Left atrial mechanics in hypertrophic cardiomyopathy: discriminating hypertrophy and predicting fibrosis

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Abstract

BACKGROUND : Hypertrophic cardiomyopathy (HCM) and left ventricular hypertrophy (LVH) secondary to systemic hypertension (HTN) may be associated with left atrial (LA) functional abnormalities. We aimed to characterize LA mechanics in HCM and HTN and determine any correlation with the extent of fibrosis measured by cardiac magnetic resonance (CMR) in HCM patients.

METHODS: Two-dimensional speckle tracking-derived longitudinal LA function was acquired from apical views in 60 HCM patients, 60 HTN patients, and 34 age-matched controls. HCM patients also underwent CMR, with measurement of late gadolinium enhancement (LGE) extension. Association with LA strain parameters was analyzed.

RESULTS: The mean LV ejection fraction did not differ across groups. The E/e' ratio was only preserved in the control group and significantly impaired in the HCM group. LA mechanics were significantly reduced in HCM, compared to the HTN group. LA strain rate in systole (LA-SRs) and late diastole (LA-SRa) were the best discriminators of HCM, with an area under the curve (AUC) of 0.8, followed by LA strain in systole (LA sys) (AUC 0.76). LA-SRs and LA-SRa had high specificity (89% and 91%, respectively) and LA sys had sensitivity of 80%. LA strain rate in early diastole (LA-SRe) was moderately correlated with the extension of LGE (r 2 0.42, p=0.027)

CONCLUSIONS: LA-SRs and LA-SRa were the best discriminators for LVH secondary to HCM. LA-SRe was best correlated with the degree of fibrosis assessed by CMR. These findings suggest that LA mechanics can be potential predictors of disease severity in HCM.

Background

Left ventricular hypertrophy (LVH) present in hypertrophic cardiomyopathy (HCM) and arterial hypertension (HTN) is often related to myocardial dysfunction and increased risk of sudden death[1,2]. In HTN, LVH occurs as a response to pressure overload. In HCM, a complex remodeling process is initiated by the responses of the cardiomyocytic and noncardiomyocytic components to dynamic mechanical and neurohumoral stimuli[1–3]. HCM is a myocardial autosomal-dominant disorder, associated with mutations in sarcomeric genes, affecting both ventricular and atrial myocardia[1,2].

Cardiovascular magnetic resonance (CMR) allows a thoroughly description of HCM characteristics, namely LV hypertrophy and fibrosis with late gadolinium enhancement (LGE)[4]. Quantitative LV LGE characterizes HCM stages, LV remodeling and systolic dysfunction and is an important predictor of sudden death[4,5]. Systolic dysfunction commonly appears in end-stage HCM, though a significant portion of patients have some extent of diastolic dysfunction[2,6].

Increased LV mass and diastolic dysfunction are associated with progressive left atrial (LA) dilatation and dysfunction. Accordingly, LA remodeling is a common feature in both HCM and HTN[2,7]. Furthermore,

LA size and volume have been shown to be determinants of both exercise capacity[8] and major adverse cardiac and cerebrovascular events in HCM patients[9].

Since the LA modulates LV performance by its reservoir function during ventricular systole, conduit function during early ventricular diastole, and booster pump function during late ventricular diastole, LA myopathy could predispose outcomes independent of LV function[7, 10]. LA function is correlated with heart failure symptoms in HCM and is a strong predictor for the development of atrial fibrillation (AF)[11,12].

The assessment of LA mechanics using two-dimensional (2D)-speckle tracking echocardiography (STE) has been shown as a feasible and reproducible marker of LA function[13,14].

Although LVH apparently is the major factor of dysfunction in LV mechanics[2], the degree of LA dysfunction in different states of LVH (particularly, LVH secondary to HTN and to HCM) is not fully understood. LVH and fibrosis, both representing substrates of LV diastolic dysfunction, might be associated with LA dysfunction in HCM. Therefore, this study aimed (1) to characterize LA mechanics in HCM and in HTN patients with significant LVH and (2) to correlate LA function with LV fibrosis assessed by CMR in HCM patients.

Methods

Study population

This prospective observational study included 60 patients diagnosed with HCM (diagnosis confirmed by CMR) and 60 HTN patients recruited from our outpatient department. We excluded patients with poor acoustic window, AF, moderate or severe valvular disease, ischemic heart disease, or pulmonary hypertension defined as pulmonary artery systolic pressure (PASP) >45 mmHg. As controls, we included 34 healthy individuals without HTN, AF, or valvular disease, and age-matched to HCM and HTN patients.

Study procedures

We analyzed the epidemiologic, clinical, and echocardiographic data of participants divided into the HCM group, HTN group, and control group. Data from CMR of HCM patients at the time of diagnosis were also assessed. Echocardiographic images were collected 43±18 days after HCM diagnosis was made by CMR. The study was approved by the scientific and bioethical committees of Centro Hospitalar e Universitário de Coimbra (Coimbra, Portugal) and was performed in accordance with the Declaration of Helsinki.

