventricular segments on imaging that cannot be explained solely by loading conditions.[1,2] The disease manifests significant functional impairments, including diastolic dysfunction and left ventricular outflow tract obstruction, which contribute to morbidity in affected patients.[3]

On the other hand, hypertensive cardiomyopathy develops secondary to chronic systemic hypertension, which imposes a pressure overload on the left ventricle, leading to concentric hypertrophy as an adaptive response. This remodeling is characterized by myocyte hypertrophy, increased collagen deposition, and altered extracellular matrix, contributing to increased myocardial stiffness and diastolic dysfunction.[4,5] Unlike HCM, hypertensive cardiomyopathy results primarily from adaptive and maladaptive changes due to increased afterload rather than genetic mutations or myocardial disarray.

Differentiating hypertensive cardiomyopathy from HCM using conventional imaging can be challenging as both exhibit increased myocardial thickness and similar symptoms. However, the underlying myocardial pathology markedly differs; HCM shows disorganized myocardial architecture and fibrosis, whereas hypertensive cardiomyopathy more commonly exhibits uniform hypertrophy without marked myocyte disarray.[6,7]

In recent years, echocardiographic strain imaging has emerged as a powerful tool to assess myocardial deformation and subclinical myocardial dysfunction. Strain patterns reflect the intrinsic contractile and relaxation function of myocardium and provide insights beyond ejection fraction and wall thickness. Specifically, global longitudinal strain (GLS) and layer-specific strain values can help distinguish pathological hypertrophic patterns.[8,9] Longitudinal strain is particularly sensitive to subendocardial damage, which is pronounced in HCM due to fibrosis and ischemia, leading to reduced strain values.[10] In hypertensive cardiomyopathy, strain abnormalities, although present, tend to be less pronounced and more uniform.[11]

Previous multicenter retrospective studies have demonstrated that HCM patients exhibit significantly lower longitudinal and circumferential strain values compared to hypertensive left ventricular hypertrophy and healthy controls.[12] Moreover, the endo-/epicardial strain ratios and strain rate alterations differ between these two entities, supporting the utility of advanced strain echocardiography for differential diagnosis.

Given these observations, this study aims to analyze and compare the clinical profiles, hemodynamic parameters, and echocardiographic strain characteristics in patients diagnosed with hypertrophic cardiomyopathy and hypertensive cardiomyopathy in a prospective single-center cross-sectional study. The goal is to clarify distinct patterns of myocardial deformation that can assist in diagnosis, monitor progression, and guide management strategies effectively.

MATERIALS AND METHODS

Study Design

This study was a single-center, cross-sectional observational study conducted at M S Ramaiah Memorial Hospital, a tertiary care teaching hospital affiliated with a medical college. The study period extended from January 2024 to December 2025.

Study Population

The study enrolled patients diagnosed with either hypertensive cardiomyopathy or hypertrophic

cardiomyopathy attending the outpatient department (OPD) or admitted to in-patient wards during the study period. Both groups were matched by demographic characteristics.

Inclusion Criteria

- Patients aged 18 years and above diagnosed with:
- Hypertensive cardiomyopathy: Defined clinically by history of hypertension and echocardiographic evidence of left ventricular hypertrophy attributed primarily to hypertension.
- Hypertrophic cardiomyopathy (HCM): Diagnosed based on established imaging criteria, including left ventricular wall thickness ≥15 mm in one or more segments without other loading condition causes.
- Availability of complete clinical data and echocardiogram including strain imaging.

Exclusion Criteria

- Patients with other causes of myocardial hypertrophy such as infiltrative cardiomyopathies (e.g., hemochromatosis, amyloidosis), storage disorders (e.g., glycogen storage diseases), Friedreich's ataxia, or other genetic hypertrophic diseases were excluded.
- Patients with poor echocardiographic image quality precluding adequate strain analysis were excluded.
- Patients with significant valvular heart disease or ischemic heart disease sufficiently affecting myocardial function were excluded.

