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Quantitative Assessment of Left Atrial and Ventricular Function in Hypertensive Heart Disease via Cardiac Magnetic Resonance Feature Tracking: An Experimental Study

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Abstract: *Objective: To investigate the value of cardiac magnetic resonance feature tracking (CMR-FT) in assessing left heart function in hypertensive heart disease (HHD) and to preliminarily explore the relationship between left atrial (LA) and left ventricular (LV) function1. Methods: Ten Bama minipigs were randomly divided into an experimental group (N=6) and a control group (N=4). The experimental group underwent laparotomy with left renal artery ligation to establish a hypertension model, while the control group underwent laparotomy without ligation. CMR-FT examinations were performed at baseline, 4 weeks, and 16 weeks post-surgery. Global LA and LV functional parameters were measured using CVI42 post-processing software. Results: Left atrial volumetric indices (LAVImin and LAVImax) did not differ between groups at baseline or 4 weeks (P>0.05), but were significantly higher in the hypertension group at 16 weeks (LAVImin 17.65±1.66 vs 11.13±1.60 mL/m², P<0.001; LAVImax 27.05±2.19 vs 20.75±3.13 mL/m², P=0.005). LA reservoir and conduit function deteriorated early: εs and εe were reduced in the hypertension group at 4 weeks (εs 36.47±3.08 vs 41.80±2.92, P=0.026; εe 20.45±1.53 vs 23.43±2.15, P=0.033) and further decreased at 16 weeks (εs 27.22±3.71 vs 39.53±2.72, P<0.001; εe 12.10±2.02 vs 23.00±1.77, P<0.001). LV volumetric parameters and LVEF remained comparable between groups at all time points (all P>0.05), whereas LV strain indices showed significant impairment in the hypertension group at 4 weeks and 16 weeks (e.g., LVRS 35.05±2.39 vs 43.45±1.80 and 29.55±1.80 vs 43.18±1.35; LVCS −18.42±1.40 vs −20.66±1.42 and −16.58±0.87 vs −19.63±0.99; LVLS −15.35±0.91 vs −18.90±0.50 and −12.63±1.73 vs −18.33±0.76; all P≤0.039). LA volumes were strongly and inversely correlated with LA strains (LAVmin with εs r=−0.845 and εe r=−0.838; LAVmax with εs r=−0.863 and εe r=−0.871; all P<0.05). Moreover, LVLS correlated positively with εs (r=0.814) and εe (r=0.875) and negatively with LA volumes (LAVmin r=−0.817; LAVmax r=−0.907), while LVRS was also associated with LA function (all P<0.05). Conclusion: CMR-FT can detect structural and functional impairments of the LA and LV in early-stage HHD earlier, more sensitively, and more accurately than conventional cardiac functional parameters. Furthermore, significant correlations exist between LA and LV functional parameters.*

Keywords: Hypertensive heart disease, Cardiac magnetic resonance, Myocardial strain, Left ventricular function, Left atrial function.

1. Introduction

Hypertension is a primary risk factor for cardiovascular, cerebrovascular, and renal diseases. Globally, it imposes a staggering clinical and socioeconomic burden, affecting approximately one billion individuals and precipitating millions of deaths annually [1]. In the heart, chronically elevated blood pressure induces hypertensive heart disease (HHD), characterized by progressive structural and functional deterioration of the myocardium. The hallmark phenotypes of HHD include left ventricular hypertrophy (LVH) and subsequent systolic and diastolic dysfunction [2]. The pathophysiology of LVH involves a complex interplay of mechanical stress, neurohormonal activation, growth factors, and cytokine-mediated signaling. Physiologically, the left atrium (LA) plays a pivotal role in cardiac hemodynamics: it functions as a reservoir to collect pulmonary venous flow during left ventricular (LV) systole and acts as a conduit to transport blood to the LV during diastole. In the setting of hypertension, increased LV afterload necessitates higher generation of LV pressure, which retrogradely elevates LV filling pressures and, consequently, LA wall stress [3]. Hypertension-mediated LA structural and functional remodeling is increasingly recognized as a fundamental substrate or precipitating factor for the development of heart failure [4]. Therefore, characterizing the process of LA remodeling in hypertension is critical. Notably, emerging evidence suggests that LA functional abnormalities may manifest prior to overt LV structural remodeling [5]. However, the precise relationship between this aberrant LA function and subclinical LV dysfunction remains insufficiently evaluated.

