SCIENTIFIC RESEARCH

ASSOCIATION OF LEFT ATRIAL VOLUME AND FUNCTION BY CMR WITH CLINICAL AND IMAGING FINDINGS IN HYPERTROPHIC CARDIOMYOPATHY

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SUMMARY

Objective: To evaluate the correlation of left atrial (LA) volume and function assessed by cardiac magnetic resonance (CMR) with selected clinical and imaging findings in patients with hypertrophic cardiomyopathy (HCM).

Subjects and Methods: A cross-sectional descriptive study was conducted on 58 patients diagnosed with HCM at Bach Mai Hospital from March 2024 to October 2025. All patients underwent CMR imaging to assess LA volume and LA function using the specialized software CVi42 (Circle Cardiovascular Imaging). Correlations were analyzed using IBM SPSS Statistics for Windows, version 20.0.

Results: The mean age was 56.6 ± 18.0 years, with a male-to-female ratio of 1.4:1. Significant correlations were found of LA indices with:

(1) Dyspnea severity: LA volume progressively increased, and function decreased with worsening dyspnea.

(2) Extent of Late Gadolinium Enhancement (LGE): Moderate to strong correlations were observed with all LA volume and function indices (all $p < 0.01$).

(3) Left Ventricular Mass (LVM): Moderate correlation with LA EFp ($r = -0.39$, $p = 0.003$) but no correlation with LA EFa and LA EFt. Maximal left ventricular wall thickness (Max LVWT) showed no significant correlation ($p > 0.05$).

Conclusion: CMR-derived LA volume and function indices demonstrate a strong correlation with dyspnea severity and the extent of LGE but show a weak correlation with the degree of left ventricular hypertrophy.

Keywords: *Hypertrophic cardiomyopathy, left atrium, cardiac magnetic resonance, left atrial volume, left atrial function, late gadolinium enhancement.*

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I. INTRODUCTION

Hypertrophic cardiomyopathy (HCM) is a common genetic cardiovascular disease with an estimated prevalence of 1 in 500 in the general population. It can lead to serious complications such as heart failure, atrial fibrillation, and sudden death, particularly in young individuals[1]. The disease is characterized by unexplained left ventricular (LV) hypertrophy, often accompanied by diastolic dysfunction, which affects hemodynamics.

Previously, studies on HCM primarily focused on left ventricle (LV) characteristics, while the role of the left atrium (LA) received limited attention. Recently, numerous studies have indicated that the LA is not merely a passive chamber but plays a central role in the pathogenesis of heart failure with preserved ejection fraction (HFpEF) - a common complication of HCM[2]. LA dilation and dysfunction are associated with adverse cardiovascular events, including atrial fibrillation, heart failure, and cardiovascular death[3,4].

In Vietnam, although there have been studies on CMR imaging characteristics of HCM[5,6], there have not been many studies focusing comprehensively on the role of LA. Therefore, we aimed to evaluate the correlation of CMR-derived LA indices with selected clinical and imaging findings in this patient group.

II. SUBJECTS AND METHODS

2.1. Subjects

The study included 58 patients diagnosed with HCM at Bach Mai Hospital from March 2024 to October 2025, according to the 2020 American Heart Association/ American College of Cardiology (AHA/ACC) guidelines[7].

- **Exclusion criteria:**
  - Patients with severe hypertension, severe diabetes mellitus, end-stage renal disease, moderate or severe aortic stenosis, aortic coarctation, cardiac amyloidosis, Fabry disease.
  - Incomplete medical records.
  - Or suboptimal CMR image quality.

