Research Square

Preprints are preliminary reports that have not undergone peer review.
They should not be considered conclusive, used to inform clinical practice,
or referenced by the media as validated information.

# Association between Left Ventricular Late Gadolinium Enhancement and Left Atrial Function in Hypertrophic Cardiomyopathy: A Cardiac MR-Feature Tracking Study

**Wenqi Liu**
The First Affiliated Hospital of Zhejiang Chinese Medical University (Zhejiang Provincial Hospital of Chinese Medicine)
**Yiyuan Gao**
The First Affiliated Hospital of Zhejiang Chinese Medical University (Zhejiang Provincial Hospital of Chinese Medicine)
**Qian Li**
The Second Affiliated Hospital, Zhejiang University School of Medicine
**Zhan Feng**
The First Affiliated Hospital, Zhejiang University School of Medicine
**Youfan Zhao**
The First Affiliated Hospital of Wenzhou Medical University
**Jingjing Shi**
The First Affiliated Hospital of Zhejiang Chinese Medical University (Zhejiang Provincial Hospital of Chinese Medicine)
**Wanzhen Li**
The First Affiliated Hospital of Zhejiang Chinese Medical University (Zhejiang Provincial Hospital of Chinese Medicine)
**Jie Lin**
The First Affiliated Hospital of Zhejiang Chinese Medical University (Zhejiang Provincial Hospital of Chinese Medicine)
**Zewen Qiao**
The First Affiliated Hospital of Zhejiang Chinese Medical University (Zhejiang Provincial Hospital of Chinese Medicine)
**Lin Lu**
Pujiang County Hospital of Traditional Chinese Medicine
**Houzhang Sun**
The First Affiliated Hospital of Wenzhou Medical University
**Wenbo Xiao**
The First Affiliated Hospital, Zhejiang University School of Medicine
**Maosheng Xu**
xums166@zcmu.edu.cn

The First Affiliated Hospital of Zhejiang Chinese Medical University (Zhejiang Provincial Hospital of Chinese Medicine)

**Research Article**

**Keywords:** Hypertrophic cardiomyopathy, cardiac magnetic resonance-feature tracking, left atrium, late gadolinium enhancement, strain

**Posted Date:** May 18th, 2026

**DOI:** https://doi.org/10.21203/rs.3.rs-9364182/v1

**License:** This work is licensed under a Creative Commons Attribution 4.0 International License. Read Full License

**Additional Declarations:** No competing interests reported.

## Abstract

# Background

Left atrial (LA) strain has been recognized as a sensitive indicator of LA dysfunction. While left ventricular (LV) late gadolinium enhancement (LGE) reflects myocardial fibrosis in hypertrophic cardiomyopathy (HCM), its association with LA function remains incompletely characterized. This study aimed to investigate the associations between LV LGE characteristics and LA phasic strain in patients with HCM using cardiac magnetic resonance-feature tracking (MR-FT).

# Methods

This retrospective, multicenter study included 431 patients with HCM (mean age 53.1 ± 14.0 years, 67.7% male) who underwent CMR between December 2023 and December 2024. LA reservoir, conduit, and booster strain (εs, εe, and εa) and corresponding strain rates (SRs, SRe, and SRa) were assessed using MR-FT. LV LGE presence, extent, and location were assessed on LGE images. Statistical analyses were performed using independent-samples t-test and sequential multivariable regression models with stepwise adjustment.

# Results

Significant differences in LA phasic strain parameters were observed across subgroups stratified by LGE presence, extent, and location (all $P < 0.05$). In multivariable regression analyses, LGE presence was independently associated with reduced LA εs (β = -3.590, $P = 0.022$) and εe (β = -1.898, $P = 0.046$) after full adjustment for demographic variables, traditional cardiovascular risk factors, and cardiac MR parameters. LGE extent was independently associated with reduced LA εs (β = -4.183, $P = 0.005$) and εe (β = -3.050, $P < 0.001$) after further adjustment for LGE location. No significant associations were observed between LGE location and LA strain parameters in the final adjusted model.

# Conclusions

LA phasic dysfunction in HCM is independently associated with LV LGE presence and extent, but not with LGE location. These findings indicate that overall fibrotic burden is more closely associated with LA functional impairment in HCM than its spatial distribution.

## Background

Hypertrophic cardiomyopathy (HCM) is a primary myocardial disorder characterized by left ventricular (LV) hypertrophy in the absence of abnormal loading conditions, and represents a major cause of sudden cardiac death (SCD) and heart failure in young and middle-aged individuals [1, 2]. Myocardial fibrosis is a key pathological feature of HCM that drives ventricular dysfunction and adverse clinical outcomes [3, 4]. Cardiac magnetic resonance (MR) has been recommended as an integral imaging modality in the contemporary evaluation of HCM, as it enables comprehensive assessment of cardiac morphology, function, and tissue characteristics [5]. In particular, late gadolinium enhancement (LGE) detected on cardiac MR provides reliable characterization of LV

myocardial fibrosis, and its presence, extent, and location have been widely adopted for prognostic evaluation and risk stratification in HCM [3, 6, 7]. However, the impact of LGE characteristics on myocardial functional abnormalities in patients with HCM remains incompletely understood.

The left atrium plays a pivotal role in modulating LV filling through its reservoir, conduit, and booster pump functions [8]. Left atrial (LA) dysfunction is prevalent in HCM and may occur before overt LA enlargement [9]. In this context, LA strain has gained increasing recognition as a more sensitive measure of atrial function than conventional volumetric indices [10]. Recently, cardiac MR-feature tracking (MR-FT) has emerged as a robust and reproducible technique for quantifying LA strain, providing incremental value for the functional assessment of HCM [11]. Given the close physiological interdependence between the left atrium and ventricle [12], LA strain may sensitively reflect the hemodynamic consequences of LV fibrosis. However, whether specific LV LGE characteristics, including presence, extent, and location, are differentially associated with LA phasic strain in HCM has not been systematically investigated in a large multicenter cohort.

Therefore, this study aimed to investigate the association between LV LGE characteristics and LA function using cardiac MR-FT in patients with HCM.

## Methods

# Study Population

This retrospective, multicenter study was conducted at four tertiary hospitals. The institutional review boards (IRB) of all participating centers approved this study, and the requirement for informed consent was waived due to the retrospective nature. Consecutive patients with a diagnosis of HCM who underwent cardiac MR between December 2023 and December 2024 were included. HCM was defined according to current guideline criteria as unexplained LV hypertrophy with an LV maximal wall thickness (LVMWT) ≥ 15 mm or ≥ 13 mm in those with a family history of HCM [2]. The exclusion criteria encompassed the following: 1) history of myocardial infarction, significant coronary artery disease (defined as ≥ 50% stenosis on coronary angiography or CT), or myocarditis; 2) atrial fibrillation; 3) prior septal reduction therapy, including septal myectomy and alcohol septal ablation; 4) LV hypertrophy secondary to other cardiovascular conditions, including but not limited to significant valvular heart disease, cardiac amyloidosis, or Fabry disease; and 5) inadequate image quality for cardiac MR-FT or LGE analysis. Eventually, a total of 431 patients were included in the final analysis and were subsequently stratified according to the presence of LV LGE (Fig. 1).

## Cardiac MR Acquisition

All cardiac MR examinations were performed on 1.5T MR scanners (Magnetom Avanto, Siemens Healthineers, Erlangen, Germany; Optima MR360 and Signa HD Excite, GE Healthcare, Chicago, IL, USA) or 3.0T MR scanners (Magnetom Vida, Siemens Healthineers, Erlangen, Germany; Discovery MR750, Signa Premier, and Signa Architect, GE Healthcare, Chicago, IL, USA; Ingenia Elition, Philips Healthcare, Best, the Netherlands) equipped with phased-array surface coils across the four centers. Cine images were acquired with a balanced steady-state free precession sequence during breath-holding and retrospective electrocardiographic gating. Standard long-axis views (two-, three-, and four-chamber) and contiguous short-axis slices were obtained to ensure complete coverage of both ventricles from base to apex. LGE imaging was performed 10–15 minutes after intravenous

administration of a gadolinium-based contrast agent (0.15–0.20 mmol/kg), using a phase-sensitive inversion recovery sequence acquired in matching short-axis planes. Detailed acquisition parameters are provided in **Table S1**.

## Cardiac MR Image Analysis

All cardiac MR analyses were performed using MR-FT with cvi42 software (cvi42, Version 6.3.1; Circle Cardiovascular Imaging, Calgary, AB, Canada) by 2 researchers (W.Q.L. and W.Z.L., with 3 and 5 years of experience in cardiac MR analysis, respectively) blinded to all clinical information. Biventricular volumetric and functional parameters, including end-diastolic volume (EDV), end-systolic volume (ESV), stroke volume (SV), ejection fraction (EF), and cardiac index (CI), were quantified from short-axis cine images. LV mass (LVM) was derived by manual delineation of endocardial and epicardial borders and was indexed to body surface area (BSA). LA volumes were measured using the biplane area–length method from two- and four-chamber long-axis cine images. Maximum LA volume (LAVmax), pre–atrial contraction LA volume (LAVpac), and minimum LA volume (LAVmin) were obtained at ventricular end-systole, at immediately before atrial contraction, and at late ventricular diastole after atrial contraction, respectively. Total, passive, and booster LA emptying fractions (LAEF) were calculated as follows [13]: 1) LAEF total = (LAVmax − LAVmin) / LAVmax × 100; 2) LAEF passive = (LAVmax − LAVpac) / LAVmax × 100; 3) LAEF booster = (LAVpac − LAVmin) / LAVpac × 100. All volumetric parameters were indexed to BSA.