Echocardiographic data

A complete echocardiographic investigation was performed in all participants, including LV- and LA-STE with global longitudinal strain (GLS) analysis. We used a Vivid 7 (GE Healthcare, Horten, Norway)

cardiovascular ultrasound device, with a 1.7/3.4-MHz tissue harmonic transducer. Standard echocardiographic views were obtained with frame-rate optimization (60–80 fps in 2D imaging). We analyzed offline the echocardiographic data using a specific software (EchoPAC 16.0; GE Healthcare).

LV dimensions and function

LV size and systolic function assessment, including measurement of LV ejection fraction (LVEF), LV end-diastolic diameter (LVDD), and LV end-systolic diameter (LVSD), followed the current recommendations[15]. STE-derived LV-GLS was obtained using a 16-segment model of the LV[16]. Diastolic function including mitral E velocity, mitral A velocity, and mean E/e' ratio, was also evaluated.

LA deformation imaging

STE-based analysis of LA mechanics was performed as previously recommended[17] and parameters analyzed offline by a specific software, with analysis of the automatically averaged longitudinal strain curves for each atrial segment[17]. For processing, the initial frame was chosen as the frame reflecting P-wave onset. LA strain and strain rate during systole (LA-εsys and LA-SRs), early diastole (LA-εe and LA-SRe), and late diastole (LA-εa and LA-SRa, respectively) were measured as indicators of the LA reservoir, conduit, and contractile functions, respectively[14].

Inter and intra-observer variability

Inter-observer reproducibility was confirmed by the analysis of a second investigator (JAF) of LA 2D-STE ε and SR measurements for 37 randomly selected HCM patients, that was compared with the analysis conducted by the first observer (PMA).

Intra-observer reproducibility was assessed by having the first observer (PMA) repeat the measurements for the same 37 participants. The observers evaluated different regions of interest in the LA and were blinded to previous measurements.

CMR

All 60 HCM patients underwent CMR, performed with 1.5 T scanners (Philips, Best, the Netherlands) using standard protocols as suggested previously[4]. LGE images were acquired 10–20 min after intravenous administration of gadolinium as recomended[2].

Quantification of LGE was then performed on all LGE-positive studies by manually adjusting a gray-scale threshold to define areas of visually identified LGE in short-axis planes and measured in centimeters (cm) of extension.

Statistical analysis

The normality of data distribution for continuous variables was assessed upon histogram observation and using the Kolmogorov–Smirnov test. Continuous variables were expressed as mean ± standard deviation, while categorical variables were expressed as percentage. Two- and three-group comparisons were conducted using the Student t-test and analysis of variance, respectively. For each variable with non-normal distribution, the homogeneity of variance was assessed using Levene's test. For categorical variables, the chi-square or Fisher's exact test was used, as appropriate. Linear regression was used to correlate several continuous parameters. Receiver operating characteristic curve analysis was performed to compute the discriminative power of LA mechanics parameters in HCM versus HTN. The curves were compared using the Delong method. We used the Bland–Altman method, intraclass correlation coefficient, and coefficient of variation to assess the inter- and intra-observer variability of LA 2D-STE ε and SR measurements. Two-sided P-values <0.05 were considered to indicate statistical significance. Stata IC for Windows (version 13; StataCorp, Lakeway Drive, TX, USA) and MedCalc for Windows (version 14.8.1; MedCalc Software, Ostend, Belgium) were used for the statistical analyses.

Results

Study population

The clinical features of the study population are summarized in *Table 1.* The mean age of HCM patients was 55±18 years, and 57% of patients were male. This did not vary significantly from HTN patients (61±12 years, 57% male) and controls (56±10 years, 55% male) (P>0.05).

In the HCM group, there was a higher use of beta-blocker therapy (75% cases), while in the HTN group, angiotensin-converting enzyme inhibitors (ACEI) or angiotensin receptor blockers (ARB) were more prescribed (75% cases), followed by calcium-channel blockers (CCB) (70% cases).

Conventional echocardiographic parameters

The echocardiographic characteristics are summarized in *Table 2.* The mean LVEF did not differ across groups. LVDD was higher in the HTN group than in the HCM group. PASP did not vary between the HCM and HTN group and was significantly lower in the control group.

With regard to LV diastolic function, mitral E velocity did not vary among groups, and mitral A velocity was lower in the control group than in the HTN group. E/e' ratio was only preserved in the control group and was significantly impaired in the HCM group.

LA function

Compared to controls, HCM and HTN patients had significantly larger LA volume indexed to the body surface area (LAVi) (33.5±2.5 and 31.1±1.3 vs 23.5±4.2 mL/m^2) (*Table 2).*. LA deformation parameters were globally decreased in either HTN or HCM groups, in relation to the control group. Particularly, in the HTN group, reservoir function was preserved (mean LA-sys 24.4±8.2% and LA-SRs 1.2±0.1%), although significantly lower compared to that in the control group. Conduit phase did not vary from the control group; contractile phase strain (but not LA-SRa) was significantly impaired in HTN patients (*Table 2, Figure 1).*.