2.2. Methods

- **Study Design:** Cross-sectional, retrospective, and prospective descriptive study.
- **CMR Equipment:** CMR was performed using a SIGNA Architect 3.0 Tesla system (GE, USA) and a Prodiva CX 1.5 Tesla system (Philips, Netherlands) at the Radiology Center, Bach Mai Hospital.
- **CMR Protocols:**
  - Initial localizers were acquired in axial, coronal, sagittal, 2-chamber, 3-chamber, 4-chamber, short-axis, and left ventricular outflow tract (LVOT) planes.
  - Cine sequences were acquired in 2-chamber, 4-chamber, and short-axis planes (8–10 slices) from base to apex to assess LA volume and function, LV mass, ejection fraction (EF), end-systolic and end-diastolic volumes, and wall thickness.
  - 3-chamber and LVOT cine sequences were used to evaluate Systolic Anterior Motion (SAM), mitral valve leaflet length, and LVOT diameter.
  - Contrast administration: Gadovist (0.2 mmol/kg) was administered intravenously at a rate of 2–3 ml/s, followed by a 20–25 ml saline flush.
  - Late Gadolinium Enhancement (LGE) imaging was performed 10 minutes post-administration. Optimal Time to Inversion (TI) was determined to null normal myocardium using an Inversion Recovery (IR) sequence. LGE images were acquired in 2-chamber, 4-chamber, and eight short-axis slices from base to apex to assess fibrosis and myocardial scarring.
- **Image Analysis:** Image analysis was performed using CVi42 software (Circle Cardiovascular Imaging, Canada) by experienced cardiac radiologists.

- **Measurement Parameters:**
  - **Left Atrial Volume:**
    - LAVmax: Maximum LA volume (end-systole).
    - LAVmin: Minimum LA volume (end-diastole).
    - LAVpreA: LA volume before atrial contraction.
    - LAVi: LA volume index = LAV/BSA (ml/m²)

      (BSA calculated using the Du Bois formula).
  - **Left Atrial Function:**
    - LAEFp (Passive Emptying Fraction) = (LAVmax - LAVpreA)/LAVmax
    - LAEFa (Active Emptying Fraction) = (LAVpreA - LAVmin)/LAVpreA
    - LAEFt (Total Emptying Fraction) = (LAVmax - LAVmin)/LAVmax
  - **Left Atrial Strain:** Measured using feature tracking on 2-chamber and 4-chamber cine sequences, including:
    - LA longitudinal strain: an index that evaluates the longitudinal deformation of the left atrium during the cardiac cycle, reflecting the expansion and contraction capability of the left atrial wall. This index is equivalent to LA reservoir strain in some other reports.
    - LA AV-junction strain: an index that characterizes the longitudinal displacement of the left atrioventricular junction during ventricular systole, indirectly reflecting the contractile and relaxation function of the left atrium.
  - **Other parameters:** Left ventricular ejection fraction (LVEF), maximal left ventricular wall thickness (Max LVWT), left ventricular mass (LVM), left ventricular mass index (LVMI), LGE mass, and percentage of LGE (%LGE).

2.3. Data Collection and Analysis

Data were collected using a standardized data collection form. Data were processed and analyzed using IBM SPSS Statistics for Windows, version 20.0.

2.4. Ethics in research

The study was approved by the Biomedical Research Ethics Council of Bach Mai Hospital. Patient personal information was kept strictly confidential and used solely for scientific research purposes.

III. RESULTS

3.1. General Characteristics

The study included 58 patients with a mean age of 56.6 ± 18.0 years; the majority were over 50 years old (72.4%). There were 34 male patients (58.6%), a male-to-female ratio of 1.4:1.

Regarding history, 32.8% had hypertension, 15.5% had diabetes, and 13.8% had a family history of HCM or sudden death. Dyspnea was the most common symptom (69%), primarily NYHA class II (48.3%). Chest pain occurred in 53.4% of patients, and syncope/presyncope in 10.3%.

3.2. Left Atrial Characteristics on CMR

Table 1. Left atrial characteristics on CMR.

Characteristic	Mean Value	Minimum	Maximum
LAVmax (ml)	82.45 ± 33.58	19.98	178.70
LAVi-max (ml/m²)	51.17 ± 19.97	12.81	101.81
LAVmin (ml)	49.17 ± 30.09	6.22	153.26

LAVi-min (ml/m^2)	30.29 ± 17.37	3.99	86.45
LAVpreA (ml)	64.28 ± 30.85	12.21	161.96
LAEFp (%)	23.51 ± 9.08	3.28	44.99
LAEFa (%)	27.32 ± 13.09	4.50	60.77
LAEFt (%)	44.14 ± 13.24	14.24	74.04
LA longitudinal strain (%)	17.82 ± 12.34	2.24	56.70
LA AV-junction strain (%)	15.36 ± 10.57	3.33	56.56

LA volume indices were significantly increased (LAVi-max = 51.17 ± 19.97 ml/m², LAVi-min = 30.29 ± 17.37 ml/m²). LA function indices were reduced: LAEFp = 23.51 ± 9.08%, LAEFa = 27.32 ± 13.09%, LAEFt = 44.14 ± 13.24%. LA strain indices were also markedly reduced.