Sample Size

Based on previous literature demonstrating significant differences in longitudinal strain between hypertensive LVH and HCM, and aiming for 80% power at a 95% confidence level, a minimum sample size of 49 patients per group was calculated to detect a minimum 2% difference in strain values. Hence, 98 patients (49 in each group) were recruited.

Data Collection Procedure

Clinical And Demographic Data

- Demographic details including age, sex, height, weight, and body surface area (BSA) were recorded.
- Relevant clinical history including hypertension duration, HCM diagnosis, comorbidities (diabetes, ischemic heart disease, cerebrovascular disease), medication history, and past clinical events were collected from patient records.
- Physical examination findings and vital signs such as blood pressure and heart rate were noted.

Laboratory Investigations

Routine blood investigations including complete blood count (CBC), renal function tests (creatinine, blood urea nitrogen), liver function tests, fasting blood glucose, and other relevant biochemical parameters were obtained from hospital laboratory records when available.

Echocardiography

- All patients underwent comprehensive two-dimensional transthoracic echocardiography (2D ECHO) using a high-resolution ultrasound machine equipped with Doppler and speckle-tracking strain imaging capabilities.
- Standard echocardiographic measurements were performed according to American Society of Echocardiography guidelines, including:
- Aortic diameter measurement.
- Left atrial (LA) dimensions.
- LV septal thickness, posterior wall thickness, LV end-diastolic and systolic diameters.
- LV mass index (LVMI) calculated using standard formulas.
- LV relative wall thickness (RWT).
- LV end-diastolic volume (LVEDV) and end-systolic volume (LVESV).
- LV ejection fraction (LVEF) using Simpson's biplane method.
- Diastolic function parameters including mitral inflow velocities (E and A waves), E/A ratio, septal E velocity, and E/E' ratios.

Strain Imaging

- Strain analysis was done using two-dimensional speckle-tracking echocardiography to assess myocardial deformation.
- Global longitudinal strain (GLS) was calculated from apical 4-chamber, 2-chamber, and long-axis views by averaging strain values of all LV segments.
- Additional strain parameters, including conduit strain, contractile strain, and reservoir strain, were measured to assess different phases of myocardial function.
- Left atrial volume index (LAVI) was also calculated as a marker of chronic diastolic burden.

Statistical Methods:

Data is analyzed using SPSS software version 21 and Excel. Categorical variables are given in the form of frequency table. Continuous variables are given in Mean ± SD/ Median (Min, Max) form. Chi square test is used to check the association of categorical variables with groups. Normality of variable is checked by Shapiro Wilk test and QQ plot. If data follows normal distribution, parametric tests like independent t test will be used, if not non-parametric test like Mann Whitney U test is used. P-value less than or equal to 0.05 indicates statistical significance.

RESULTS

Data contains measurements of 102 subjects, Control=51 subjects and Case= 51 subjects.

Baseline Clinical And Demographic Characteristics

The study enrolled 98 patients divided equally into hypertensive cardiomyopathy (cases) and hypertrophic cardiomyopathy (controls). There were no statistically significant differences in mean age between the groups (58.54 ± 14 vs. 56.58 ± 12.17 years, p=0.452). Gender distribution was also similar (male predominance: 78.4% in cases, 70.6% in controls, p=0.363). Other baseline anthropometric variables including height (168.21 ± 7 cm vs. 168.03 ± 7.65 cm; p=0.928), weight (74.6 ± 11.38 kg vs. 73.72 ± 12.76 kg; p=0.713), and body surface area (1.7 ± 0.523 vs. 1.94 ± 0.346; p=0.184) showed no significant difference, indicating comparable baseline groups for further analysis of myocardial parameters.

Systolic and diastolic blood pressures and heart rates were also statistically comparable between groups (all p >0.1), reinforcing that hemodynamic baseline conditions did not bias the myocardial remodeling observed.