## Cardiac MR-FT Analysis

LA myocardial deformation was quantified on two- and four-chamber cine images using the same post-processing software by the same 2 researchers without access to any clinical data. LA endocardial and epicardial borders were manually delineated at end-diastolic phase with the pulmonary veins and LA appendage excluded. After initialization, myocardial tracking was automatically propagated throughout the cardiac cycle based on voxel motion. The tracking performance was visually inspected in all frames, and initial contours were corrected if suboptimal tracking was identified. LA strain-time curves and strain rate-time curves were generated, and LA reservoir strain (εs) and peak positive strain rate (SRs), conduit strain (εe) and peak early negative strain rate (SRe), and booster strain (εa) and peak late negative strain rate (SRa) were calculated accordingly (Fig. 2). Endocardial borders of ventricles were delineated using a semi-automated threshold-based approach, followed by manual adjustment where necessary. Biventricular epicardial contours were manually traced with the interventricular septum excluded. Biventricular strain parameters, including global radial strain (GRS), global circumferential strain (GCS), and global longitudinal strain (GLS), were subsequently derived.

## LGE Quantification

Late gadolinium enhancement was assessed by the same 2 researchers on contiguous short-axis images covering the entire left ventricle. The presence and spatial distribution of LGE were visually evaluated, and the myocardial segments involved were identified according to the American Heart Association 17-segment model [14]. For quantitative analysis, endocardial and epicardial contours were manually delineated on each short-axis slice to define the total myocardial area. A region of interest was manually placed within visually normal myocardium without evidence of LGE, and myocardial areas with a mean signal intensity > 6 standard deviations

(SD) [15] of the reference region were defined as LGE. LGE extent was calculated by summing the enhanced myocardial areas across all short-axis slices and expressed as both absolute mass and as a percentage of total LV myocardial mass (LGE%).

For subgroup analyses, HCM patients with detectable LV LGE were classified into three groups according to LGE extent (LGE < 10%, 10–20%, and > 20%) [16]. In addition, LGE location was categorized according to septal involvement as isolated septal LGE and non-isolated septal LGE [17]. Because septal involvement is the most prevalent and clinically relevant LGE pattern in HCM, location was simplified into isolated septal versus non-isolated septal involvement to improve clinical interpretability and to ensure adequate subgroup size for analysis. Isolated septal LGE was defined as LGE confined to the interventricular septum only, and non-isolated septal LGE as involvement of any non-septal myocardial segment, regardless of concomitant septal involvement.

# Reproducibility

A random subset of 40 patients was reanalyzed by two observers (W.Q.L. and W.Z.L., with 3 years and 5 years of experience in cardiac MR analysis, respectively) to assess inter-observer reproducibility in LA strain and LGE measurements. For intra-observer reproducibility, the same observer (W.Q.L.) repeated LA strain measurements after an interval of at least one month blinded to the initial results.

# Statistical Analysis

All statistical analyses were conducted using SPSS (version 27.0, IBM Corp., Armonk, NY, USA) and GraphPad Prism (version 9.5, GraphPad Software, San Diego, CA, USA). Continuous variables are presented as mean ± SD or median (interquartile range) as appropriate. Categorical variables are presented as frequencies and percentages. Shapiro-Wilk test was used to assess the normality of continuous variables. Two-group comparisons were performed using the independent-samples t-test or Mann–Whitney U test. For multiple-group comparisons, one-way analysis of variance (ANOVA) with Bonferroni post hoc tests or the Kruskal–Wallis test with Dunn's post hoc test was applied according to data distribution. Categorical variables were compared using the $\chi^2$ test or Fisher's exact test. Multivariable linear regression analyses were performed to evaluate the associations between LV LGE and LA strain indices. LGE presence was analyzed in the full cohort, whereas analyses of LGE extent (< 10%, 10–20%, and > 20%) and LGE location (isolated septal LGE vs. non-isolated septal LGE) were restricted to patients with detectable LV LGE. Covariates were selected a priori based on clinical relevance and prior literature [5, 17]. All models were adjusted for demographic variables (including age and sex), traditional cardiovascular risk factors (including body mass index [BMI], hypertension, diabetes, current smoking, and family history of HCM or SCD), and cardiac MR parameters (including LVMWT, LVEF, LVEDV index [LVEDVi], and LVGLS). In addition, models assessing LGE extent were further adjusted for LGE location, and conversely, models assessing LGE location were additionally adjusted for LGE extent. Reproducibility was evaluated using intraclass correlation coefficients (ICCs) and Bland–Altman analysis. An ICC value > 0.80 was considered indicative of excellent reproducibility. A two-sided *P* value < 0.05 was considered statistically significant.

## Results

# Baseline Characteristics

The final analysis included a total of 431 patients with HCM, consisting of 292 males (67.7%) and 139 females (32.3%) with a mean age of 53.1 ± 14.0 years, and were divided into patients with LV LGE [LGE (+), n = 373, 86.5%] and without LV LGE [LGE (-), n = 58, 13.5%] cohorts. Among patients with LV LGE, 174 exhibited a LGE extent of < 10%, 117 had an extent of 10–20%, and 82 showed an extent > 20%. Based on LGE location, 152 patients demonstrated isolated septal LGE, and 221 non-isolated septal LGE. Clinical characteristics and cardiac MR measurements of HCM patients according to LV LGE presence, extent, and location are summarized in Table 1, **Table S2** and **Table S3**, respectively.

Table 1
Baseline Characteristics and Biventricular Cardiac MR Measurements According to LGE Presence

| **Age, years** | **All HCM patients (n = 431)** | **LGE (-) (n = 58)** | **LGE (+) (n = 373)** | ***P* value** |
|---|---|---|---|---|
| | **53.1 ± 14.0** | **53.6 ± 13.1** | **53.1 ± 14.2** | **0.796** |
| Male, n (%) | 292 (67.7) | 34 (58.6) | 258 (69.2) | 0.131 |
| Weight, kg | 69.6 ± 13.8 | 72.9 ± 18.2 | 69.1 ± 12.9 | 0.058 |
| Height, cm | 166.5 ± 8.0 | 167.0 ± 9.1 | 166.4 ± 7.8 | 0.586 |
| BMI, $kg/m^2$ | 25.0 ± 3.6 | 25.9 ± 4.7 | 24.8 ± 3.4 | **0.043** |
| BSA, $m^2$ | 1.7 ± 0.5 | 1.8 ± 0.4 | 1.7 ± 0.5 | 0.113 |
| SBP, mm Hg | 129.2 ± 18.2 | 134.0 ± 18.8 | 128.5 ± 18.1 | **0.034** |
| DBP, mm Hg | 75.9 ± 11.2 | 78.8 ± 11.6 | 75.5 ± 11.1 | **0.036** |
| FBG, mmol/L | 6.1 ± 2.0 | 5.9 ± 1.5 | 6.1 ± 2.1 | 0.448 |
| Hypertension, n (%) | 202 (46.9) | 34 (58.6) | 168 (45.0) | 0.054 |
| Diabetes, n (%) | 55 (12.8) | 5 (8.6) | 50 (13.4) | 0.310 |
| Current smoking, n (%) | 89 (20.6) | 11 (19.0) | 78 (20.9) | 0.733 |
| NYHA class II-IV, n (%) | 264 (61.3) | 37 (63.8) | 227 (60.9) | 0.669 |
| Family history of HCM, n (%) | 70 (16.2) | 9 (15.5) | 61 (16.4) | 0.872 |
| Family history of SCD, n (%) | 21 (4.9) | 4 (6.9) | 17 (4.6) | 0.659 |
| LVMWT, mm | 19.1 ± 3.9 | 16.4 ± 1.9 | 19.5 ± 4.0 | **< 0.001** |
| LVOT obstruction, n (%) | 103 (23.9) | 14 (24.1) | 89 (23.9) | 0.963 |
| LVMi, $g/m^2$ | 92.0 ± 30.2 | 79.5 ± 18.9 | 94.0 ± 31.2 | **< 0.001** |
| LVEF, % | 59.1 ± 10.8 | 63.0 ± 11.1 | 58.5 ± 10.7 | **0.003** |
| LVEDVi, $mL/m^2$ | 81.7 ± 17.5 | 81.6 ± 16.4 | 81.7 ± 17.6 | 0.956 |
| LVESVi, $mL/m^2$ | 34.3 ± 14.5 | 31.3 ± 15.3 | 34.8 ± 14.3 | 0.093 |
| LVSVi, $mL/m^2$ | 47.4 ± 11.7 | 50.3 ± 10.4 | 46.9 ± 11.8 | **0.045** |