Although echocardiography is widely utilized for cardiac assessment, it is inherently limited by operator dependence and low reproducibility. Cardiac Magnetic Resonance (CMR) offers a comprehensive evaluation of cardiac morphology, function, and tissue characterization. A significant diagnostic challenge in HHD is that conventional global functional parameters often remain preserved or even elevated during the compensated phase, rendering them insensitive markers for early myocardial injury [6]. In contrast, CMR Feature Tracking (CMR-FT) has emerged as a robust technique capable of quantifying subclinical myocardial dysfunction through myocardial strain parameters.

Animal models are indispensable for elucidating the pathogenesis of hypertension and its associated cardiac remodeling [7]. Porcine models, in particular, possess cardiovascular physiology, anatomy, and function highly homologous to humans [8]. Furthermore, the larger body size of swine allows for better tolerance of surgical interventions and CMR acquisition. In this study, we established a porcine model of HHD via left renal artery ligation to longitudinally assess left heart mechanics. By leveraging CMR-FT, we aimed to quantitatively evaluate subclinical left heart dysfunction and preliminarily investigate the dynamic coupling between LA and LV function. This study seeks to establish a reliable non-invasive imaging strategy for the early diagnosis of HHD, provide a theoretical basis for further mechanistic studies on atrioventricular coupling, and offer reference data regarding the temporal progression of HHD in a clinical context.

2. Materials and Methods

This study was approved by the Hospital Animal Ethics Committee (No. YXKT2024L016). Ten Bama minipigs (aged 9–12 months; weight 25–30 kg) were included and randomized into a hypertension group (N=6) and a control group (N=4).

2.1 Animal Model Establishment

All surgical procedures were performed under general anesthesia. Animals were restrained, and venous access was established via the marginal ear vein. Anesthesia was induced and maintained with Propofol

(AstraZeneca UK Limited) administered as bolus injections (5–8 mL/dose). Preoperative Ceftiofur Hydrochloride (5 mg/kg) was administered for infection prophylaxis. In the hypertension group, the abdomen was prepared and draped. A laparotomy was performed through a left paramedian incision (along the lateral edge of the rectus abdominis) to expose the retroperitoneum. The left renal artery was isolated and ligated using No. 1 suture material. The control group underwent an identical surgical procedure with renal artery isolation but without ligation. Post-modeling analgesia was provided by intramuscular injection of Ketoprofen (3 mg/kg) once daily for three consecutive days.

2.2 Acquisition of Left Heart Functional Parameters

CMR Image Acquisition Cardiac Magnetic Resonance (CMR) examinations were performed at baseline, 4 weeks, and 16 weeks post-operation using a 3.0-T scanner (MAGNETOM Skyra, Siemens Healthineers). Balanced steady-state free precession (bSSFP) cine sequences were acquired in the left ventricular (LV) two-chamber, four-chamber, and short-axis planes (covering the entire ventricle from base to apex). Imaging parameters were as follows: slice thickness 6 mm; TR 38.52 ms; TE 1.40 ms; FOV 270 × 270 mm; and flip angle 49°.

Image Post-processing Image analysis was performed using CVI42 (Circle Cardiovascular Imaging Inc, Calgary, Canada). Left Atrial (LA) Volumetry: Using the biplane Long-Axis (LAX) module, LA endocardial contours were manually traced on cine images at LV end-systole (LAVmax) and LV end-diastole (LAVmin). Volumes were indexed to body surface area (BSA) to derive the LA maximum volume index (LAVImax) and minimum volume index (LAVImin). Left Ventricular (LV) Function: Using the 3D Short-Axis (SAX) module, endocardial and epicardial contours (excluding papillary muscles and epicardial fat) were semi-automatically traced on the short-axis stack and manually corrected. LV functional parameters, including end-diastolic volume (LVEDV), end-systolic volume (LVESV), stroke volume (SV), and ejection fraction (LVEF), were calculated. Volumetric indices (LVEDVI, LVESVI, LVSVI) were derived based on BSA. Feature Tracking (CMR-FT) LA Strain: LA endocardial contours (excluding pulmonary veins and the left atrial appendage) were tracked in the LV systolic and diastolic phases using the strain analysis module. The software automatically calculated global LA longitudinal strain parameters: total strain (εs), active strain (εa), and passive strain εe = (εs - εa).LV Strain: LV radial strain (LVRS), circumferential strain (LVCS), and longitudinal strain (LVLS) were derived using tissue feature tracking technology.