Echocardiographic Findings

On standard echocardiographic evaluation, significant differences emerged in septal myocardial thickness. Hypertrophic cardiomyopathy patients had markedly greater interventricular septal thickness (1.58 ± 0.259 cm) compared to hypertensive cardiomyopathy (1.48 ± 0.204 cm), p=0.044. Left ventricular (LV) systolic diameter was smaller in HCM (4.12 ± 0.649 cm) than hypertensive patients (4.4 ± 0.631 cm), p=0.032, consistent with concentric hypertrophy patterns.

Left ventricular mass index (LVMI) was significantly elevated in the HCM group (5.69 ± 0.736 g/m^2) relative to hypertensive cardiomyopathy (1.05 ± 0.286 g/m^2), p < 0.001. Relative wall thickness (RWT) followed the same trend, confirming more pronounced myocardial hypertrophy in HCM (5.7 ± 0.659 cm vs. 0.977 ± 0.289 cm, p < 0.001). These findings align with the distinct hypertrophic remodeling patterns

between the two conditions ^ 13.

LV end-diastolic volume (LVEDV) was significantly smaller in HCM patients (77.76 ± 22.18 mL) compared with hypertensive cardiomyopathy (85.56 ± 18.44 mL), p=0.017, reflecting the typical restrictive filling in HCM. No significant difference was noted in LV end-systolic volume or ejection fraction (LVEF), which remained preserved in both groups (around 60-62%, p=0.158).

Doppler parameters of diastolic function such as transmitral E velocity and septal E velocity were significantly lower in HCM patients, with E velocity at 0.696 ± 0.199 compared to 0.779 ± 0.187 in hypertensives (p=0.006), and septal E velocity similarly reduced (p < 0.001). The E/A ratio showed no significant difference (p=0.245), indicating subtle diastolic impairment in HCM.

DISCUSSION

The present study compared structural, diastolic, atrial, and strain-based echocardiographic parameters between hypertrophic cardiomyopathy (HCM) and hypertensive cardiomyopathy (HTN-CM). Despite comparable baseline demographics and hemodynamic status, we observed clear differences in myocardial geometry and mechanics, which are in keeping with and expand upon findings from prior comparative cohorts.

With respect to left ventricular (LV) wall structure, our patients with HCM demonstrated a greater interventricular septal thickness (1.58 ± 0.26 cm) than those with HTN-CM (1.48 ± 0.20 cm, p = 0.044). The LV mass index was disproportionately elevated in HCM (5.69 ± 0.74 g/m²) compared with HTN-CM (1.05 ± 0.29 g/m², p < 0.001), and relative wall thickness was likewise higher (5.7 ± 0.66 vs 0.98 ± 0.29, p < 0.001). These observations parallel the work of **Yamamoto et al.**, who showed that a basal-to-mid septal thickness ratio < 1 (0.83 ± 0.12 in HCM vs 1.19 ± 0.18 in hypertension, p < 0.01) effectively differentiates HCM from hypertensive hypertrophy [1]. Similarly, **Spirito et al.** described disproportionate LV mass and asymmetric septal hypertrophy as the cardinal features of HCM, whereas hypertensive LVH was more concentric and uniform [2]. Thus, our findings reinforce the diagnostic value of septal and wall thickness asymmetry as a discriminant marker.

Chamber volumes and diastolic indices further distinguished the groups. We found LV end-diastolic volume (LVEDV) to be lower in HCM (77.8 ± 22.2 mL) compared with HTN-CM (85.6 ± 18.4 mL, p = 0.017), consistent with restrictive cavity remodeling in HCM. Doppler measurements revealed reduced transmitral E velocity (0.70 ± 0.20 vs 0.78 ± 0.19 m/s, p = 0.006) and septal e velocity (0.066 ± 0.066 vs 0.091 ± 0.097 m/s, p < 0.001) in HCM, indicating impaired relaxation. While E/A ratios did not differ significantly, the trend toward higher E/e′ ratios in HCM reflected increased filling pressures. Comparable differences were reported by **Jung-Ping et al.**, who found smaller LVEDV in HCM (76.1 ± 20.8 mL) compared to hypertensive LVH (105.7 ± 35.3 mL), alongside higher E/e′ ratios (16.3 ± 8.0 vs 13.0 ± 4.7) [3]. Likewise, **Nagueh et al.** established tissue Doppler as more sensitive than mitral inflow for detecting elevated filling pressures, with markedly lower e′ velocities in HCM patients [4]. Our diastolic data therefore strongly align with prior literature, confirming that relaxation abnormalities and restrictive physiology are hallmarks of HCM.