Data were presented as means ± standard deviations or n (%), *P* values were calculated using the independent samples t-test comparing the LGE (-) and LGE (+) subgroups, and bold indicates significant *P* values. BMI = body mass index; BSA = body surface area; MR = magnetic resonance; DBP = diastolic blood pressure; FBG = fasting blood glucose; HCM = hypertrophic cardiomyopathy; LGE = late gadolinium enhancement; LVEDVi = left ventricular end-diastolic volume index; LVEF = left ventricular ejection fraction; LVESVi = left ventricular end-systolic volume index; LVGCS = left ventricular global circumferential strain; LVGLS = left ventricular global longitudinal strain; LVGRS = left ventricular global radial strain; LVMi = left ventricular mass index; LVSVi = left ventricular stroke volume index; LVMWT = left ventricular maximal wall thickness; LVOT = left ventricular outflow tract; NYHA = New York Heart Association; SBP = systolic blood pressure; SCD = sudden cardiac death.

| Age, years | All HCM patients (n = 431) | LGE (-) (n = 58) | LGE (+) (n = 373) | *P* value |
|---|---|---|---|---|
| | **53.1 ± 14.0** | **53.6 ± 13.1** | **53.1 ± 14.2** | **0.796** |
| LVGRS, % | 20.8 ± 8.3 | 24.9 ± 9.7 | 20.2 ± 7.9 | **< 0.001** |
| LVGCS, % | -13.1 ± 3.8 | -15.1 ± 3.8 | -12.8 ± 3.0 | **< 0.001** |
| LVGLS, % | -10.2 ± 3.5 | -12.7 ± 3.0 | -9.9 ± 3.4 | **< 0.001** |

Data were presented as means ± standard deviations or n (%), *P* values were calculated using the independent samples t-test comparing the LGE (-) and LGE (+) subgroups, and bold indicates significant *P* values. BMI = body mass index; BSA = body surface area; MR = magnetic resonance; DBP = diastolic blood pressure; FBG = fasting blood glucose; HCM = hypertrophic cardiomyopathy; LGE = late gadolinium enhancement; LVEDVi = left ventricular end-diastolic volume index; LVEF = left ventricular ejection fraction; LVESVi = left ventricular end-systolic volume index; LVGCS = left ventricular global circumferential strain; LVGLS = left ventricular global longitudinal strain; LVGRS = left ventricular global radial strain; LVMi = left ventricular mass index; LVSVi = left ventricular stroke volume index; LVMWT = left ventricular maximal wall thickness; LVOT = left ventricular outflow tract; NYHA = New York Heart Association; SBP = systolic blood pressure; SCD = sudden cardiac death.

Compared with patients without LV LGE, those with LGE showed lower BMI, systolic blood pressure (SBP), diastolic blood pressure (DBP), LVEF, left ventricular stroke volume index (LVSVi), and LV strain parameters (all $P < 0.05$), whereas LVMWT and left ventricular mass index (LVMi) were significantly greater in the LGE (+) group ($P < 0.001$). When further stratified by LGE extent, patients with higher LGE extent (LGE 10–20% and LGE > 20%) exhibited lower LVEF and LV strain values, as well as higher LVMWT, LVMi, and LVESV index (LVESVi) compared with LGE < 10% subgroup (all $P < 0.05$). Patients with LGE > 20% had a significantly higher prevalence of diabetes compared with those in the LGE < 10% and LGE 10–20% subgroups ($P < 0.05$ for both). According to LGE location, patients with non-isolated septal LGE demonstrated higher SBP, LVMWT, LVMi, and LVEDVi (all $P < 0.05$), but lower LV strain parameters compared with those with isolated septal LGE (all $P < 0.05$).

# LA Parameters

LA volumes, LAEFs, and LA strain parameters stratified by LGE presence are summarized in Table 2. Overall, patients with LV LGE exhibited significantly larger LA volumes and lower LAEFs than those without LV LGE (all $P < 0.05$). As detailed in **Table S4**, patients with LGE > 20% demonstrated more pronounced LA remodeling and functional impairment, with significantly higher LAVpac index (LAVpaci), alongside lower LAEF total, LAEF passive, and LAEF booster, compared with those with LGE < 10% (all $P < 0.05$). Similarly, patients with LGE 10–20% demonstrated significantly higher LAVmax index (LAVmaxi), LAVpaci, and LAVmin index (LAVmini), as well as lower LAEF total and LAEF passive, than the LGE < 10% group (all $P < 0.05$). When stratified by LGE location, patients with non-isolated septal LGE exhibited higher LAVmaxi and LAVpaci, and lower LAEF passive compared with those with isolated septal LGE (all $P < 0.05$), whereas LAVmini and the remaining LAEFs were comparable between the two subgroups (**Table S5**).

Table 2
LA parameters According to LGE Presence

| Volume parameters | All HCM patients (n = 431) | LGE (-) (n = 58) | LGE (+) (n = 373) | *P* value |
|---|---|---|---|---|
| LAVmaxi, mL/m$^2$ | 47.4 ± 20.2 | 42.3 ± 15.1 | 48.3 ± 20.8 | **0.040** |
| LAVpaci, mL/m$^2$ | 38.9 ± 18.7 | 32.8 ± 13.0 | 39.9 ± 19.3 | **0.008** |
| LAVmini, mL/m$^2$ | 30.2 ± 20.4 | 23.6 ± 13.2 | 31.2 ± 21.1 | **0.009** |
| **Emptying fractions** | | | | |
| LAEF total, % | 47.4 ± 12.7 | 53.9 ± 9.2 | 46.3 ± 12.8 | **< 0.001** |
| LAEF passive, % | 23.2 ± 9.4 | 27.3 ± 9.0 | 22.6 ± 9.3 | **< 0.001** |
| LAEF booster, % | 31.8 ± 11.8 | 36.5 ± 9.9 | 31.0 ± 11.9 | **< 0.001** |
| **Strain parameters** | | | | |
| LA εs, % | 25.4 ± 10.3 | 30.8 ± 9.1 | 24.5 ± 10.2 | **< 0.001** |
| LA εe, % | 11.8 ± 6.1 | 14.9 ± 6.2 | 11.4 ± 5.9 | **< 0.001** |
| LA εa, % | 13.5 ± 6.0 | 15.9 ± 6.1 | 13.2 ± 6.0 | **0.002** |
| LA SRs, $s^{-1}$ | 1.3 ± 0.6 | 1.5 ± 0.5 | 1.2 ± 0.5 | **< 0.001** |
| LA SRe, $s^{-1}$ | -1.2 ± 0.6 | -1.4 ± 0.7 | -1.1 ± 0.6 | **< 0.001** |
| LA SRa, $s^{-1}$ | -1.5 ± 0.7 | -1.8 ± 0.8 | -1.4 ± 0.7 | **< 0.001** |

Data were presented as means ± standard deviations. *P* values were calculated using the independent samples t-test comparing the LGE (-) and LGE (+) subgroups, and bold indicates significant *P* values. εa = booster strain; εe = conduit strain; εs = reservoir strain; LA = left atrial; LAEF = left atrial emptying fraction; LAVmaxi = left atrial maximum volume index; LAVmini = left atrial minimum volume index; LAVpaci = left pre-atrial contraction volume index; HCM = hypertrophic cardiomyopathy; LGE = late gadolinium enhancement; SRa = peak late negative strain rate; SRe = peak early negative strain rate; SRs = peak positive strain rate.

Figure 3 illustrates the differences in LA strains across LV LGE subgroups. Patients with LV LGE had significantly lower LA εs, εe, and εa values compared with those without LGE (all $P < 0.05$). When stratified by LGE extent, patients with LGE < 10% showed significantly higher LA εs, εe, and εa compared with LGE > 20% (all $P < 0.001$). Although LA εs showed a graded reduction with increasing LV fibrosis burden (all $P < 0.05$), there was no significant difference observed in LA εe between the LGE 10–20% and LGE > 20% subgroups, nor in LA εa between LGE < 10% and LGE 10–20% ($P = 0.052$ and 0.129, respectively). When stratified by LGE location, patients with non-isolated septal LGE exhibited significantly lower LA εs and εa compared with those with isolated septal LGE (both $P < 0.05$). Representative cases illustrating LA strain measurements and corresponding time–strain curves stratified by LGE extent and LGE location are presented in Figs. 4 and 5, respectively.

# Associations between LA strains and LV LGE Characteristics

In multivariable linear regression analysis, LV LGE presence was significantly associated with LA strain parameters across sequential adjustment models (Table 3). In Model 1, adjusted for demographic data (including age and sex), LV LGE presence was independently associated with LA εs (β = -6.213, *P* < 0.001), LA εe (β = -3.579, *P* < 0.001), and LA εa (β = -2.631, *P* = 0.002). After additional adjustment for cardiovascular risk factors in Model 2 (including BMI, hypertension, diabetes, current smoking, and family history of HCM or SCD), the associations remained significant for all three LA strain components, including LA εs (β = -7.158, *P* < 0.001), LA εe (β = -3.719, *P* < 0.001), and LA εa (β = -3.434, *P* = 0.003). In the fully adjusted Model 3, which further included cardiac MR parameters (including LVMWT, LVEF, LVEDVi, and LVGLS), LV LGE presence remained independently associated with LA εs (β = -3.590, *P* = 0.022) and LA εe (β = -1.898, *P* = 0.046). In contrast, no independent association was observed between LV LGE presence and LA εa after full adjustment (β = -1.687, *P* = 0.121).