2.3 Statistical Analysis

Continuous variables were tested for normality using the Kolmogorov-Smirnov test. Data are presented as mean ± standard deviation (SD) or median (interquartile range). Intra-group comparisons: Differences across the three time points (baseline, 4 weeks, 16 weeks) within groups were analyzed using one-way analysis of variance (ANOVA) followed by Games-Howell post-hoc tests. Inter-group comparisons: Differences between the hypertension and control groups at each time point were assessed using the independent samples t-test or Mann-Whitney U test. Correlations: The relationship between LA and LV functional parameters was evaluated using Pearson correlation analysis. All statistical analyses were performed using SPSS version 27.0((IBM Corp, Armonk, NY, USA). Statistical significance was defined as $P < 0.05$.

3. Results

All animals successfully completed the experimental protocol. A total of 10 Bama minipigs were included in the final analysis, consisting of 6 in the hypertension group and 4 in the control group. CMR-

FT image acquisition and post-processing analysis were completed for all animals at baseline, 4 weeks, and 16 weeks post-surgery.

3.1 Left Atrial Functional Parameters Analysis

Longitudinal analysis revealed progressive LA remodeling in the hypertension group. Both LAVmin and LAVmax increased continuously from baseline to 16 weeks in both groups. While no significant inter-group differences were observed at baseline or 4 weeks ($P > 0.05$), the hypertension group exhibited significantly higher LAVmin, LAVmax, LAVImin, and LAVImax compared to the control group at 16 weeks ($P < 0.05$).LA strain parameters demonstrated a progressive decline in the hypertension group. εs and εe were significantly lower in the hypertension group compared to controls at both 4 weeks and 16 weeks ($P < 0.05$). εa showed no significant difference between groups at baseline or 4 weeks ($P > 0.05$) but was significantly reduced in the hypertension group at 16 weeks ($P < 0.05$). (Table 1).

3.2 Left Ventricular Functional Parameters Analysis

Systolic and diastolic blood pressures in the hypertension group increased progressively, with significant elevations compared to controls at 4 and 16 weeks ($P < 0.05$). Although LVEDV, LVESV, and SV increased over time within groups (reflecting physiological growth), there were no statistically significant differences in LVEDV, LVESV, SV, or LVEF between the hypertension and control groups at any time point ($P > 0.05$). This indicates preserved global systolic function and volume in the early stages of HHD. In contrast to volumetric parameters, LV deformation markers deteriorated early. LVRS, LVCS, and LVLS in the hypertension group decreased progressively from baseline. Significant reductions in these strain parameters compared to controls were evident as early as 4 weeks and persisted at 16 weeks ($P < 0.05$). (Table 2)

Table 1: Comparison of left atrial function parameters at baseline, 4 weeks and 16 weeks after model induction between the hypertension group and the experimental group