All LA deformation phases were significantly reduced in the HCM group than in the HTN group (*Table 2, Figure 1).*. Of all LA mechanics parameters, LA-SRs and LA-SRa were the best discriminators of HCM (versus HTN), with an area under the curve (AUC) of 0.8, followed by LA sys (AUC 0.76). LA-SRs and LA-SRa had high specificity (89 and 91%, respectively), and LA sys had sensitivity of 80% (*Table 3).*. Although with lower discriminative power (AUC 0.65), LA e had the highest specificity (94%) and LA a the highest sensitivity (95%) (*Table 3).*.

CMR parameters

All HCM patients underwent CMR (*gold standard* for diagnosis). The mean indexed end-diastolic volume (EDVi) was 96±32 mL/m^2, the mean measured interventricular septum thickness (IVS) was 18.7±3.5 mm, approximately 34% of the patients had systolic anterior movement of the mitral valve, and 12% of the patients had apical HCM. LGE was present in 52 (87%) patients, and the mean measured area of extension was 2.8 cm.

CMR and echocardiographic parameters

Table 4 summarizes the results of the linear regression analysis between the extension of LGE (in cm) and several CMR and echocardiographic parameters. LGE was not correlated to other LV measures, namely, EDVi (by CMR), LVDD and LVSD by echocardiography, or even LV-GLS and E/E' ratio. LGE extension was mildly related to IVS thickness measured by CMR, but not by echography. A moderate correlation was found between the extension of LGE and LA sys and LA-SRe (*Figure 2).*.

Inter- and intra-observer variability in 2D-STE measurements of LA deformation

All LA deformation parameters exhibited intraclass correlation coefficient values of 0.64 to 0.94, indicating good to excellent reproducibility of such measurements (*Supplemental Table).* The Bland–Altman plots revealed very small inter-observer (*Figure 3)* and intra-observer*Figure 4)* discrepancy in the measurements of LA strain and strain rate.

Discussion

This study examined LA mechanics in LVH secondary to HTN and HCM and analyzed if LA remodeling was associated with the extent of LV hypertrophy and fibrosis in HCM. The results provide several notable insights into LA function. We could demonstrate that LA mechanics is globally decreased in either HTN or HCM. Particularly, LA function is significantly impaired in HCM, in relation to HTN (*Table 2, Figure 1).*. The best discriminators of HCM were LA-SRs and LA-SRa with an AUC of 0.8. LA e had the highest specificity (94%) and LA a the highest sensitivity (95%) (*Table 3).*. Furthermore, we could demonstrate a moderate correlation between LA mechanics and the degree of fibrosis assessed by CMR in HCM, namely, LA-SRe and extension of LGE (*Table 4).*.

LA reservoir function impairment is accompanied by deterioration of LV function, with greater LA dysfunction in HCM than in HTN. The reduction in LA reservoir function in HCM is related to the LV longitudinal dysfunction, due to a reduction in the systolic descent of LV base, which leads to an impaired LA relaxation and stiffness[7,18].

Although LA conduit function is associated to LV systolic function, namely ventricular desynchrony, it is less related to the extent of hypertrophy[2,18]. In our study, LA-SRe was the parameter that correlated better with the extent of fibrosis. This is probably explained by reduction of LV compliance due to myocardial fibrosis, with accompanying reduction of the atrial conduit function in HCM.

LA contractile phase was also impaired in HCM, which is somewhat inconsistent with previous reports that showed a trend toward increased LA contractile booster pump function in HCM with absent LV fibrosis (although not statistically significant)[2]. This might be related to the fact that 87% of our HCM patients already presented with LGE on CMR, so LA contractile function might already be compromised.

When evaluating diastolic dysfunction in HCM versus HTN, we noted that LAVi and mitral E and A velocities did not vary between groups. E/E' ratio, albeit varying significantly between groups, was not a good discriminator between groups and was not correlated to the extent of myocardial fibrosis in HCM. LA mechanics appear as a more discriminator factor between LVH groups and is related to the degree of myocardial fibrosis in HCM. This suggests that LA mechanics might be an earlier marker of both atrial and myocardial dysfunctions. Furthermore, in HTN patients with significant LVH, in which excluding HCM might be challenging, the evaluation of LA mechanics might be useful, since the three phases are not very impaired in this group. In our study, albeit being statistically inferior from HCM, the IVS thickness values of our HTN cohort were still high (mean of 14.3±3.6 mm).

Our study has several limitations. First, we only included 60 HCM patients and 60 HTN patients, which may have influenced the statistical power of the analysis. Second, this was not an outcome-based study, so we could not draw any conclusions about the prognostic value of LA strain rate in this population. Nevertheless, this study attempted to clarify the LA deformation mechanics in LVH secondary to HCM and to HTN. Future studies with large sample size are warranted to clarify the prognostic value of LA strain rate in LVH.