Table 3
Multivariable Regression Analysis for Association between LA strain and LGE Presence

| | **Model 1** | | **Model 2** | | **Model 3** | |
|---|---|---|---|---|---|---|
| | **β Coefficient (95% CI)** | ***P* value** | **β Coefficient (95% CI)** | ***P* value** | **β Coefficient (95% CI)** | ***P* value** |
| **LA εs, %** | -6.213 (-8.854, -3.572) | **< 0.001** | -7.158 (-10.569, -3.747) | **< 0.001** | -3.590 (-6.666, -0.514) | **0.022** |
| **LA εe, %** | -3.579 (-5.108, -2.050) | **< 0.001** | -3.719 (-5.641, -1.797) | **< 0.001** | -1.898 (-3.760, -0.037) | **0.046** |
| **LA εa, %** | -2.631 (-4.268, -0.994) | **0.002** | -3.434 (-5.690, -1.178) | **0.003** | -1.687 (-3.820, 0.446) | 0.121 |

The β coefficient expresses the mean difference in each LA strain parameters and LGE presence after adjustment for the covariates in each model. Bold indicates significant *P* values. Model 1 was adjusted for age and sex. Model 2 was adjusted for age, sex, BMI, hypertension, diabetes, current smoking, and family history of HCM and SCD. Model 3 was adjusted for age, sex, BMI, hypertension, diabetes, current smoking, family history of HCM and SCD, LVMWT, LVEF, LVEDVi, and LVGLS. BMI = body mass index; CI = confidence interval; εa = booster strain; εe = conduit strain; εs = reservoir strain; HCM = hypertrophic cardiomyopathy; LA = left atrial; LGE = late gadolinium enhancement; LVEDVi = left ventricular end-diastolic volume index; LVEF = left ventricular ejection fraction; LVGLS = left ventricular global longitudinal strain; LVMWT = left ventricular maximal wall thickness; SCD = sudden cardiac death.

Table 4 demonstrated that greater LGE extent was significantly associated with LA strain parameters. After adjustment for demographic data (Model 1), LGE extent was associated with LA εs (β = -5.096, *P* < 0.001), LA εe (β = -2.857, *P* < 0.001), and LA εa (β = -2.240, *P* < 0.001). These associations remained robust after further adjustment for cardiovascular risk factors in Model 2, with significant coefficients for LA εs (β = -7.611, *P* < 0.001), LA εe (β = -4.484, *P* < 0.001), and LA εa (β = -3.125, *P* < 0.001). With further adjustment for cardiac MR parameters in Model 3, significant associations persisted for LA εs (β = -4.208, *P* = 0.004) and LA εe (β = -3.070, *P* < 0.001), whereas the association with LA εa was no longer significant. Moreover, after further adjustment for LGE location in Model 4, the associations for LA εs (β = -4.183, *P* = 0.005) and LA εe (β = -3.050, *P* < 0.001) remained stable, while LA εa remained non-significant.

Table 4
Multivariable Regression Analysis for Association between LA strain and LGE Extent

| | **Model 1** | | **Model 2** | | **Model 3** | | **Model 4** | |
|---|---|---|---|---|---|---|---|---|
| | **β Coefficient (95% CI)** | ***P* value** | **β Coefficient (95% CI)** | ***P* value** | **β Coefficient (95% CI)** | ***P* value** | **β Coefficient (95% CI)** | ***P* value** |
| **LA εs, %** | -5.096 (-7.099, -3.093) | **< 0.001** | -7.611 (-10.374, -4.847) | **< 0.001** | -4.208 (-7.038, -1.378) | **0.004** | -4.183 (-7.064, -1.302) | **0.005** |
| **LA εe, %** | -2.857 (-3.996, -1.718) | **< 0.001** | -4.484 (-6.017, -2.951) | **< 0.001** | -3.070 (-4.732, -1.409) | **< 0.001** | -3.050 (-4.741, -1.358) | **< 0.001** |
| **LA εa, %** | -2.240 (-3.473, -1.007) | **< 0.001** | -3.125 (-4.877, -1.374) | **< 0.001** | -1.137 (-2.941, 0.667) | 0.215 | -1.132 (-2.968, 0.705) | 0.225 |

The β coefficient expresses the mean difference in each LA strain parameters and LGE extent after adjustment for the covariates in each model. Bold indicates significant *P* values. Model 1 was adjusted for age and sex. Model 2 was adjusted for age, sex, BMI, hypertension, diabetes, current smoking, and family history of HCM and SCD. Model 3 was adjusted for age, sex, BMI, hypertension, diabetes, current smoking, family history of HCM and SCD, LVMWT, LVEF, LVEDVi, and LVGLS. Model 4 was adjusted for age, sex, BMI, hypertension, diabetes, current smoking, family history of HCM and SCD, LVMWT, LVEF, LVEDVi, LVGLS, and LGE location. BMI = body mass index; CI = confidence interval; εa = booster strain; εe = conduit strain; εs = reservoir strain; HCM = hypertrophic cardiomyopathy; LA = left atrial; LGE = late gadolinium enhancement; LVEDVi = left ventricular end-diastolic volume index; LVEF = left ventricular ejection fraction; LVGLS = left ventricular global longitudinal strain; LVMWT = left ventricular maximal wall thickness; SCD = sudden cardiac death.

Regarding LGE location, associations with LA εs (β = -2.157, *P* = 0.035) and εa (β = -1.233, *P* = 0.047) were significant in Model 1 after adjustment for demographic data, whereas no significant association was observed for LA εe (Table 5). However, after further adjustment for cardiovascular risk factors, cardiac MR parameters, and LGE extent, none of the associations between LGE location and LA strain parameters remained statistically significant.

Table 5
Multivariable Regression Analysis for Association between LA strain and LGE Location

| | Model 1 | | Model 2 | | Model 3 | | Model 4 | |
|---|---|---|---|---|---|---|---|---|
| | **β Coefficient (95% CI)** | ***P* value** | **β Coefficient (95% CI)** | ***P* value** | **β Coefficient (95% CI)** | ***P* value** | **β Coefficient (95% CI)** | ***P* value** |
| **LA εs, %** | -2.157 (-4.166, -0.148) | **0.035** | -2.701 (-5.434, 0.033) | 0.053 | -0.744 (-3.260, 1.772) | 0.560 | -0.127 (-2.624, 2.369) | 0.920 |
| **LA εe, %** | -0.926 (-2.070, 0.218) | 0.112 | -1.477 (-3.009, 0.056) | 0.059 | -0.554 (-2.051, 0.943) | 0.466 | -0.105 (-1.570, 1.361) | 0.888 |
| **LA εa, %** | -1.233 (-2.451, -0.014) | **0.047** | -1.225 (-2.886, 0.436) | 0.147 | -0.192 (-1.762, 1.379) | 0.810 | -0.025 (-1.616, 1.567) | 0.975 |

The β coefficient expresses the mean difference in each LA strain parameters and LGE location after adjustment for the covariates in each model. Bold indicates significant *P* values. Model 1 was adjusted for age and sex. Model 2 was adjusted for age, sex, BMI, hypertension, diabetes, current smoking, and family history of HCM and SCD. Model 3 was adjusted for age, sex, BMI, hypertension, diabetes, current smoking, family history of HCM and SCD, LVMWT, LVEF, LVEDVi, and LVGLS. Model 4 was adjusted for age, sex, BMI, hypertension, diabetes, current smoking, family history of HCM and SCD, LVMWT, LVEF, LVEDVi, LVGLS, and LGE extent. BMI = body mass index; CI = confidence interval; εa = booster strain; εe = conduit strain; εs = reservoir strain; HCM = hypertrophic cardiomyopathy; LA = left atrial; LGE = late gadolinium enhancement; LVEDVi = left ventricular end-diastolic volume index; LVEF = left ventricular ejection fraction; LVGLS = left ventricular global longitudinal strain; LVMWT = left ventricular maximal wall thickness; SCD = sudden cardiac death.

# Reproducibility

Intra- and inter-observer reproducibility of LA strain and LGE measurements is summarized in **Table S6**. Excellent agreement was observed for both intra- and inter-observer assessments with all ICCs > 0.939. Bland–Altman plots further demonstrated excellent reproducibility with narrow limits of agreement (**Figures S1–S2**).

# Discussion

In this large-scale, multicenter study, we comprehensively investigated the associations between LA phasic strain and LV LGE characterized by presence, extent, and location in patients with HCM. Our results demonstrated that both LGE presence and extent were independently associated with impaired LA strain, with consistent associations observed for LA εs and εe, whereas the associations for LA εa were no longer significant after full adjustment. In contrast, no independent association between LGE location and LA strain parameters was retained in adjusted models. These findings suggest that the quantitative burden of LV myocardial fibrosis, rather than its spatial location, is of greater relevance to LA mechanical impairment in HCM.