	baseline		*t*	*P*	*4 weeks*		*t*	*P*	*16 weeks*		*t*	*P*
	HHD	*Control*			*HHD*	*Control*			*HHD*	*Control*		
LAVmin	*9.62±1.45*	*9.95±1.30*	*-0.369*	*0.722*	*11.48±1.47**	*11.40±1.24*	*0.093*	*0.928*	*22.23±1.69*#*	*17.20±2.93*#*	*3.482*	*0.008*
LAVmax	*15.65±2.25*	*15.43±2.85*	*0.140*	*0.892*	*19.18±1.65**	*19.63±2.92*	*-0.309*	*0.765*	*34.03±1.98*#*	*25.08±3.45*#*	*5.276*	*0.001*
LAVImin	*11.52±1.62*	*12.40±1.49*	*-0.869*	*0.410*	*11.35±1.43*	*11.38±1.26*	*-0.028*	*0.978*	*17.65±1.66*#*	*11.13±1.60*	*6.174*	*0.000*
LAVImax	*18.70±2.37*	*19.25±3.58*	*-0.296*	*0.775*	*18.80±2.35*	*19.58±2.88*	*-0.469*	*0.652*	*27.05±2.19*#*	*20.75±3.13*	*3.779*	*0.005*
εs	*44.22±2.81*	*42.85±3.27*	*0.708*	*0.499*	*36.47±3.08**	*41.80±2.92*	*-2.738*	*0.026*	*27.22±3.71*#*	*39.53±2.72*	*-5.659*	*0.000*
εa	*19.88±1.33*	*19.43±0.86*	*0.604*	*0.563*	*18.02±1.71**	*18.88±1.05*	*-0.890*	*0.400*	*15.20±1.65*#*	*17.88±1.75*	*-2.456*	*0.040*
εe	*24.33±1.59*	*23.93±2.29*	*0.336*	*0.746*	*20.45±1.53**	*23.43±2.15*	*-2.575*	*0.033*	*12.10±2.02*#*	*23.00±1.77*	*-8.760*	*0.000*

Note: Left Atrial Minimum Volume (LAVmin), Left Atrial Maximum Volume (LAVmax), Left Atrial Minimum Volume Index (LAVImin), Left Atrial Maximum Volume Index (LAVImax), Total strain (εs), active strain (εa), passive strain (εe)

* indicates a difference within the same group compared with the baseline (P<0.05)

# indicates a difference within the same group compared with 4 weeks (P<0.05).

Table 2: Comparison of left ventricular function parameters at baseline, 4 weeks and 16 weeks after model induction between the hypertension group and the control group at each time point

	baseline		*t*	*P*	*4 weeks*		*t*	*P*	*16 weeks*		*t*	*P*
	HHD	*Control*			*HHD*	*Control*			*HHD*	*Control*		
SBP	*108.17±4.02*	*109.25±10.69*	*-0.231*	*0.823*	*133.00±4.98**	*105.75±8.10*	*6.667*	*0.000*	*144.33±3.72*#*	*109.75±6.45*	*10.877*	*0.000*
DBP	*67.33±3.33*	*71.50±7.23*	*-1.253*	*0.246*	*88.17±3.87**	*71.50±5.97*	*5.416*	*0.001*	*96.67±3.01*#*	*71.75±6.4*	*8.421*	*0.000*
LVEDV	*45.00±3.50*	*43.95±3.99*	*0.441*	*0.671*	*54.88±2.02**	*56.33±1.50**	*-1.214*	*0.260*	*63.93±3.33*#*	*64.38±4.02*#*	*-0.190*	*0.854*
LVESV	*13.07±3.77*	*13.70±3.01*	*-0.280*	*0.787*	*17.27±3.29**	*17.85±3.41**	*-0.271*	*0.793*	*20.18±3.38*#*	*21.43±3.17* #*	*-0.582*	*0.577*
LVSV	*32.02±3.93*	*30.25±1.31*	*0.853*	*0.418*	*37.62±3.11**	*38.48±2.05**	*-0.482*	*0.643*	*43.75±4.43*#*	*42.95±4.64*#*	*0.275*	*0.790*
LVEF	*70.20±7.67*	*69.10±4.16*	*0.495*	*0.634*	*68.58±5.51*	*68.38±5.27*	*0.060*	*0.954*	*67.88±5.76*	*66.73±5.19*	*0.323*	*0.755*
LVEDVI	*53.88±3.57*	*54.75±3.99*	*-0.360*	*0.728*	*54.25±2.23*	*56.20±1.15*	*-1.592*	*0.150*	*52.90±2.96*	*53.08±2.50*	*-0.097*	*0.925*
LVESVI	*15.63±4.45*	*17.05±3.43*	*-0.535*	*0.607*	*17.07±3.25*	*17.80±3.31*	*-0.347*	*0.738*	*16.65±2.83*	*17.68±2.52*	*-0.585*	*0.575*
LVSVI	*38.38±4.49*	*37.68±1.00*	*0.305*	*0.768*	*36.92±3.29*	*38.40±2.16*	*-0.788*	*0.454*	*37.20±2.40*	*35.43±3.57*	*0.950*	*0.370*
LVRS	*42.58±2.24*	*44.48±3.71*	*-1.018*	*0.338*	*35.05±2.39**	*43.45±1.80*	*-5.939*	*0.000*	*29.55±1.80*#*	*43.18±1.35*	*-12.857*	*0.000*
LVCS	*-20.78±1.66*	*-21.13±1.81*	*0.308*	*0.766*	*-18.42±1.40**	*-20.66±1.42*	*2.458*	*0.039*	*-16.58±0.87*#*	*-19.63±0.99*	*5.151*	*0.001*
LVLS	*-18.27±1.28*	*-18.60±1.08*	*0.428*	*0.680*	*-15.35±0.91**	*-18.90±0.50*	*7.031*	*0.000*	*-12.63±1.73*#*	*-18.33±0.76*	*6.092*	*0.000*