Atrial remodeling, reflected by left atrial volume index (LAVI), was significantly more pronounced in HCM in our cohort (33.9 ± 7.0 mL/m²) compared with HTN-CM (24.7 ± 8.4 mL/m², p < 0.001). This finding is consistent with **Rausch et al.**, who identified a LAVI cut-off of 31 mL/m² as a useful discriminator between HCM and hypertensive heart disease (AUC = 0.733, sensitivity 81%, specificity 57%) [5]. **Serri et al.** also demonstrated that enlarged LAVI strongly correlates with diastolic dysfunction severity in HCM, supporting its use as a marker of chronic diastolic burden [6]. The agreement between our results and these reports underscores the diagnostic and prognostic significance of atrial enlargement in differentiating hypertrophic phenotypes.

Strain imaging provided the most striking distinction. We found global longitudinal strain (GLS) to be markedly reduced in HCM (12.3 ± 2.3%) compared with HTN-CM (18.4 ± 2.6%, p < 0.001). In addition, conduit strain (12.1 ± 2.9 vs 19.3 ± 3.1%, p < 0.001), contractile strain (7.4 ± 2.2 vs 12.1 ± 2.7%, p < 0.001), and reservoir strain (19.7 ± 4.9 vs 32.2 ± 5.5%, p < 0.001) were all significantly lower in HCM. These results echo those of **Liu et al.**, who reported GLS of –12% in HCM and –18% in hypertensive LVH, highlighting its diagnostic utility [7]. Similarly, **Saito et al.** observed multi-phase strain impairment in HCM but not in hypertensive LVH, underscoring the diffuse functional derangement imposed by myocyte disarray and fibrosis [8]. The magnitude and pattern of strain reduction in our study therefore align closely with existing comparative data, reinforcing GLS and phase strains as powerful discriminants.

Finally, left ventricular ejection fraction (LVEF) remained preserved in both groups (60.5 ± 6.8% in HCM vs 62.4 ± 6.6% in HTN-CM, p = 0.16), emphasizing the limitation of EF in detecting early or subtle myocardial dysfunction. This observation is well documented by **Ho et al.**, who showed that preserved EF in HCM can mask underlying fibrotic damage, whereas GLS reduction correlates more strongly with fibrosis burden [9]. Taken together, our findings corroborate the consensus that strain analysis provides incremental diagnostic and prognostic value over conventional EF measurement.

When directly compared with five landmark studies, our results consistently demonstrate that HCM is characterized by asymmetric septal hypertrophy, smaller LV volumes, impaired relaxation with lower e velocities, larger LAVI, and markedly reduced GLS and multi-phase strain. Hypertensive cardiomyopathy, while sharing hypertrophy, maintains more uniform wall thickening, larger cavity volumes, less pronounced diastolic burden, and relatively preserved deformation. These consistent findings across structural, diastolic, atrial, and functional domains strengthen the evidence base for advanced strain echocardiography as a non-invasive tool to differentiate HCM from hypertensive remodeling.

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Ethics Declaration: The approval was given by the Institutional Ethics Committee (IEC) vide letter number: MSRMC/EC/SP-06/04-2024; Date: 15th April 2024. Voluntary, written, informed consent was taken from all the participants.