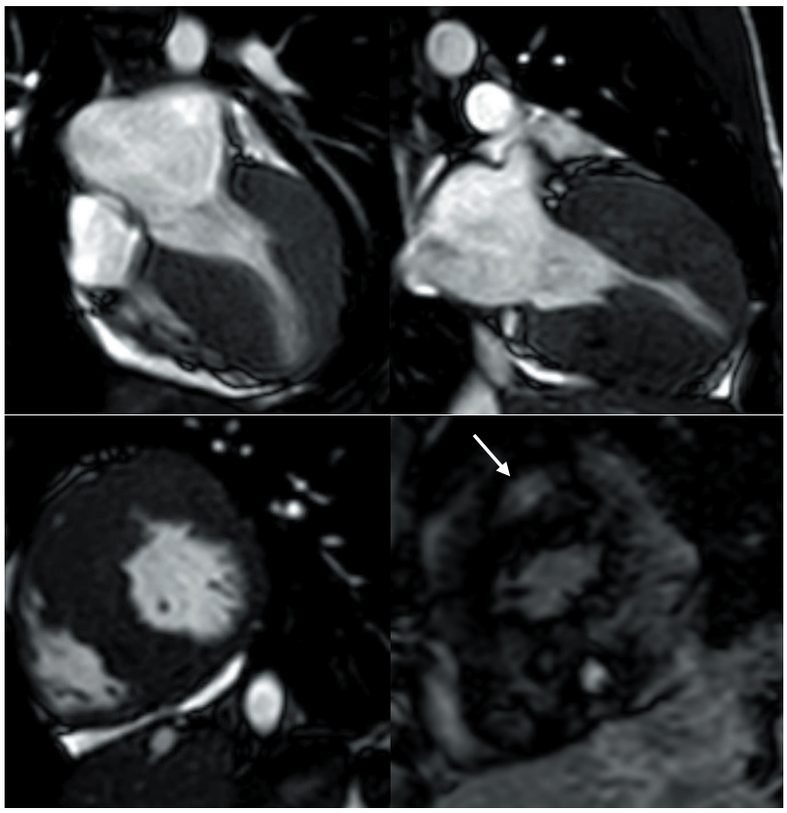

Figure 1. *A 16-year-old male, presenting with multi-segmental left ventricular hypertrophy and left atrial dilation. Late gadolinium enhancement (LGE) is observed in the mid-anterior segment (arrow).*