Note: Systolic Blood Pressure (SBP), Diastolic Blood Pressure (DBP),Left Ventricular End-Diastolic Volume (LVEDV), Left Ventricular End-Systolic Volume (LVESV) Left Ventricular Stroke Volume (LVSV), Left Ventricular Ejection Fraction (LVEF) Left Ventricular End-Diastolic Volume Index (LVEDVI), Left Ventricular End-Systolic Volume Index (LVESVI), Left Ventricular Stroke Volume Index (LVSVI), Left Ventricular Radial Strain (LVRS) Left Ventricular Circumferential Strain (LVCS), Left Ventricular Longitudinal Strain (LVLS).

* indicates a difference within the same group compared with the baseline (P<0.05)

# indicates a difference within the same group compared with 4 weeks (P<0.05).

3.3 Correlation Analysis of Left Atrial Functional Parameters

The results demonstrated a strong inverse correlation between LA structure and function: LAVmin was negatively correlated with εs (r=-0.845, P < 0.05) and εe (r=-0.838, P < 0.05). LAVmax was negatively correlated with εs (r=-0.863, P < 0.05) and εe (r=-0.871, P < 0.05). (Figure 1).

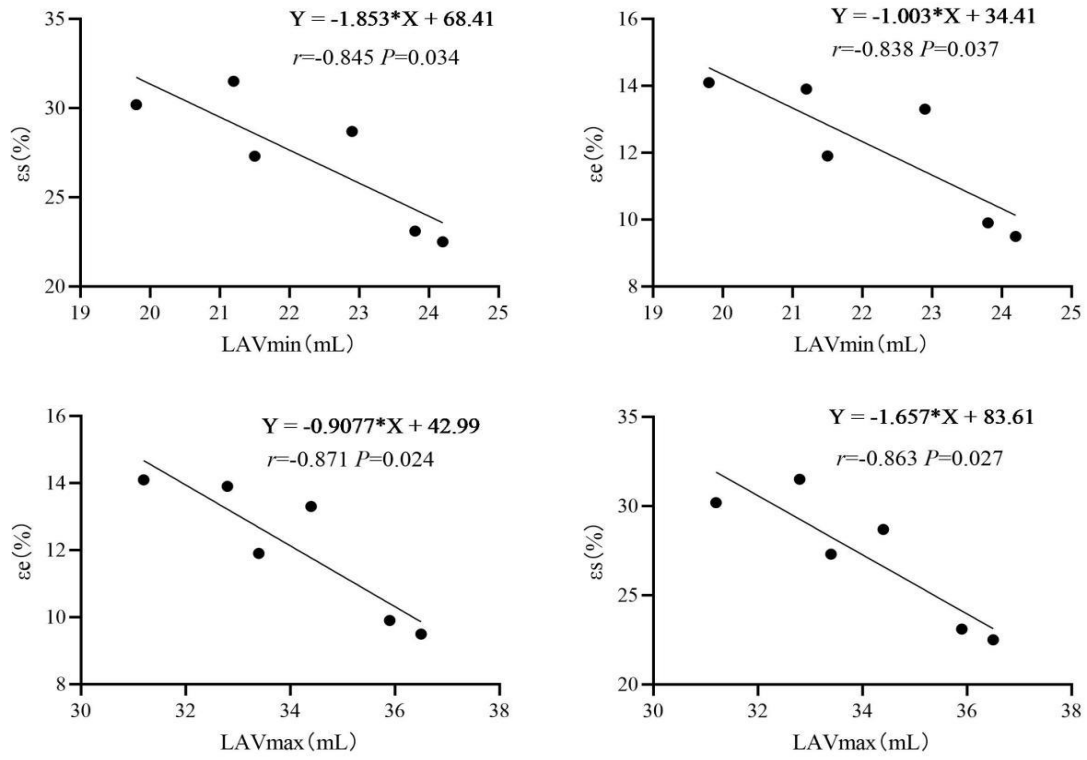

Figure 1: illustrates the correlation between left atrial volume and left atrial strain.