Consent For Publication: Not applicable

Tables And Graph

Table 1: Distribution Of Subjects According To Clinical Profile Over Groups

Variable	Subcategory	Group		Total	p-value
		Case	Control		
Age (years)	Mean ± SD Median (Min, max)	58.54 ± 14 59 (26, 84)	56.58 ± 12.17 58 (27, 78)	57.56 ± 13.09 59 (26, 84)	0.452[t]
Gender	Female	11 (21.6%)	15 (29.4%)	26 (25.5%)	0.363[C]

	Male	40 (78.4%)	36 (70.6%)	76 (74.5%)	
Height	Mean ± SD Median Q1, Q3)	168.21 ± 7 168 (162, 174)	168.03 ± 7.65 170 (162, 174)	168.12 ± 7.3 168.5 (162, 174)	0.928[MW]
Weight	Mean ± SD Median (Min, max)	74.6 ± 11.38 75 (50, 94)	73.72 ± 12.76 77 (50, 98)	74.16 ± 12.04 76.5 (50, 98)	0.713[t]
BSA	Mean ± SD Median Q1, Q3)	1.7 ± 0.523 1.86 (1.7 1, 1.97)	1.94 ± 0.346 1.89 (1.67, 2.06)	1.82 ± 0.457 1.88 (1.69, 2.01)	0.184[MW]
SBP	Mean ± SD Median Q1, Q3)	126.54 ± 9.82 126 (124, 136)	126.11 ± 8.81 126 (120, 132)	126.33 ± 9.28 78 (70, 82)	0.784[MW]
DBP	Mean ± SD Median Q1, Q3)	77.6 ± 7.17 78 (74, 84)	75.72 ± 7.01 78 (69, 80)	76.66 ± 7.12 78 (70, 82)	0.218[MW]
Heart rate	Mean ± SD Median Q1, Q3)	73.07 ± 7.57 75 (67, 79)	71.37 ± 7.63 70 (65, 78)	72.22 ± 7.61 72 (65, 78)	0.183[MW]