In this study, we have demonstrated that LA strain and strain rate were potential discriminators between HCM and HTN, not only in physiologic response to LVH, but also to the determinants of dysfunction. This represents an important finding of our study, since the LAVi, E/E' ratio, and even IVS thickness were not sufficient reliable to do so (*Tables 2 and 3)*.. In addition, LA mechanics was moderately correlated to fibrosis extension in HCM, which can potentially become a marker of severity and prognosis in earlier stages or doubtful cases.

Conclusion

LA mechanics is globally impaired in LVH secondary to HTN and to HCM. Compared to HTN, the best discriminators of HCM were LA-SRs and LA-SRa. However, LA-SRe was best correlated with the degree of fibrosis assessed by CMR. These findings suggest that LA mechanics can help differentiating LVH between HTN and HCM and even can be potential predictors of disease severity in HCM.

Abbreviations

ACEI—angiotensin converter enzyme inhibitors

AF—atrial fibrillation

ARB—angiotensin receptor blockers

CCB—calcium-channel blockers

CI—confidence interval

CMR—cardiac magnetic resonance

—strain

HCM—hypertrophic cardiomyopathy

HTN—arterial hypertension

LA—left atrium

LAVi—left atrial volume (indexed to the body surface area)

LA-sys—left atrial systolic strain (reservoir function)

LA-e—left atrial early diastolic strain (conduit function)

LA-a—left atrial late diastolic strain (contractile function)

LA-SRs—left atrial systolic strain rate (reservoir function)

LA-SRe—left atrial early diastolic strain rate (conduit function)

LA-SRa—left atrial late diastolic strain (contractile function)

LGE—late gadolinium enhancement

LV-GLS—left ventricular global longitudinal strain

LVDD—left ventricular end-diastolic diameter

LVSD—left ventricular end-systolic diameter

LVEF—left ventricular ejection fraction

LVH—left ventricular hypertrophy

PASP—pulmonary artery systolic pressure

STE—speckle-tracking echocardiography

Declarations

Ethics approval and consent to participate

Since it was an observational analysis of a previously anonymized database from exams that are routinely done in the specific group of patients, only verbal informed consent was obtained. The study was approved by the ethics committee of Coimbra Hospital and University Centre.

Consent for publication

Not applicable.

Availability of data and materials

The datasets used and/or analyzed during the current study are available from the corresponding author on reasonable request.

Competing interests

The authors declare that they have no competing interests in this section.

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Authors' contributions

PMA, conception and design, analysis and interpretation of data, manuscript writing. CD, AVM, JAF, JPA, acquisition, analysis and interpretation of data. RB conception and design, analysis and interpretation of data, revising the manuscript critically for important intellectual content. GC, RM, PD, MJF, attending physicians, analysis and interpretation of data, revising the manuscript critically for important intellectual content. MJF, LG interpretation of data, revising the manuscript critically for important intellectual content. All authors read and approved the final manuscript

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Tables

Table 1. Demographic and clinical characteristics of the study population.

Characteristic	Controls (n=37)	HCM group (n=60)	HTN group (n=60)	P-value
Age, years	56±10	55±18	61±12	0.081
Male sex	20 (55)	34 (57)	34 (57)	0.124
Beta-blocker use		45 (75)	37 (62)	0.068
ACEI/ARB use		41 (68)	45 (75)	0.072
CCB use		27 (45)	42 (70)	0.032
Other anti-HTN use		12 (20)	20 (34)	0.056
Data are given as mean ± standard deviation or as frequency (percentage). ACEI/ARB, angiotensin converting enzyme inhibitor/angiotensin receptor blockers; CCB, calcium channel blocker; HCM, hypertrophic cardiomyopathy; HTN, arterial hypertension.				

Table 2. Echocardiographic parameters of the study population.

Parameters	Controls	HTN group	HCM group	Global P-value	P value: controls vs HTN	P-value: HCM vs HTN
LVEF, %	62.9±4.3	62.9±4.9	66.5±10.1	0.083	0.969	0.055
LVDD, mm	48.3±5.2	51.9±0.8	49.4±1.0	0.108	0.019	0.083
LVSD, mm	30.3±3.2	32.3±0.7	30.7±0.9	0.369	0.119	0.225
IVS, mm	10.2±2.8	14.3±3.6	16.5±5.4	0.028	<0.001	0.032
LV-GLS, %	-20.6±1.1	-17.5±0.7	-12.7±0.5	<0.001	0.192	0.008
PASP, mmHg	22.1±4.7	26.3±0.2	28.6±1.3	0.021	0.009	0.245
LAVi, mL/m^2	23.5±4.2	31.1±1.3	33.5±2.5	<0.001	0.001	0.067
Mitral E velocity, m/s	0.8±0.1	0.7±0.2	0.8±0.2	0.156	0.068	0.182
Mitral A velocity, m/s	0.5±0.1	0.8±0.2	0.7±0.3	0.005	<0.001	0.151
E/e' ratio	7.0±1.65	13.2±1.2	16±1.0	<0.001	<0.001	0.035
LA-sys, %	36.9±10.8	24.4±8.2	17.2±9.0	<0.001	<0.001	<0.001
LA-e, %	25.9±13.3	19.9±8.7	15.4±9.1	<0.001	0.067	0.022
LA-a, %	10.9±6.2	5.1±0.9	1.9±0.3	<0.001	0.003	<0.001
LA-SRs, %	1.9±0.5	1.2±0.1	0.8±0.1	<0.001	<0.001	<0.001
LA-SRe, %	-2.1±0.6	-1.8±0.1	-0.6±0.1	<0.001	0.082	<0.001
LA-SRa, %	-1.9±0.7	-1.7±0.1	-0.9±0.1	<0.001	0.344	<0.001
HCM, hypertrophic cardiomyopathy; HTN, hypertension; IVS, interventricular septum; LA sys, left atrial systolic strain (reservoir function); LA e, left atrial early diastolic strain (conduit function); LA a, left atrial late diastolic strain (contractile function); LA SRs, left atrial systolic strain rate (reservoir function); LA SRe, left atrial early diastolic strain rate (conduit function); LA SRa, left atrial late diastolic strain (contractile function); LAVi, left atrial volume indexed; LVDD, left ventricular end-diastolic diameter; LVSD, left ventricular end-systolic diameter; LVEF, left ventricular ejection fraction; LV-GLS, left ventricular global longitudinal strain.						