3.3. Correlations with Clinical and Imaging Findings

- **Correlation with Clinical Symptoms**

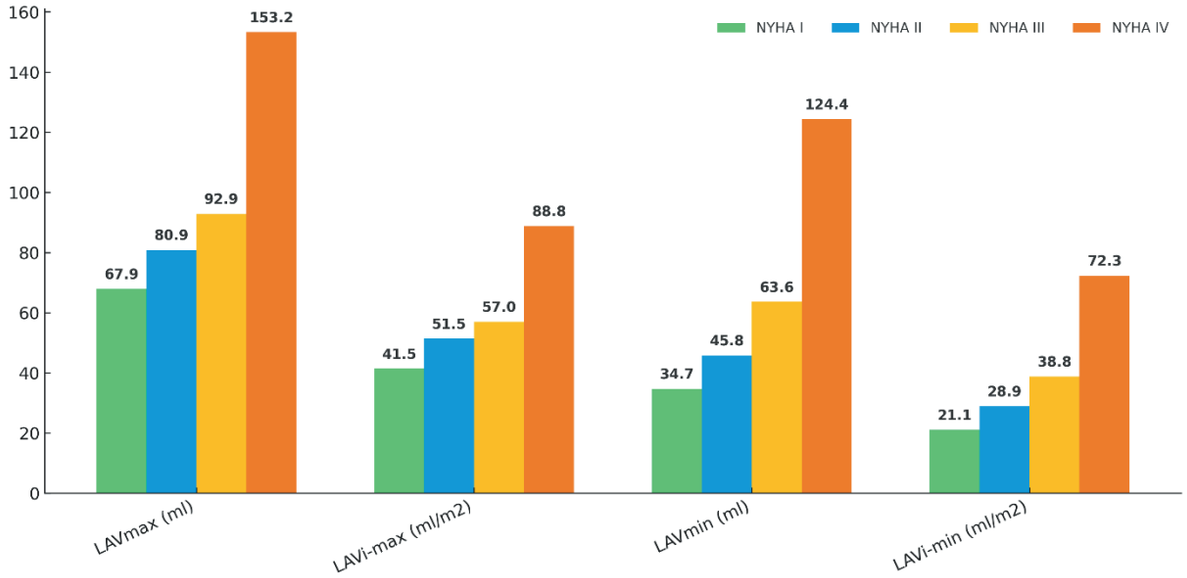

Figure 1. LA volume indices by dyspnea severity.

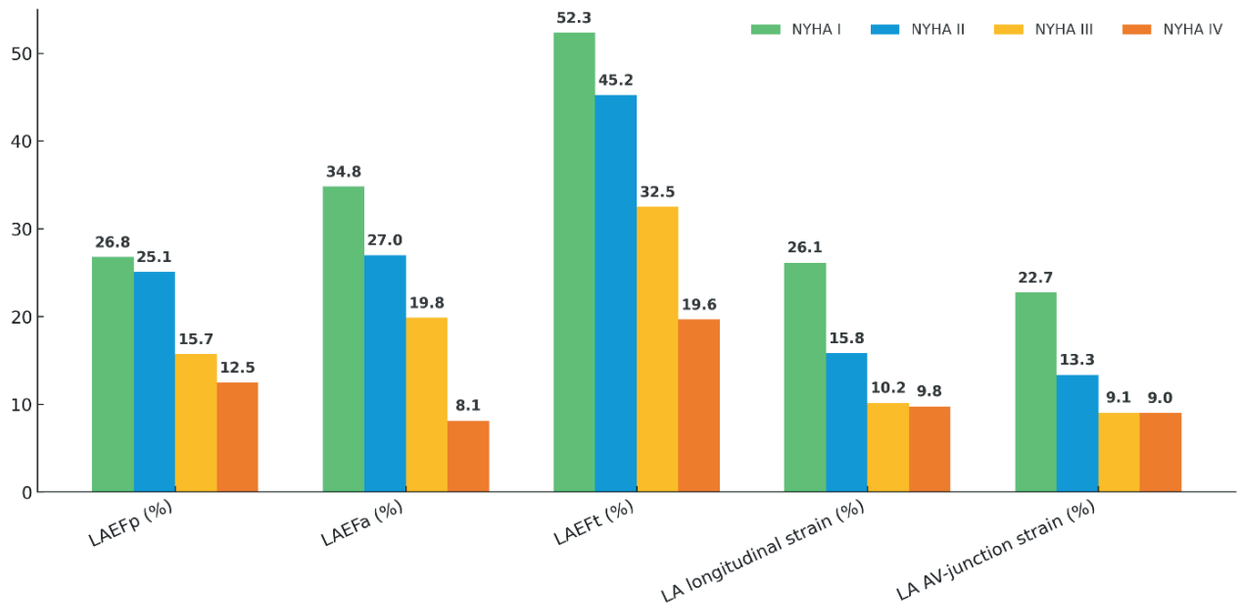

Figure 2. LA function indices by dyspnea severity.

There was a significant correlation between the severity of dyspnea and LA volume and function ($p < 0.01$). Dyspnea severity was positively correlated with LA volume indices and negatively correlated with LA function indices. However, no statistically significant difference was found between groups with and without chest pain, or between groups with and without syncope ($p > 0.05$).