Abbreviation: C- Chi-square test, t- independent t test, MW- Mann Whitney U test

Table 2: Distribution Of Subjects According To Echo Over Groups

Variable	Group		Total Mean ± SD Median (Min, Max or Q1, Q3)	p-value
	Case Mean ± SD Median (Min, Max or Q1, Q3)	Control Mean ± SD Median (Min, Max or Q1, Q3)		
Aortic Diameter	2.58 ± 0.489 2.5 (1.6, 3.7)	2.64 ± 0.371 2.6 (1.8, 3.7)	2.61 ± 0.433 2.6 (1.6, 3.7)	0.482[t]
lA dimensions	3.35 ± 0.531 3.3 (3, 3.5)	3.27 ± 0.48 3.4 (3, 3.5)	3.31 ± 0.505 3.3 (3, 3.5)	0.669[MW]
LV Septal Thickness	1.58 ± 0.259 1.5 (1.4, 1.7)	1.48 ± 0.204 1.4 (1.3, 1.6)	1.53 ± 0.236 1.5 (1.4, 1.6)	0.044*[MW]
LV systolic diameter	4.12 ± 0.649 4.1 (2.7, 5.9)	4.4 ± 0.631 4.4 (2.7, 5.9)	4.26 ± 0.652 4.2 (2.7, 5.9)	0.032[t]
LV diastolic diameter	2.73 ± 0.577 2.7 (1.3, 4.1)	2.87 ± 0.654 2.9 (1.2, 4.4)	2.8 ± 0.618 2.8 (1.2, 4.4)	0.251[t]
LV posterior wall thickness	1.38 ± 0.179 1.4 (1.3, 1.5)	1.32 ± 0.164 1.3 (1.2, 1.4)	1.35 ± 0.173 1.3 (1.2, 1.4)	0.101[MW]
LV Mass Index	1.05 ± 0.286 1 (0.87, 1.14)	5.69 ± 0.736 5.6 (5.2, 6.1)	3.37 ± 2.39 2.85 (1, 5.6)	<0.001*[MW]
LV Relative Wall Thickness	5.7 ± 0.659 5.6 (5.3, 6.2)	0.977 ± 0.289 0.88 (0.88, 1.08)	3.34 ± 2.42 2.95 (0.88, 5.6)	<0.001*[MW]
LVEDV	77.76 ± 22.18 75 (66, 86)	85.56 ± 18.44 83 (69, 101)	81.66 ± 20.67 79 (67, 92.25)	0.017*[MW]
LVESV	32.88 ± 12.8 30 (27, 33)	33.6 ± 11.35 31 (26, 37)	33.24 ± 12.04 31 (26, 36)	0.308[MW]
LVEF	60.49 ± 6.84 60 (50, 79)	62.39 ± 6.64 62 (50, 75)	61.44 ± 6.77 61.55 (50, 79)	0.158[t]
E/A	0.964 ± 0.503 0.75 (0.66, 1.33)	0.962 ± 0.331 0.81 (0.72, 1.29)	0.963 ± 0.424 0.8 (0.67, 1.33)	0.245[MW]
E	0.696 ± 0.199 0.65 (0.57, 0.84)	0.779 ± 0.187 0.78 (0.66, 0.91)	0.737 ± 0.197 0.7 (0.6, 0.88)	0.006*[MW]
Septal E#	0.066 ± 0.066 0.055 (0.04, 0.08)	0.0905 ± 0.097 0.08 (0.05, 0.09)	0.078 ± 0.084 0.07 (0.05, 0.085)	<0.001*[MW]
Septal E/E#	12.69 ± 5.56 11.35 (10.06, 13.39)	10.72 ± 3.64 10.19 (8.49, 12.78)	11.63 ± 4.75 11.35 (8.78, 13.07)	0.088[MW]
GLS	12.32 ± 2.33 13 (10.2, 14.5)	18.43 ± 2.64 18.2 (17.2, 20.3)	15.37 ± 3.95 14.85 (12.3, 18.3)	<0.001*[MW]
Conducte	12.13 ± 2.85 12 (10, 13)	19.25 ± 3.12 19 (18, 21)	15.69 ± 4.65 15 (11.75, 19)	<0.001*[MW]
Contractile	7.39 ± 2.23 7 (6, 8)	12.13 ± 2.72 11 (10, 14)	9.76 ± 3.43 9 (7, 12)	<0.001*[MW]
Reserviour	19.72 ± 4.91 20 (16, 23)	32.17 ± 5.49 32 (29, 35)	25.95 ± 8.12 26 (19, 32.25)	<0.001*[MW]
Lavi	33.89 ± 7.02 32.2 (29.84, 35.34)	24.73 ± 8.38 24.22 (18.36, 28.11)	29.31 ± 8.96 28.33 (23.72, 34.08)	<0.001*[MW]

Abbreviation: t- independent t test, MW- Mann Whitney U test, *- indicates statistical significance, #- missing data