LV LGE detected on cardiac MR is widely regarded as a surrogate marker of replacement myocardial fibrosis in HCM, reflecting accumulated and irreversible myocardial injury [18]. Substantial evidence has demonstrated that, regardless of LGE location, both the presence and extent of LGE are closely associated with adverse clinical outcomes in HCM, including ventricular arrhythmias, sudden cardiac death, and heart failure progression, underscoring its central role in contemporary risk stratification [15, 19]. From a pathophysiological perspective, myocardial fibrosis impairs LV diastolic function with reduced relaxation and compliance, resulting in elevated

filling pressures and adverse atrial hemodynamics, which can be sensitively captured by LA strain [20, 21]. Accordingly, investigation of the association between LV LGE and LA strain may offer insights into the underlying pathophysiology of HCM.

In the present study, LGE presence was independently associated with lower LA εs and εe, indicating impaired LA reservoir and conduit function in patients with LV myocardial fibrosis detectable by cardiac MR examination. This observation is consistent with a previous cardiac MR-FT study [22], and supports the concept that LA dysfunction represents an integrated downstream manifestation of LV myocardial pathology, rather than an isolated atrial abnormality [23]. Pathophysiologically, both LA reservoir and conduit function are critically dependent on LV diastolic compliance [24]. Impairment of LV compliance secondary to myocardial fibrosis elevates filling pressures and attenuates early diastolic suction, reducing the atrioventricular pressure gradient that facilitates passive LA emptying [8]. Impaired longitudinal relaxation also limits atrial expansion during systole, compromising reservoir function [25]. Accordingly, the presence of myocardial fibrosis may be associated with reduced LA εs and εe in HCM, highlighting LA strain as a sensitive marker of hemodynamic consequences of ventricular fibrosis.

Evidence on the relationship between LV LGE extent and LA dysfunction in HCM remains conflicting. Specifically, a case–control study of 79 HCM patients and 20 controls reported that LA remodeling and dysfunction were determined by LVMi rather than LV LGE extent [26]. By contrast, Yu et al. [27] investigated 45 HCM patients with concomitant diabetes and found that LGE extent was independently associated with impaired LA strain. Drawing on a larger cohort of HCM, the present study adds evidence that greater LGE extent was independently associated with reduced LA εs and εe in the fully adjusted model. As a marker of cumulative replacement fibrosis, LGE extent reflects the degree of chronic ventricular remodeling in HCM [28], which may amplify its downstream impact on LA reservoir and conduit function and account for the closer relationship observed between LGE and LA εs and εe in this study. LA booster pump function, by contrast, reflects intrinsic atrial contractility and is dually modulated by loading conditions and compensatory mechanisms [29], which may explain the attenuation of its association with LGE extent after full adjustment. This pathological pattern in passive and active atrial phases may therefore provide insight into the underlying pathophysiological states of the left atrium in HCM and merits longitudinal evaluation as a marker of disease progression.

In the present study, LV LGE location, categorized as isolated septal LGE and non-isolated septal LGE, was not independently associated with LA strains after adjustment for demographic variables, traditional cardiovascular risk factors, cardiac MR parameters, and LGE extent. Our findings have provided evidence that the association between LGE location and LA dysfunction may be more influenced by the overall burden of LV myocardial fibrosis rather than specific regional involvement [30]. Consistent with this notion, LGE location has been reported as not a determinant of adverse outcomes in HCM [15], and is predominantly associated with regional rather than global LV mechanical impairment [31]. In addition, a previous study has observed similar impairment of mitral annular motion in both septal and lateral wall, regardless of regional LGE involvement [32]. These findings may explain the absence of association between LGE location and LA strain in our study, as LA strain primarily reflects overall LV hemodynamics and mitral annular plane motion rather than segmental myocardial deformation [33]. Intriguingly, a divergent pattern has been observed in acute myocarditis (AM), where septal LGE—but not LGE extent—was independently associated with impaired LA reservoir and conduit function [34]. This distinction is likely explained by the underlying pathology. In AM, LGE predominantly reflects active inflammation and edema [35], and acute septal injury may induce abrupt and disproportionate alterations in LV hemodynamics [34]. In

contrast, the chronic replacement fibrosis characteristic of HCM permits ventricular adaptation and compensatory remodeling, such that global functional impairment is more closely related to the overall burden of fibrosis rather than its specific anatomical distribution [36]. This contrast reinforces the plausibility that in a chronic, compensated disease such as HCM, LGE location alone does not appear to determine LA dysfunction.

Overall, our results support the view that LA phasic strain captures the functional consequences of LV myocardial fibrosis in HCM. Combined assessment of LA strain and LV fibrosis may therefore facilitate more comprehensive phenotypic characterization and may help refine imaging-based evaluation of disease severity.

# Limitations

Several limitations of the present study should be acknowledged. First, although this was a multicenter study, all patients were enrolled from tertiary referral hospitals with exclusion of atrial fibrillation, which may induce potential patient selection bias and limit the generalizability of the findings. Second, echocardiographic parameters regarding mitral regurgitation and LVOT obstruction were not comprehensively available across participating centers, and their potential influence on LA function requires further confirmation. Third, cardiac MR examinations were performed using scanners with different field strengths (1.5T and 3.0T) and vendors across all centers, potentially affecting LA MR-FT and LGE analyses. However, prior studies have demonstrated a limited influence of field strength and vendor on these measurements [37–39], lending support to the robustness of our results. Finally, this study was based on a retrospective, cross-sectional analysis, and future longitudinal studies investigating the temporal relationship between LGE progression and LA dysfunction in HCM are warranted.

## Conclusions

In patients with HCM, LV LGE is associated with impaired LA reservoir and conduit function with independent contributions from LGE presence and extent, rather than from LGE location. These findings suggest that LV fibrotic burden is more closely associated with LA mechanical impairment than the spatial distribution of fibrosis and provide further insight into atrioventricular interaction in HCM.

## Abbreviations

εa, booster strain

εe, conduit strain

εs, reservoir strain

HCM, hypertrophic cardiomyopathy

LA, left atrium

LAEF, left atrial emptying fraction

LAVmaxi, left atrial maximum volume index

LAVmini, left atrial minimum volume index

LGE, late gadolinium enhancement

LV, left ventricle

LVEDVi, left ventricular end-diastolic volume index

LVEF, left ventricular ejection fraction

LVESVi, left ventricular end-systolic volume index

LVGCS, left ventricular global circumferential strain

LVGLS, left ventricular global longitudinal strain

LVGRS, left ventricular global radial strain

LVM, left ventricular mass

LVMWT, left ventricular maximal wall thickness

LVOT, left ventricular outflow tract

LVSVi, left ventricular stroke volume index

MR-FT, magnetic resonance-feature tracking

SR, strain rate

SRa, late diastolic strain rate

SRe, early diastolic strain rate

SRs, systolic strain rate

# Declarations

### Ethics Approval and Consent to Participate

This retrospective study was approved by the Ethics Committee of the The First Affiliated Hospital of Zhejiang Chinese Medical University (2022-KLS-207-01) and was conducted in accordance with the ethical standards of the Declaration of Helsinki. The requirement for informed consent was waived due to the retrospective nature of the study.

### Data Availability

Data analyzed in the current study are not publicly available because of restrictions from institutional agreements and privacy policies across the participating hospitals in this multicenter collaboration. Data may be available from the corresponding author on reasonable request and subject to approval from the relevant participating institutions.

**Competing Interests**

The authors declare that they have no competing interests.

**Funding**

The study has received funding by the National Natural Science Foundation of China (No. 81971600), the “Pioneer” and “Leading Goose” R&D Program of Zhejiang (No. 2022C03046), Science and Technology Plan of the Health Industry in Zhejiang Province (No. 2025HY1377), and Jinhua Public Welfare Technology Application Research Project (No. 2025-4-274).

**Author Contributions**

W.Q.L. and Y.Y.G. completed the statistical analyses, prepared tables & figures and wrote the main manuscript text; J.J.S., W.Z.L., Q.L., Y.F.Z., Z.W.Q., and L.L. collected the data; H.Z.S., Z.F. and J.L. prepared tables & figures; M.S.X. was responsible for project administration; W.B.X. and M.S.X. designed the study, reviewed and edited the manuscript. All authors read and approved the final manuscript and agree to be personally accountable for their contributions.

**Acknowledgements**

Not applicable.

**Consent for Publication**

Not applicable.