Table 3. Discriminative power of echocardiographic parameters (HCM vs HTN groups)

	AUC	95% CI	*P-value*	Sensitivity (%)	Specificity (%)	Criterion
LA sys (%)	0.76	0.66-0.84	<0.001	80	71	21.8
LA e (%)	0.65	0.54-0.74	0.012	32	94	9.9
LA a (%)	0.65	0.54-0.75	0.016	95	34	5.1
LA SRs (%)	0.80	0.71-0.88	<0.001	65	89	0.8
LA SRe (%)	0.69	0.59-0.79	<0.001	54	87	-0.8
LA SRa (%)	0.80	0.71-0.88	<0.001	64	91	-0.9
IVS (mm)	0.62	0.51-0.70	0.012	55	74	15.2
LV-GLS (%)	0.74	0.64-0.83	<0.001	57	84	-13.5
E/e' ratio	0.67	0.55-0.78	0.009	67	71	13

AUC, area under the curve; HTN, arterial hypertension; HCM, hypertrophic cardiomyopathy; IVS, interventricular septum; LA sys, left atrial systolic strain (reservoir function); LA e, left atrial early diastolic strain (conduit function); LA a, left atrial late diastolic strain (contractile function); LA SRs, left atrial systolic strain rate (reservoir function); LA SRe, left atrial early diastolic strain rate (conduit function); LA SRa, left atrial late diastolic strain (contractile function); LV-GLS, left ventricular global longitudinal strain.

Table 4. Linear regression analysis between the extension of LGE (in cm) and several CMR and echocardiographic parameters.

LGE	R^2	P-value
IVS by CMR	**0.32**	**0.051**
IVS by echocardiography	0.24	0.088
EDVi by CMR	0.01	0.843
LVDD	0.01	0.795
E/E' ratio	0.01	0.802
LV-GLS	0.04	0.467
LA sys	0.12	0.085
LA e	0.15	0.092
LA a	**0.35**	**0.045**
LA SRs	0.12	0.073
LA SRe	**0.42**	**0.027**
LA SRa	0.21	0.066

AUC, area under the curve; CMR, cardiac magnetic resonance; EDVi, indexed end-diastolic volume; IVS, interventricular septum; LA sys, left atrial systolic strain (reservoir function); LA e, left atrial early diastolic strain (conduit function); LA a, left atrial late diastolic strain (contractile function); LA SRs, left atrial systolic strain rate (reservoir function); LA SRe, left atrial early diastolic strain rate (conduit function); LA SRa, left atrial late diastolic strain (contractile function); LGE, late gadolinium enhancement; LVDD left ventricular end-diastolic diameter; LV-GLS, left ventricular global longitudinal strain