3.4 Correlation Analysis of Left Ventricular and Left Atrial Parameters

Significant coupling between LV mechanics and LA function: LVLS showed a significant positive correlation with εs (r=0.814; εe :r=0.875, both P < 0.05) and a significant negative correlation with LA volumes (LAVmin: r=-0.817; LAVmax: r=-0.907; both P < 0.05).LVRS was positively correlated with εe (r=0.855, P < 0.05) and negatively correlated with LA volumes (LAVmin: r=-0.812; LAVmax: r=-0.860; both P < 0.05).(Figure 2).

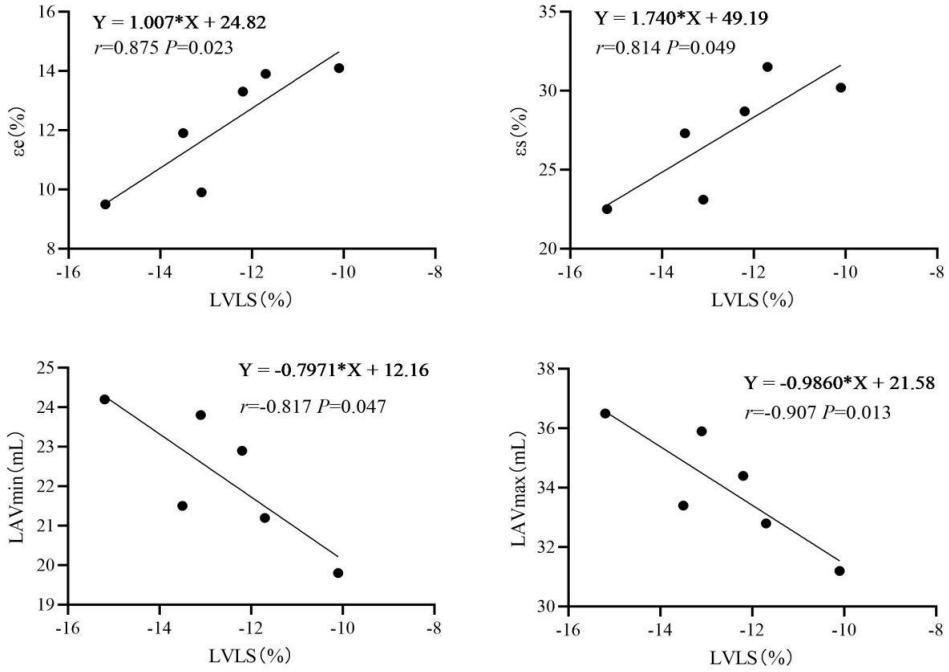

Figure 2: illustrates the correlation between left ventricular longitudinal strain (LVLS) and left atrial volume and strain.

4. Discussion

Clinically, Left Ventricular Ejection Fraction (LVEF) remains the standard metric for assessing systolic function. However, LVEF is inherently limited as it reflects only global volumetric changes, failing to capture intrinsic myocardial contractile properties or regional deformation abnormalities [9]. Myocardial strain, defined as the fractional change in myocardial length throughout the cardiac cycle, offers a superior measure of deformation capability that is independent of global cardiac translation and tethering effects [10].