# References

1. Canepa M, Fumagalli C, Tini G, et al. Temporal Trend of Age at Diagnosis in Hypertrophic Cardiomyopathy: An Analysis of the International Sarcomeric Human Cardiomyopathy Registry. Circ Heart Fail. 2020;13(9):e007230.
2. Ommen SR, Ho CY, Asif IM, et al. 2024 AHA/ACC/AMSSM/HRS/PACES/SCMR Guideline for the Management of Hypertrophic Cardiomyopathy: A Report of the American Heart Association/American College of Cardiology Joint Committee on Clinical Practice Guidelines. Circulation. 2024;149(23):e1239–311.
3. Chan RH, van der Wal L, Liberato G, et al. Myocardial Scarring and Sudden Cardiac Death in Young Patients With Hypertrophic Cardiomyopathy: A Multicenter Cohort Study. JAMA Cardiol. 2024;9(11):1001–8.
4. Argirò A, Parikh V, Jurcut R, et al. Hypertrophic cardiomyopathy. Nat Rev Dis Primer. 2025;11(1):58.
5. Zhao K, Zhu Y, Chen X, et al. Machine Learning in Hypertrophic Cardiomyopathy: Nonlinear Model From Clinical and CMR Features Predicting Cardiovascular Events. JACC Cardiovasc Imaging. 2024;17(8):880–93.
6. Greulich S, Seitz A, Herter D, et al. Long-term risk of sudden cardiac death in hypertrophic cardiomyopathy: a cardiac magnetic resonance outcome study. Eur Heart J Cardiovasc Imaging. 2021;22(7):732–41.
7. Li X, Lai L, Luo R, et al. The Clinical Prognosis of Presence and Location of Late Gadolinium Enhancement by Cardiac Magnetic Resonance Imaging in Patients with Hypertrophic Cardiomyopathy: a Single-Center Cohort Study. J Cardiovasc Transl Res. 2021;14(5):1001–16.

8. Aronson D, Sliman H, Abadi S, et al. Conduit Flow Compensates for Impaired Left Atrial Passive and Booster Functions in Advanced Diastolic Dysfunction. Circ Cardiovasc Imaging. 2024;17(5):e016276.
9. Yang Y, Yin G, Jiang Y, Song L, Zhao S, Lu M. Quantification of left atrial function in patients with non-obstructive hypertrophic cardiomyopathy by cardiovascular magnetic resonance feature tracking imaging: a feasibility and reproducibility study. J Cardiovasc Magn Reson Off J Soc Cardiovasc Magn Reson. 2020;22(1):1.
10. Chen L, Qiu B, Abdu FA, et al. Prognostic Value of Strain by Tissue Tracking Cardiac Magnetic Resonance in Myocardial Infarction With Nonobstructive Coronary Arteries. J Am Heart Assoc. 2025;14(8):e039395.
11. Chahine Y, Chamoun N, Kassar A, Bockus L, Macheret F, Akoum N. Atrial fibrillation substrate and impaired left atrial function: a cardiac MRI study. Eur Eur Pacing Arrhythm Card Electrophysiol J Work Groups Card Pacing Arrhythm Card Cell Electrophysiol Eur Soc Cardiol. 2024;26(11):euae258.
12. Qin S, Zhang L, Ji M, et al. Clinical Utility of Atrioventricular Coupling Index in Cardiovascular Disease. J Am Heart Assoc. 2025;14(11):e041392.
13. Qian WL, Yang ZG, Shi R, et al. Left atrioventricular interaction and impaired left atrial phasic function in type 2 diabetes mellitus patients with or without anemia: a cardiac magnetic resonance study. Cardiovasc Diabetol. 2023;22(1):178.
14. Cerqueira MD, Weissman NJ, Dilsizian V, et al. Standardized myocardial segmentation and nomenclature for tomographic imaging of the heart. A statement for healthcare professionals from the Cardiac Imaging Committee of the Council on Clinical Cardiology of the American Heart Association. Circulation. 2002;105(4):539–42.
15. Liu J, Zhao S, Yu S, et al. Patterns of Replacement Fibrosis in Hypertrophic Cardiomyopathy. Radiology. 2022;302(2):298–306.
16. Kowallick JT, Silva Vieira M, Kutty S, et al. Left Atrial Performance in the Course of Hypertrophic Cardiomyopathy: Relation to Left Ventricular Hypertrophy and Fibrosis. Invest Radiol. 2017;52(3):177–85.
17. Halliday BP, Baksi AJ, Gulati A, et al. Outcome in Dilated Cardiomyopathy Related to the Extent, Location, and Pattern of Late Gadolinium Enhancement. JACC Cardiovasc Imaging. 2019;12(8 Pt 2):1645–55.
18. Aguiar Rosa S, Thomas B, Fiarresga A, et al. The Impact of Ischemia Assessed by Magnetic Resonance on Functional, Arrhythmic, and Imaging Features of Hypertrophic Cardiomyopathy. Front Cardiovasc Med. 2021;8:761860.
19. Kiaos A, Daskalopoulos GN, Kamperidis V, Ziakas A, Efthimiadis G, Karamitsos TD. Quantitative Late Gadolinium Enhancement Cardiac Magnetic Resonance and Sudden Death in Hypertrophic Cardiomyopathy: A Meta-Analysis. JACC Cardiovasc Imaging. 2024;17(5):489–97.
20. Ommen SR, Mital S, Burke MA, et al. 2020 AHA/ACC Guideline for the Diagnosis and Treatment of Patients With Hypertrophic Cardiomyopathy: Executive Summary: A Report of the American College of Cardiology/American Heart Association Joint Committee on Clinical Practice Guidelines. Circulation. 2020;142(25):e533–57.
21. Ferkh A, Clark A, Thomas L. Left atrial phasic function: physiology, clinical assessment and prognostic value. Heart Br Card Soc. 2023;109(22):1661–9.
22. Ozden O, Unlu S, Kilic DI, et al. [The association between cardiac mr feature tracking strain and myocardial late gadolinium enhancement in patients with hypertrophic cardiomyopathy]. Kardiologiia. 2023;63(2):52–8.

23. Iannaccone G, Graziani F, Del Buono MG, et al. Left atrial strain analysis improves left ventricular filling pressures non-invasive estimation in the acute phase of Takotsubo syndrome. Eur Heart J Cardiovasc Imaging. 2023;24(6):699–707.
24. Hopman LHGA, Mulder MJ, van der Laan AM, et al. Impaired left atrial reservoir and conduit strain in patients with atrial fibrillation and extensive left atrial fibrosis. J Cardiovasc Magn Reson Off J Soc Cardiovasc Magn Reson. 2021;23(1):131.
25. Barbier P, Solomon SB, Schiller NB, Glantz SA. Left atrial relaxation and left ventricular systolic function determine left atrial reservoir function. Circulation. 1999;100(4):427–36.
26. Kim KJ, Choi HM, Yoon YE, et al. Left Atrial Mechanical Function and Global Strain in Hypertrophic Cardiomyopathy. PLoS ONE. 2016;11(6):e0157433.
27. Yu SQ, Shi K, Li Y, et al. The impact of diabetes mellitus on cardiac function assessed by magnetic resonance imaging in patients with hypertrophic cardiomyopathy. Cardiovasc Diabetol. 2024;23:293.
28. Ellims AH, Iles LM, Ling L, han, Hare JL, Kaye DM, Taylor AJ. Diffuse myocardial fibrosis in hypertrophic cardiomyopathy can be identified by cardiovascular magnetic resonance, and is associated with left ventricular diastolic dysfunction. J Cardiovasc Magn Reson Off J Soc Cardiovasc Magn Reson. 2012;14(1):76.
29. Kazui T, Henn MC, Watanabe Y, et al. The impact of 6 weeks of atrial fibrillation on left atrial and ventricular structure and function. J Thorac Cardiovasc Surg. 2015;150(6):1602–8.
30. Prinz C, Van Buuren F, Bogunovic N, Bitter T, Faber L, Horstkotte D. In patients with hypertrophic cardiomyopathy myocardial fibrosis is associated with both left ventricular and left atrial dysfunction. Acta Cardiol. 2012;67(2):187–93.
31. Urbano-Moral JA, Rowin EJ, Maron MS, Crean A, Pandian NG. Investigation of global and regional myocardial mechanics with 3-dimensional speckle tracking echocardiography and relations to hypertrophy and fibrosis in hypertrophic cardiomyopathy. Circ Cardiovasc Imaging. 2014;7(1):11–9.
32. Saba SG, Chung S, Bhagavatula S, et al. A novel and practical cardiovascular magnetic resonance method to quantify mitral annular excursion and recoil applied to hypertrophic cardiomyopathy. J Cardiovasc Magn Reson. 2014;16(1):35.
33. Tayal B, Malahfji M, Buergler JM, Shah DJ, Nagueh SF. Hemodynamic determinants of left atrial strain in patients with hypertrophic cardiomyopathy: A combined echocardiography and CMR study. PLoS ONE. 2021;16(2):e0245934.
34. Cau R, Muscogiuri G, Pisu F, et al. Effect of late gadolinium enhancement on left atrial impairment in myocarditis patients. Eur Radiol. 2023;34(3):1846–53.
35. Ferreira VM, Plein S, Wong TC, et al. Cardiovascular magnetic resonance for evaluation of cardiac involvement in COVID-19: recommendations by the Society for Cardiovascular Magnetic Resonance. J Cardiovasc Magn Reson Off J Soc Cardiovasc Magn Reson. 2023;25(1):21.
36. Casas G, Rodríguez-Palomares JF. Multimodality Cardiac Imaging in Cardiomyopathies: From Diagnosis to Prognosis. J Clin Med. 2022;11(3):578.
37. Schuster A, Morton G, Hussain ST, et al. The intra-observer reproducibility of cardiovascular magnetic resonance myocardial feature tracking strain assessment is independent of field strength. Eur J Radiol. 2013;82(2):296–301.