Figures

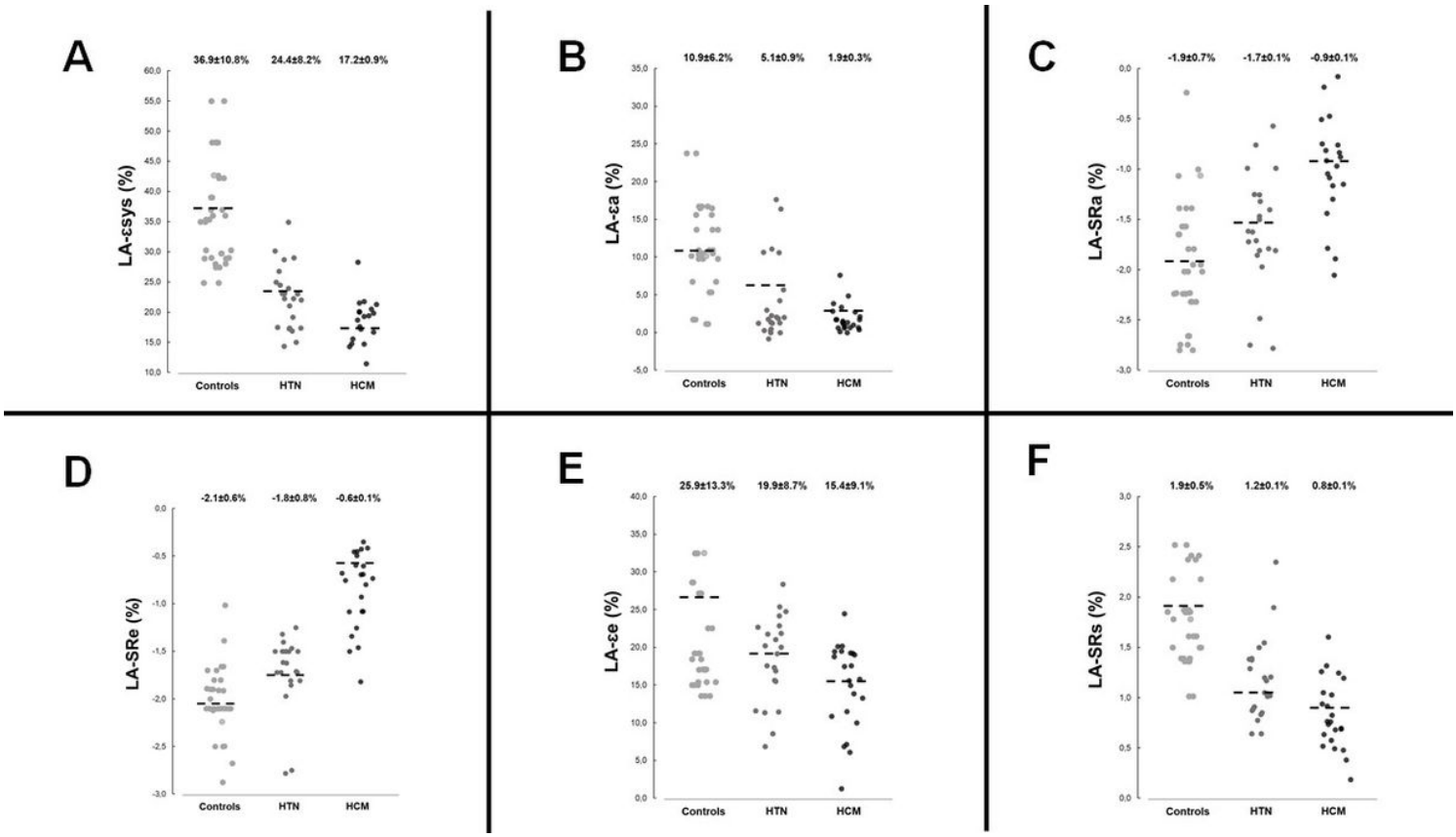

Figure 1

LA deformation parameters in the control, HTN, and HCM groups. Reservoir phase strain (A) and strain rate (D), conduit phase (B) strain and strain rate (E), contractile phase strain (C), and strain rate (E) were evaluated. HCM, hypertrophic cardiomyopathy; HTN, arterial hypertension; LA εsys, left atrial systolic strain (reservoir function); LA εe, left atrial early diastolic strain (conduit function); LA εa, left atrial late diastolic strain (contractile function); LA SRs, left atrial systolic strain rate (reservoir function); LA SRe, left atrial early diastolic strain rate (conduit function); LA SRa, left atrial late diastolic strain (contractile function).