- **Correlation with Other CMR Parameters**

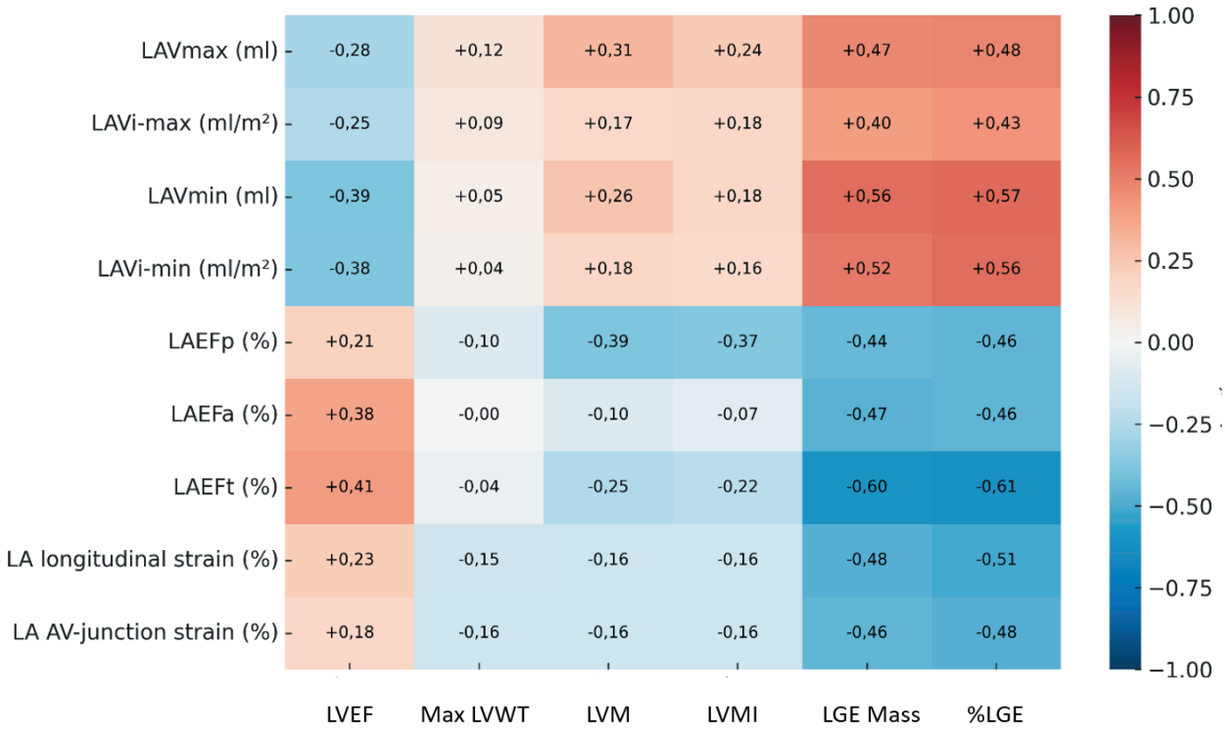

Figure 3. Heatmap of correlations between LA volume, function indices and CMR parameters.

Correlation analysis showed that the extent of myocardial LGE had the strongest correlation with LA indices: a moderate-to-strong positive correlation with volume (r = 0.40 to 0.57, $p < 0.01$) and a moderate-to-strong negative correlation with LA function (r = -0.44 to -0.61, $p < 0.001$).

LVM and LVMI showed a moderate negative correlation with LAEFp (r = -0.37 to -0.39, $p < 0.01$).

LVEF showed a moderate negative correlation with LA volume (r = -0.25 to -0.39).

Max LVWT showed no significant correlation with LA indices ($p > 0.05$).

IV. DISCUSSION

1. Correlation between LA indices and Dyspnea Severity

Our study found a strong and statistically significant correlation between LA indices and dyspnea severity classified by NYHA ($p < 0.01$ for all indices). This close relationship can be explained by a pathophysiological cascade initiated by LV hypertrophy. This condition precipitates LV diastolic dysfunction, leading to increased LV filling pressure and subsequent LA pressure overload. Consequently, the left atrium undergoes dilation and functional decline, which impairs the regulation of LV filling pressure. This chain of events results in elevated pulmonary venous pressure and pulmonary congestion, ultimately manifesting as dyspnea. This strong correlation provides a clear explanation for dyspnea symptoms in HCM patients with preserved LVEF.