38. Gao Y, Zhang Z, Guo L, et al. Age- and Sex-Specific MR-Feature Tracking Reference Values of Right Atrial Deformation in Healthy Adults. J Magn Reson Imaging JMRI. 2025;61(1):263–73.
39. Kamali R, Schroeder J, DiBella E, et al. Reproducibility of clinical late gadolinium enhancement magnetic resonance imaging in detecting left atrial scar after atrial fibrillation ablation. J Cardiovasc Electrophysiol. 2020;31(11):2824–32.

# Figures

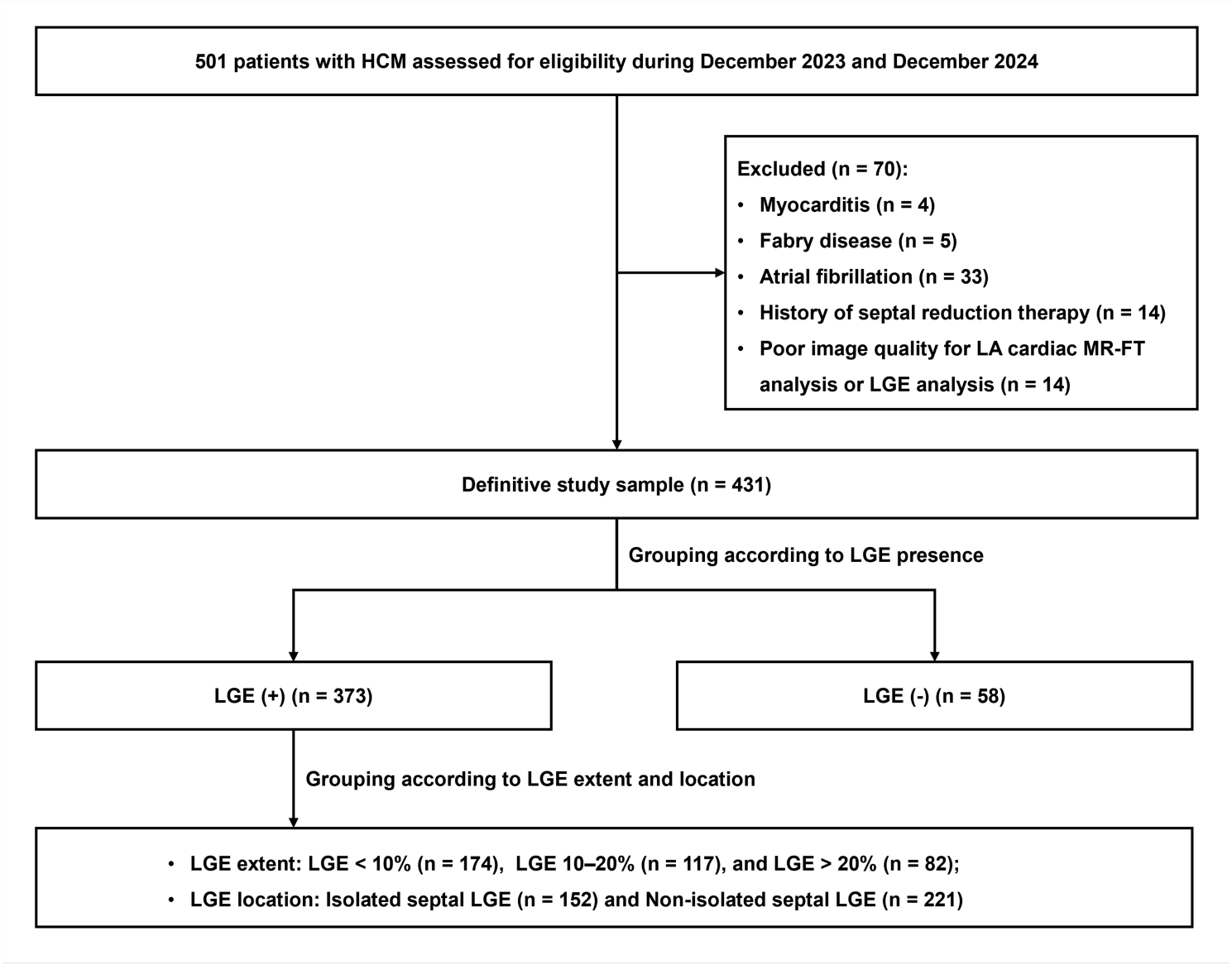
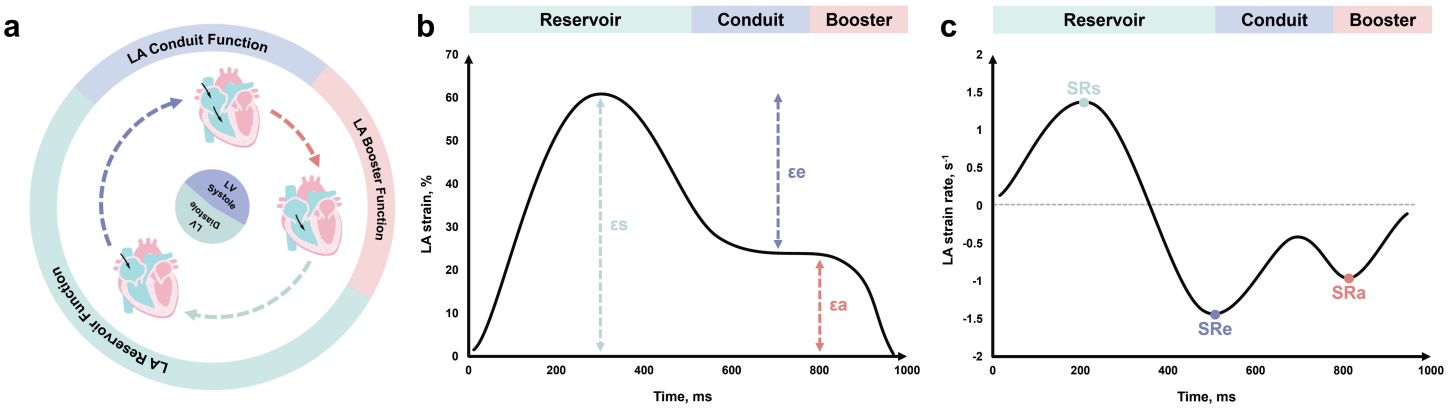


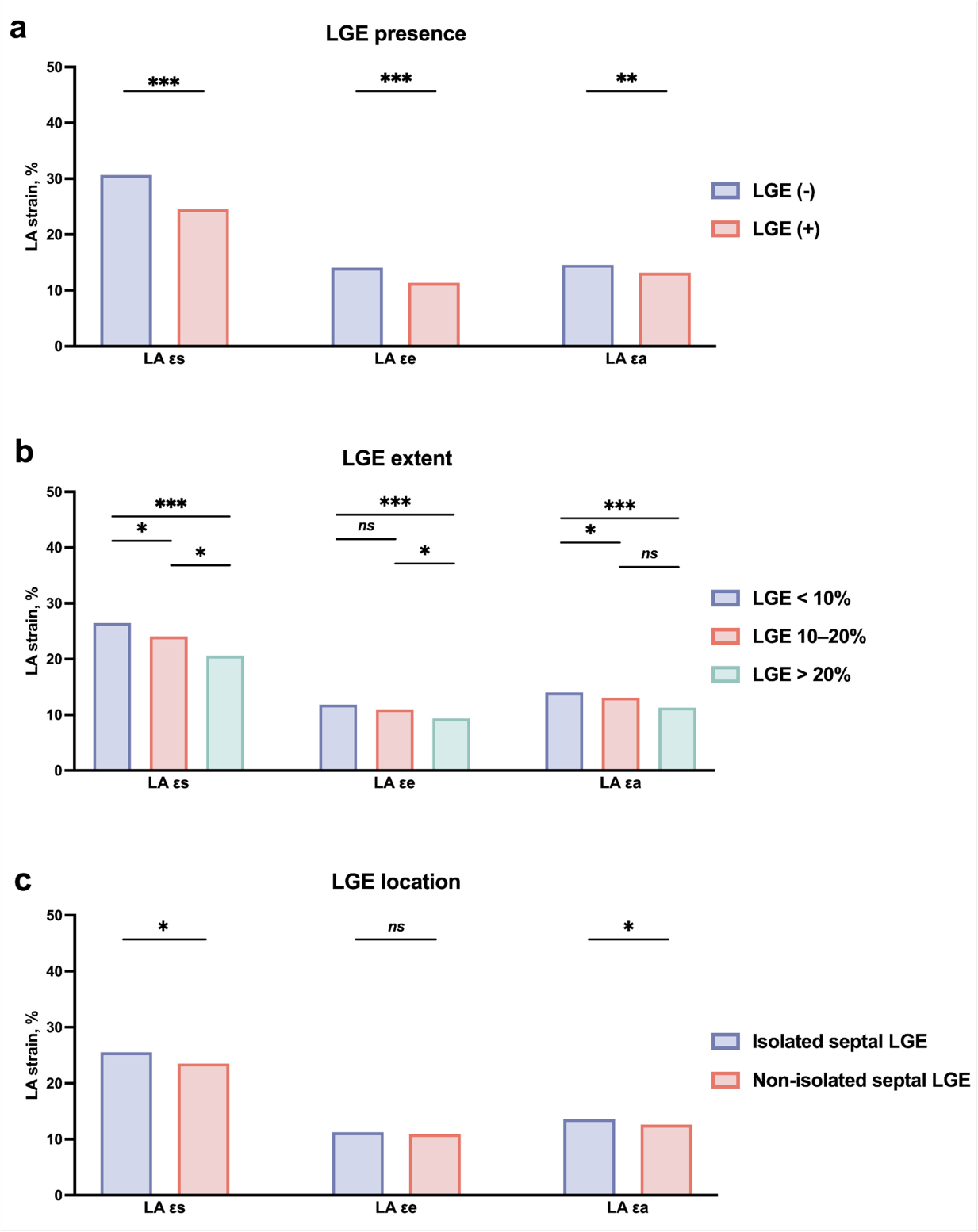

**Figure 1**

Flowchart of Patient Inclusion. HCM = hypertrophic cardiomyopathy; LA = left atrial; LGE = late gadolinium enhancement; MR-FT = magnetic resonance-feature tracking.