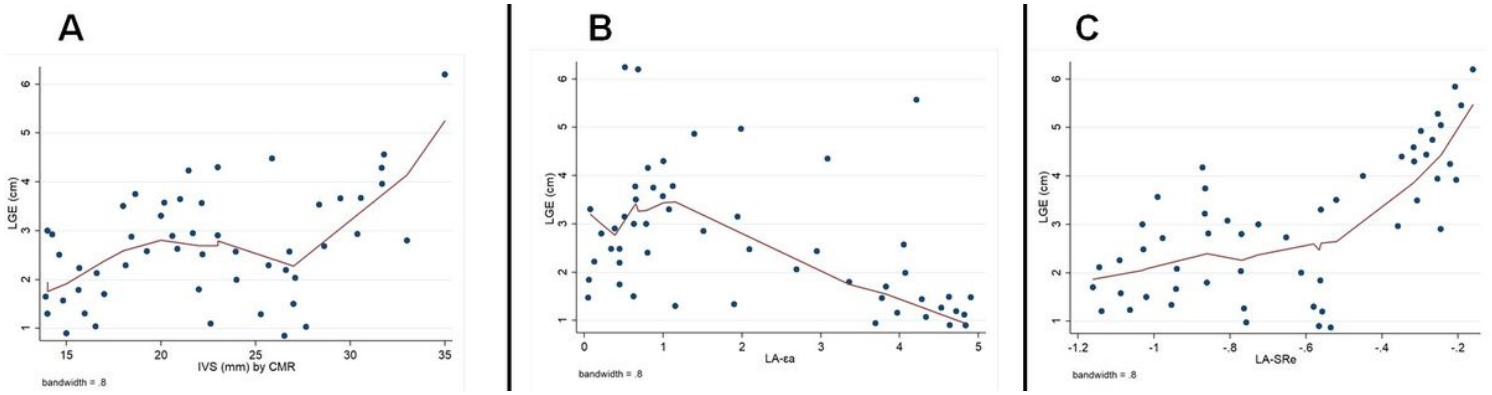

Figure 2

Linear correlation between LGE extension and IVS thickness measured by CMR (A), LA-εa (B) and LA-SRe (C). CMR, cardiac magnetic resonance; IVS, interventricular septum; LA εa, left atrial late diastolic strain (contractile function); LA SRs, left atrial systolic strain rate (reservoir function); LA SRe, left atrial early diastolic strain rate (conduit function); LA SRa, left atrial late diastolic strain (contractile function); LGE, late gadolinium enhancement; LVDD left ventricular end-diastolic diameter; LV-GLS, left ventricular global longitudinal strain

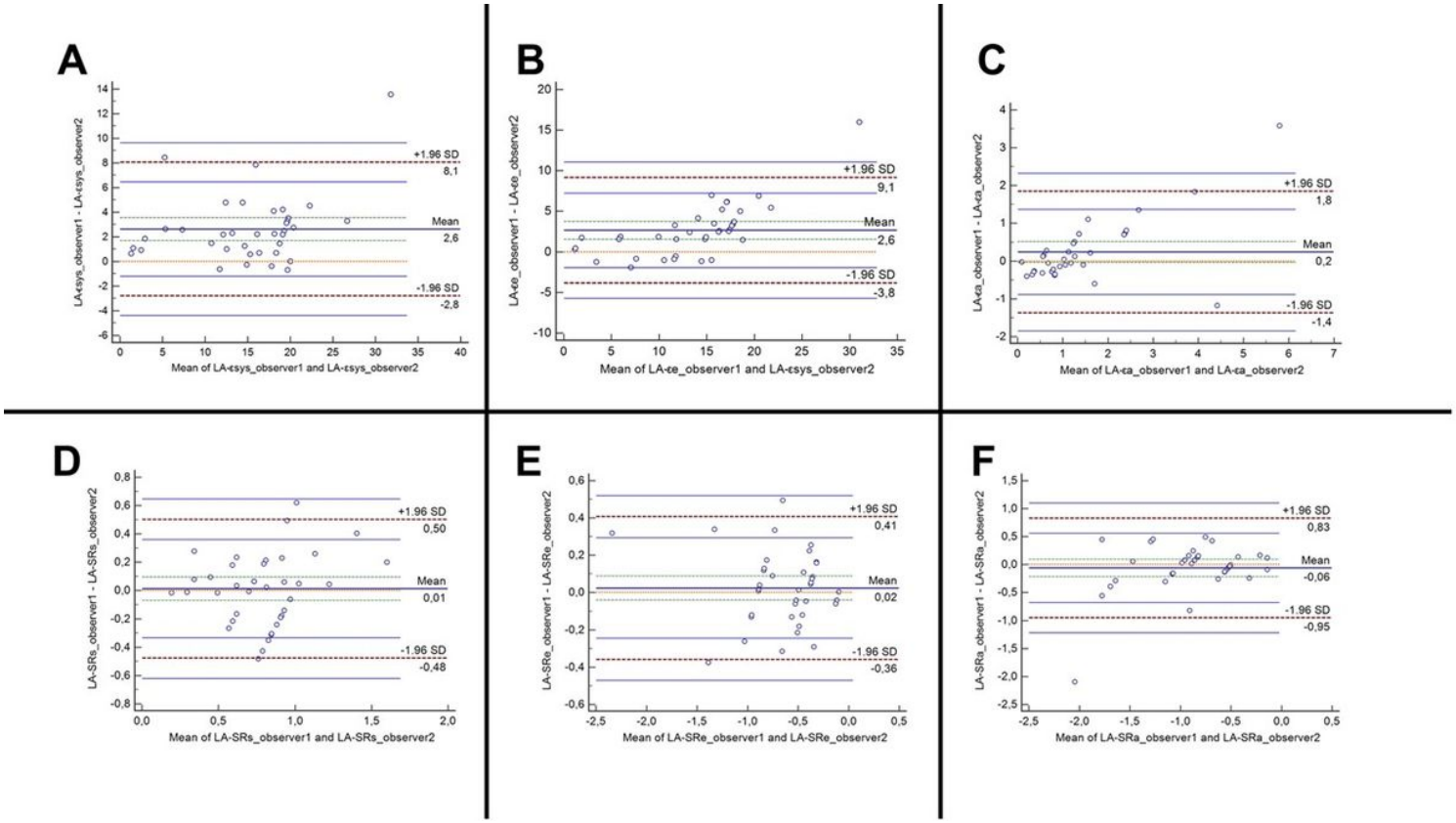

Figure 3

Bland–Altman plots for inter-observer variability of left atrial strain (A, B, C) and strain rate (D, E, F) measurements. LA-εsys, left atrial systolic strain (reservoir function); LA-εe, left atrial early diastolic strain (conduit function); LA-εa, left atrial late diastolic strain (contractile function); LA-SRs, left atrial systolic strain rate (reservoir function); LA-SRe, left atrial early diastolic strain rate (conduit function); LA-SRa, left atrial late diastolic strain rate (contractile function).