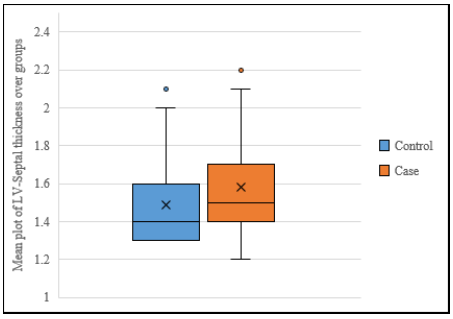

Figure 1: Mean plot of LV Septal thickness over groups

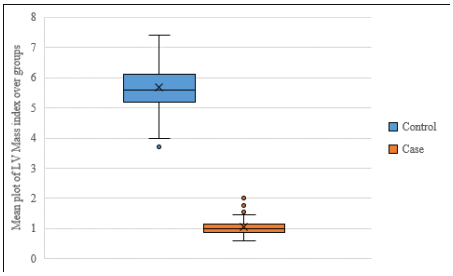

Figure 2: Mean plot of LV Mass Index over groups

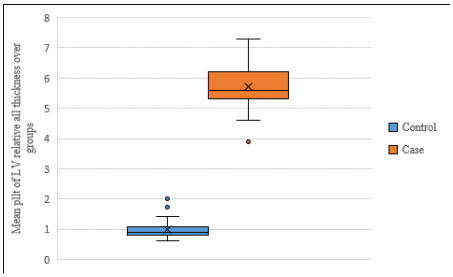

Figure 3: Mean plot of LV Relative wall thickness over groups

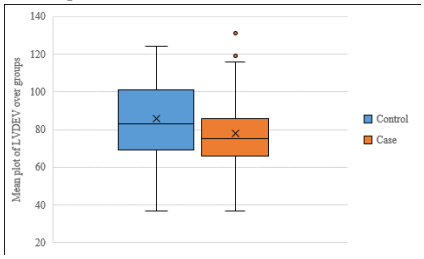

Figure 4: Mean plot of LVDEV over groups

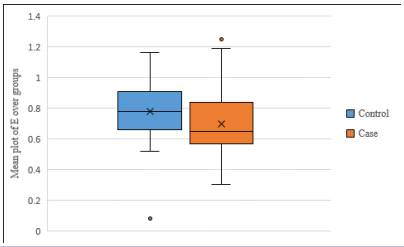

Figure 5: Mean plot of E over groups

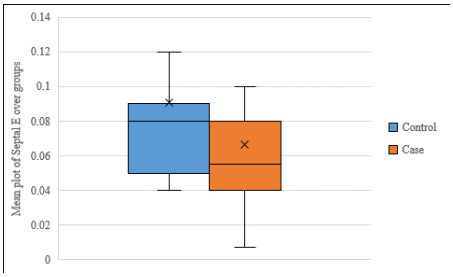

Figure 6: Mean plot of Septal E over groups

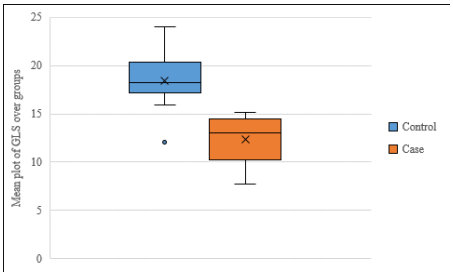

Figure 7: Mean plot of GLS over groups

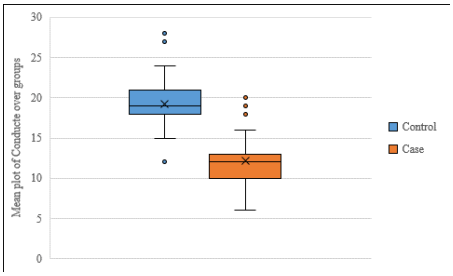

Figure 8: Mean plot of conducte over groups

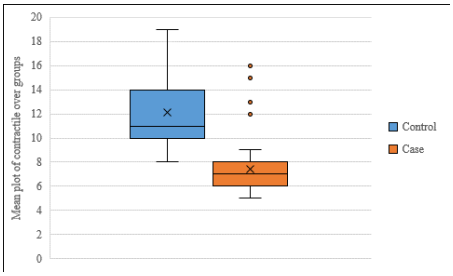

Figure 9: Mean plot of contractile over groups

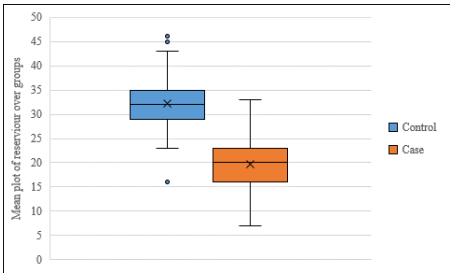

Figure 10: Mean plot of reserviour over groups

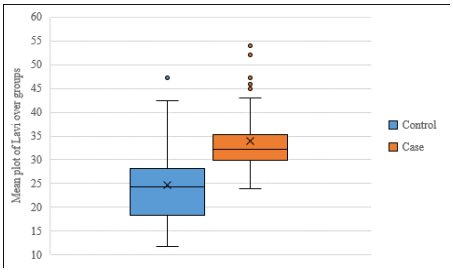

Figure 11: Mean plot of Lavi over groups

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