## Figure 2

LA Phasic Functions during Cardiac Cycle (**a**), and LA Phasic Strain-time Curve (**b**) and Strain Rate-time Curve (**c**). εa = booster strain; εe = conduit strain; εs = reservoir strain; LA = left atrial; LV = left ventricular; SRa = peak late negative strain rate; SRe = peak early negative strain rate; SRs = peak positive strain rate.




## Figure 3

Comparisons of LA Strain Parameters in Subgroups Stratified by LGE Presence (**a**), Extent (**b**), and Location (**c**). * Indicates $P < 0.05$, ** indicates $P < 0.01$, and *** indicates $P < 0.001$. εa = booster strain; εe = conduit strain; εs = reservoir strain; LA = left atrial; LGE = late gadolinium enhancement.

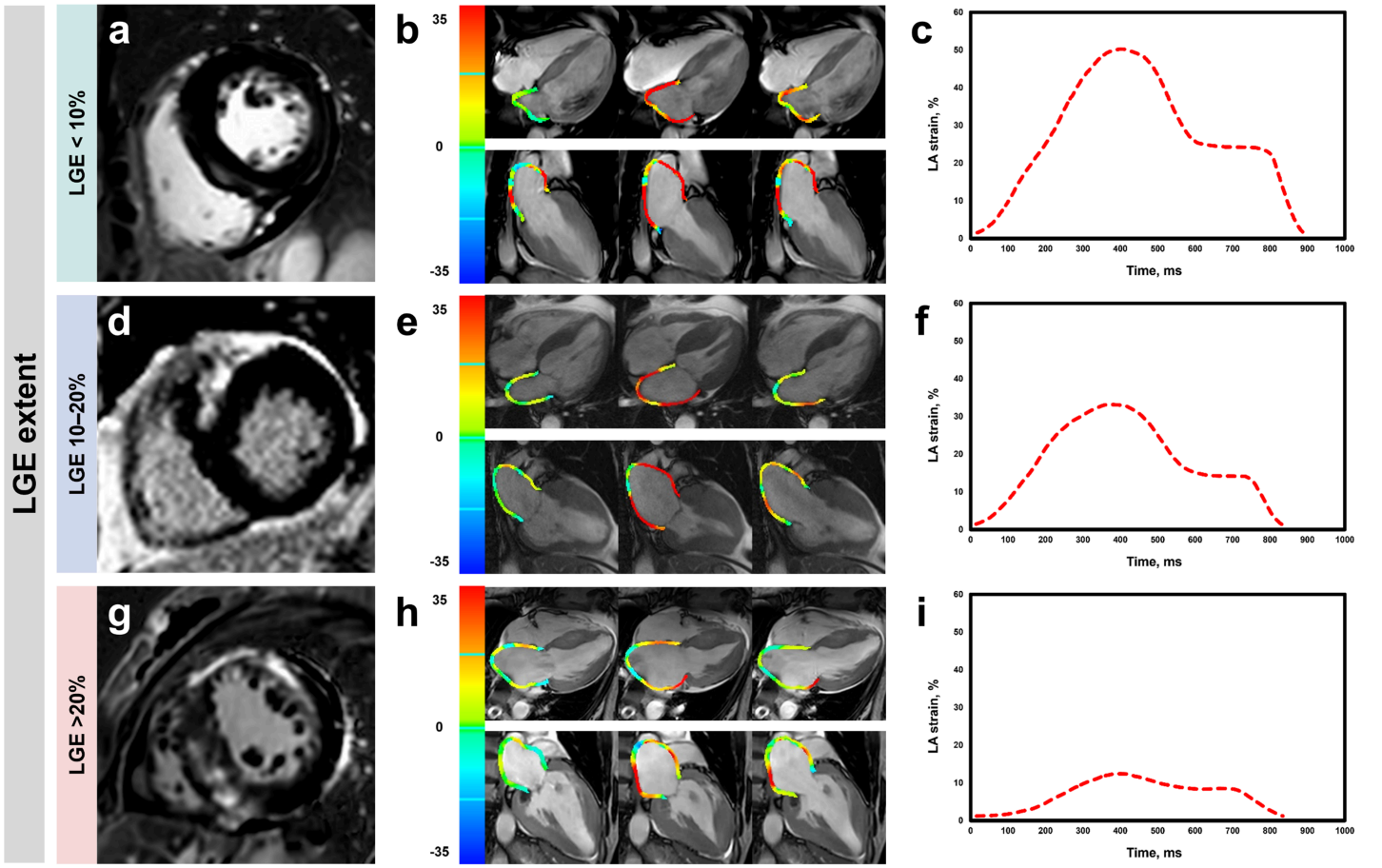



**Figure 4**

Representative Cases of LA Strain Measurements in LGE < 10% (**a–c**), LGE 10–20% (**d–f**), and LGE > 20% Subgroups (**g–i**). LA = left atrial; LGE = late gadolinium enhancement.

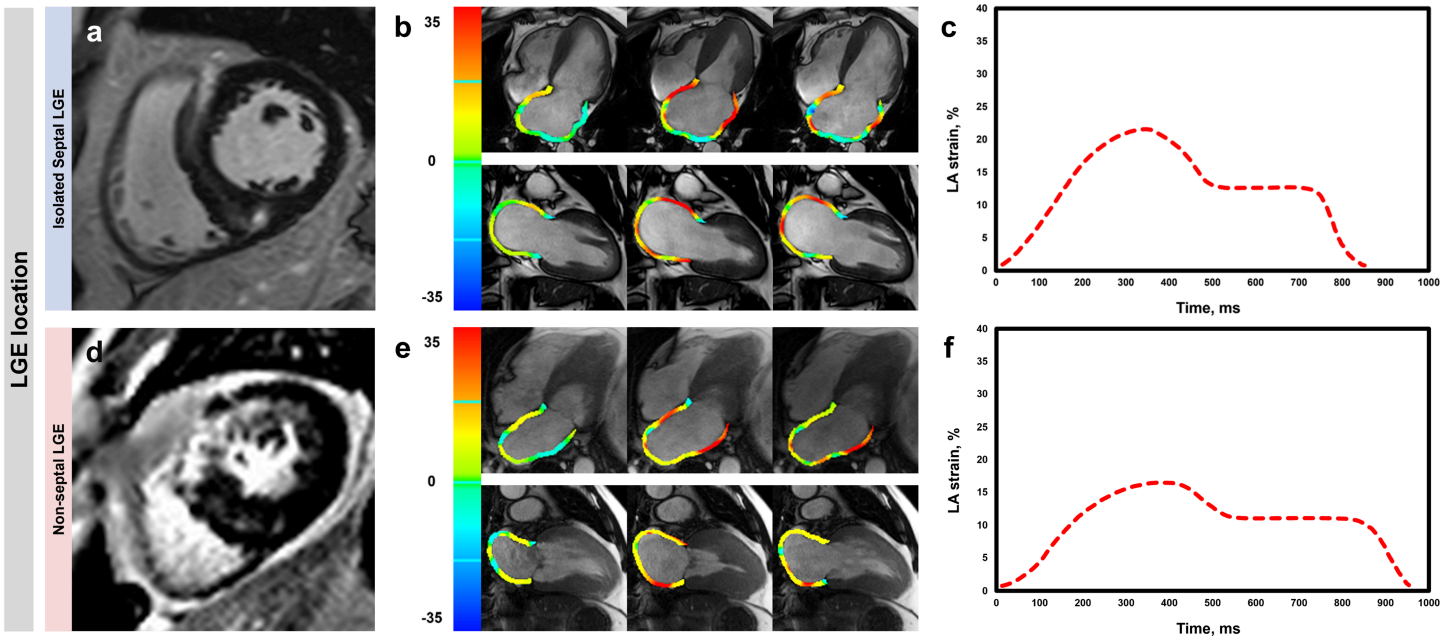


**Figure 5**

Representative Cases of LA Strain Measurements in Isolated Septal LGE (**a–c**) and Non-isolated Septal LGE Subgroups (**d–f**). LA = left atrial; LGE = late gadolinium enhancement.

## Supplementary Files

This is a list of supplementary files associated with this preprint. Click to download.

- SupplementaryMaterial.docx