In this study, we conducted a comprehensive comparative analysis of conventional functional parameters versus myocardial strain characteristics in HHD. Notably, conventional indices (LVEF, LVEDV, LVESV, LVSV, and their indexed values) remained comparable between the hypertension and control groups at matched time points. This suggests that overt volumetric remodeling is absent during the early stages of HHD. Conversely, myocardial strain parameters exhibited significantly higher sensitivity in detecting subclinical dysfunction. Our data demonstrated significant differences in LVLS, LVRS, and LVCS between the groups as early as 4 weeks post-surgery, persisting at 16 weeks. Mechanistically, these early functional impairments likely stem from pathological processes such as microvascular rarefaction and dysregulated calcium handling, which precede overt cardiomyocyte hypertrophy and interstitial fibrosis, ultimately compromising myocardial deformation capability [11]. These findings are concordant with Li et al. [12], who reported reduced multidirectional strain (LVLS, LVRS, LVCS) in HHD patients despite preserved conventional functional parameters, indicating that hypertensive remodeling is a global phenomenon affecting the myocardium diffusely rather than segmentally.

The LA modulates LV filling through three distinct phases: the reservoir phase (collecting pulmonary venous flow during LV systole), the conduit phase (passive transit during early diastole), and the

booster pump phase (active contraction during late diastole). These functions are correspondingly quantified by total strain (εs), passive strain (εe), and active strain（εa) [13].

Previous studies by Fung et al. [14]and Chen et al. [15] observed reduced strain parameters despite preserved LA volumes in hypertensive cohorts. In agreement with our findings, we observed significant reductions in εs and εe at 4 weeks, while LAVmax, LAVmin, and εa remained comparable to controls. However, our longitudinal assessment revealed that by 16 weeks, significant LA dilation (increased LAVmax and LAVmin) and a decline in εa became evident. This trajectory underscores that LA strain is a more sensitive marker of dysfunction than volumetric indices. Our results align with Li et al. [16], who noted that εa remains preserved in hypertensive patients without LVH but declines in those with LVH. We hypothesize that in the nascent stages of HHD, a compensatory mechanism augments LA contractile function εa to maintain adequate LV filling pressures and cardiac output against early diastolic dysfunction. As the disease progresses, worsening LV fibrosis and diastolic stiffness eventually overwhelm this compensation, leading to a significant decrease in LA compliance and a subsequent decline in contractile function [17].

Atrioventricular Coupling: The LA-LV InteractionFurther analysis of atrioventricular coupling revealed a robust correlation between LA and LV mechanics. Specifically, LVLS was positively correlated with εs and εe, while both LVRS and LVLS showed negative correlations with LA volumes (LAVmin, LAVmax). Additionally, LVRS was positively correlated with εe. These associations are consistent with findings by Fung et al. [14]. Moreover, we observed strong inverse correlations between LA volumes (LAVmin, LAVmax) and strain parameters (εs, εe), corroborating the results reported by Zhang et al. [18]. These correlations highlight the intrinsic mechanical coupling between the left atrium and ventricle, suggesting that LA dysfunction is not an isolated event but is closely linked to LV compliance and longitudinal mechanics.

5. Conclusion

In conclusion, myocardial strain parameters derived from CMR-FT allow for the assessment of structural and functional impairments of the left heart in early-stage HHD earlier, more sensitively, and more accurately than conventional cardiac functional parameters. Additionally, significant correlations exist between left atrial and left ventricular functional parameters. The advancement and application of myocardial strain analysis hold substantial value for further exploring the intrinsic link between myocardial mechanics and function. Furthermore, the detection of myocardial strain abnormalities is of significant clinical relevance for the subclinical diagnosis and evaluation of HHD, potentially guiding clinicians in formulating personalized therapeutic strategies for patients in the subclinical phase.

6. Limitations

Several limitations of this study warrant consideration. First, the sample size was relatively small; to some extent, this may have influenced the statistical power of the correlation analyses. However, the data exhibited low dispersion and high consistency, supporting the reliability of our findings. Second, all CMR data were acquired using a single vendor's scanner. Given the known inter-vendor and inter-software variability in myocardial strain quantification, results may vary across different platforms. Finally, this study was conducted exclusively in a preclinical model of HHD. While the porcine model is physiologically relevant, further validation in large-scale human cohorts is necessary to fully confirm the clinical applicability of these findings.

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Conflict of Interest:

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper

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