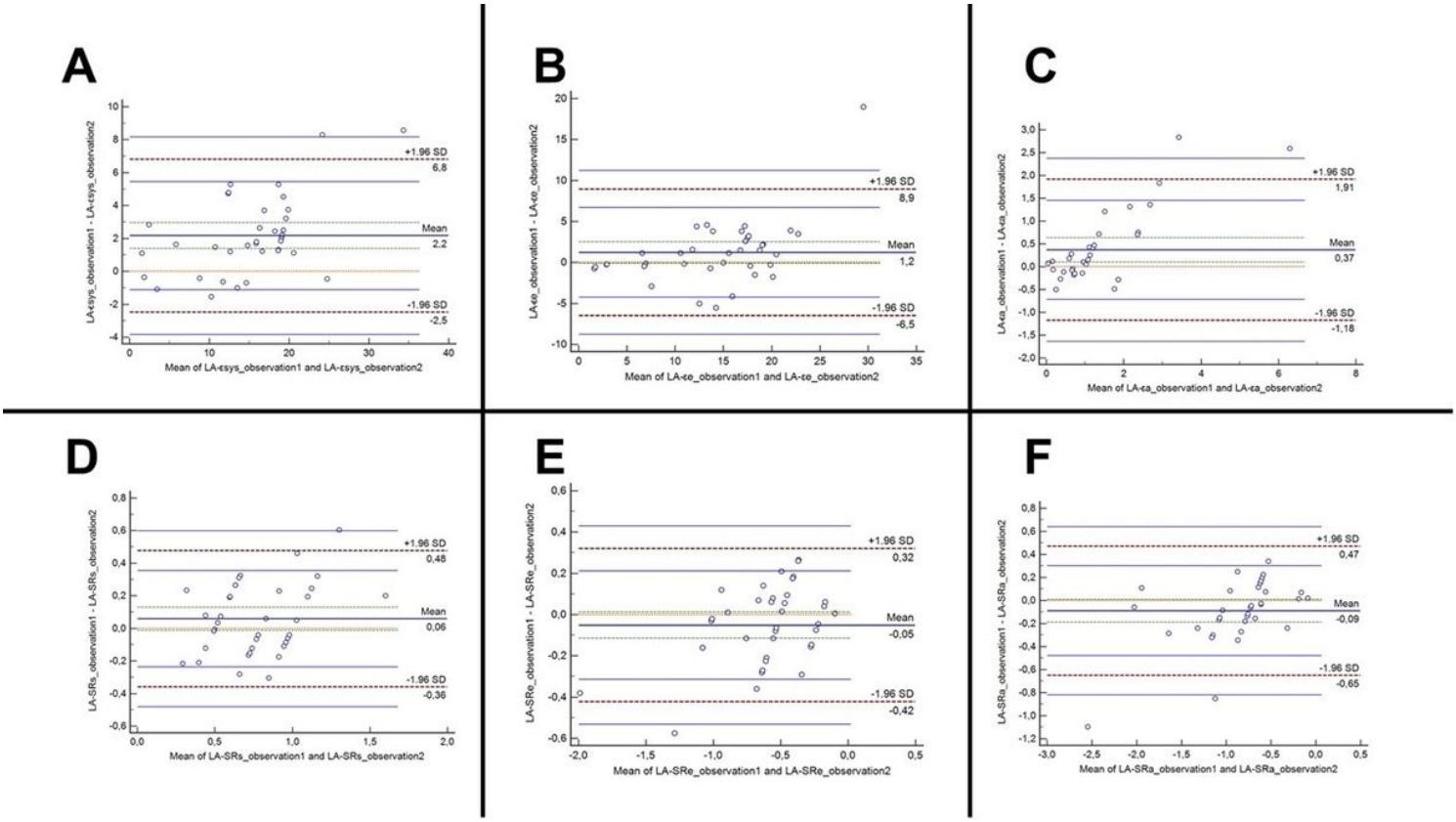

Figure 4

Bland–Altman plots for intra-observer variability of left atrial strain (A, B, C) and strain rate (D, E, F) measurements. LA-εsys, left atrial systolic strain (reservoir function); LA-εe, left atrial early diastolic strain (conduit function); LA-εa, left atrial late diastolic strain (contractile function); LA-SRs, left atrial systolic strain rate (reservoir function); LA-SRe, left atrial early diastolic strain rate (conduit function); LA-SRa, left atrial late diastolic strain rate (contractile function).

Supplementary Files

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