2. Correlation between LA indices and LGE

This was the strongest and most significant correlation in our study. The mass and percentage of LGE (indicators of myocardial fibrosis) showed a moderate-to-strong positive correlation with all LA volume indices and a moderate-to-strong negative correlation with all LA function indices. The results suggest that fibrosis in HCM is not localized solely to the LV but is a process affecting the LA via three mechanisms:

1. **Parallel LA fibrosis:** Sarcomere gene mutations affect both ventricular and atrial myocardium, with fibrosis occurring simultaneously in both chambers. A study by Farhad et al reported similar findings [3].
2. **Secondary LA damage from LV fibrosis:** Increased LV stiffness leads to elevated diastolic pressure, resulting in chronic pressure overload. This hemodynamic burden induces LA dilation and subsequent reactive fibrosis, ultimately causing reduced LA function.
3. **Microvascular dysfunction:** Fibrosis induces microvascular obstruction, leading to reduced myocardial perfusion. This results in chronic ischemia, ultimately causing injury to the LA.

Structurally, fibrosis affects the LA by replacing myocytes with non-contractile fibrous tissue, disrupting myofiber arrangement, decoupling myocytes, and reducing capillary density. Functionally, fibrosis reduces the number of functional myocytes (decreasing LAEFa), increases atrial wall stiffness (decreasing LAEFp), and causes loss of elasticity (decreasing LA strain) and electrical conduction disturbances (increasing atrial fibrillation risk). Studies by Farhad[3] and Di Tian[8] indicated that high LGE increases the risk of adverse cardiovascular events. Although we could not directly assess the correlation with event risk, our results suggest a potential link between LA indices and cardiovascular outcomes.

3. Correlation between LA indices and LV Hypertrophy

The results showed no statistically significant correlation between maximal LV wall thickness and any LA index ($p > 0.05$). This may be explained by several factors. Fisrt, maximal thickness reflects only localized hypertrophy, not diffuse severity. Second, hypertrophy location is more important than thickness (basal septal hypertrophy causing obstruction affects the LA more than apical hypertrophy). Third, determinants are total muscle mass, fibrosis, diastolic dysfunction, and LVOT obstruction rather than thickness alone.

LVM showed a weak correlation with LA volume and a moderate negative correlation with LAEFp, but no significant correlation with LAEFa, LAEFt, or LA strain ($p > 0.05$). The mechanism is that LAEFp reflects passive conduit function during early diastole, which depends on the atrial-ventricular pressure gradient and LV compliance. Increased LVM leads to heightened LV stiffness and elevated diastolic pressure. This elevation reduces the AV pressure gradient, thereby impairing passive emptying. Conversely, LAEFa reflects active atrial contraction, depending primarily on the condition of the atrial myocardium (fibrosis, remodeling). Thus, LVM acts indirectly via diastolic dysfunction (affecting LAEFp) rather than directly on the atrial myocardium (affecting LAEFa).

4.4. Comparison between Factors

Comparing the degree of correlation with LAEFt revealed distinct differences: Maximal wall thickness ($r = -0.04$, $r^2 = 0.002$, $p = 0.788$) explained only 0.2% of LAEFt variation; LVM ($r = -0.25$, $r^2 = 0.063$, $p = 0.055$) explained 6.3%; whereas LGE ($r = -0.60$ to -0.61, $r^2 = 0.360$–0.372, $p < 0.001$) explained 36–37.2% of the variation. This indicates that myocardial fibrosis is the most decisive factor affecting LA function, while LVM has an intermediate effect via diastolic dysfunction, and maximal wall thickness alone is a poor predictor.

V. CONCLUSION

The study showed that CMR-derived left atrial volume and function confirmed the central role of the LA in the mechanism of dyspnea and heart failure with preserved ejection fraction syndrome. Myocardial fibrosis is the most decisive factor affecting LA structure and function, far exceeding other factors, but it does not have a high correlation with the degree of left ventricular hypertrophy.

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Received: 19/12/2025. Assessed:: 19/12/2025. Accepted: 25/12/2025