Original Article | Cardiovascular Imaging

eISSN 2005-8330
https://doi.org/10.3348/kjr.2024.1143
Korean J Radiol 2025;26(4):324-332

Korean Journal of Radiology
KJR

Coronary Artery Dimension-Adjusted Subtended Myocardial Mass Obtained With Coronary CT Angiography as a Potential Biomarker of Myocardial Ischemia in Patients With Hypertrophic Cardiomyopathy

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Objective: To compare coronary artery dimension-adjusted subtended myocardial mass between patients with hypertrophic cardiomyopathy (HCM) and a normal population without detectable atherosclerosis, and between HCM patients with and without chest pain.

Materials and Methods: Twenty-five patients with HCM but no detectable atherosclerosis on coronary computed tomography angiography (CCTA) were included in the study. This group comprised 14 patients with chest pain and 11 patients without chest pain. They were matched with 25 healthy participants based on sex, age, coronary dominance pattern, and body surface area. The minimal lumen area (MLA) and subtended myocardial volume (V_{sub}) were assessed in the left main (LM), proximal left anterior descending (pLAD), proximal left circumflex (pLCx), and proximal right coronary (pRCA) arteries. Additionally, an index of the subtended myocardial mass adjusted for the MLA, calculated as V_{sub}/MLA^2, was determined.

Results: MLA was significantly larger in patients with HCM compared to the control group in LM (20.93 ± 6.31 mm^2 vs. 15.24 ± 3.90 mm^2, $P < 0.001$), pLAD (14.28 ± 3.55 mm^2 vs. 11.36 ± 2.07 mm^2, $P = 0.001$), pLCx (10.94 ± 3.60 mm^2 vs. 9.15 ± 2.93 mm^2, $P = 0.045$), and pRCA (13.41 ± 4.85 mm^2 vs. 11.22 ± 3.20 mm^2, $P = 0.018$). Despite an increase in coronary luminal area, patients with HCM exhibited significantly higher V_{sub}/MLA^2 compared to the control group in both the pLAD (403.56 ± 200.35 mm^{-1} vs. 241.70 ± 85.87 mm^{-1}, $P < 0.001$) and the pRCA (186.06 ± 95.07 mm^{-1} vs. 125.07 ± 70.18 mm^{-1}, $P = 0.007$). V_{sub}/MLA^2 was significantly elevated in patients with chest pain compared to those without in the pLAD (473.75 ± 227.38 mm^{-1} vs. 314.24 ± 110.74 mm^{-1}, $P = 0.018$) and the pLCx (417.04 ± 182.65 mm^{-1} vs. 275.29 ± 112.97 mm^{-1}, $P = 0.044$).

Conclusion: CCTA-derived V_{sub}/MLA^2 may more accurately reflect the balance between myocardial blood supply and demand, offering insights into the occurrence of demand angina in patients with HCM without obstructive coronary artery disease.

Keywords: Hypertrophic cardiomyopathy; Coronary artery disease; Coronary computed tomography angiography; Myocardial mass; Chest pain

INTRODUCTION

Hypertrophic cardiomyopathy (HCM) is a genetic cardiac disorder characterized by left ventricular (LV) hypertrophy [1,2]. Patients with HCM often experience chest pain suggestive of angina pectoris, and their electrocardiography (ECG) results may mimic those seen in myocardial infarction even in the absence of coronary artery disease (CAD) [3]. Myocardial ischemia without CAD is a well-recognized occurrence in patients with HCM. Various etiologic factors

Received: November 8, 2024 **Revised:** January 21, 2025 **Accepted:** February 9, 2025

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have been proposed, including distortion of the arteriolar architecture, intramural small-vessel disease, a high prevalence of myocardial bridging, and impairment of endothelium-dependent vasodilation.

A significant factor contributing to myocardial ischemia in HCM is the imbalance between oxygen supply and demand in the hypertrophied myocardium [4]. In 1996, Kaufmann et al. [5] demonstrated that coronary artery size increased as LV mass increased in both primary and secondary hypertrophy. Besides the epicardial coronary arteries, multiple studies have emphasized the role of microvascular dysfunction in triggering myocardial ischemia in patients with HCM [6-10]. Impaired coronary microvascular function serves as a robust predictor of adverse outcomes, including the progression to heart failure and sudden cardiac death, highlighting its clinical significance in HCM management [11]. Recent drug trials, such as the EXPLORER-HCM and VALOR-HCM studies [6,12], have yielded promising results for patients with symptomatic HCM, emphasizing the necessity for a more comprehensive understanding of the pathophysiology of chest pain in HCM.

A recent study by Yang et al. [13] utilized coronary computed tomography angiography (CCTA) to assess the subtended myocardial mass (V_{sub}). Based on this assessment, the study proposed a mathematical index, V_{sub}/minimal lumen area^2 (MLA), for detecting ischemia-inducing lesions, demonstrating comparability to on-site CT fractional flow reserve (FFR). Our study aimed to compare the corrected subtended myocardial mass for coronary artery dimensions between patients with HCM patients and atherosclerosis-free individuals. Additionally, we examined differences among patients with HCM based on the presence and absence of chest pain. The goal was to establish a novel imaging biomarker from CCTA to elucidate the mechanism of myocardial ischemia in HCM, emphasizing the potential imbalance between myocardial oxygen supply and demand due to myocardial hypertrophy.

MATERIALS AND METHODS

This retrospective study received approval from the Institutional Review Board of Chung-Ang University Hospital, with a waiver for patient consent regarding the utilization of clinical data (IRB No. 2112-037-19398).

Study Population

We examined patients diagnosed with HCM who underwent CCTA between January 2014 and February 2021. Exclusion criteria comprised a history of percutaneous coronary intervention, cardiovascular surgery, or cardiac implantable electronic device insertion. Patients with poor-quality CT images were also excluded. A total of 68 eligible patients with HCM were identified. To exclude patients with CAD, those with coronary artery calcium (coronary artery calcium score >0) or detectable coronary atherosclerosis on CT were excluded, resulting in 25 HCM patients without CAD. The presence of chest pain in each patient was determined based on electronic medical records documentation and categorized as 'chest pain' or 'no chest pain.' For comparison, the control group was selected from the general population who underwent CCTA as part of a health check-up, matched with HCM patients by sex, age (±4 years), coronary dominance, and body surface area (BSA) calculated using the Du Bois formula (±0.1 m^2). The coronary artery dominance pattern and segmentation were defined following the SYNTAX study criteria adapted for CCTA [14]. Finally, 50 patients were enrolled (25 HCM patients, including 14 with chest pain and 11 without, and 25 matched controls) (Fig. 1).

Acquisition of Coronary CT Angiography

CCTA was conducted using a Philips Brilliance 64 or iCT 256 scanner (Philips Healthcare, Cleveland, OH, USA) with a slice collimation of 64 x 0.625, tube voltage of 120 kV, gantry rotation time of 270 ms, and retrospective ECG gating with ECG-based tube current modulation. Prior to CCTA, patients with a heart rate >65 bpm received oral ß-receptor blockers (atenolol 50 mg; Tenormin®, AstraZeneca, Stockholm, Sweden), and all patients were given 0.8 mg of nitroglycerin sublingually. The contrast-enhanced scanning began 10 seconds after triggering using the bolus tracking technique (Bolus Pro Ultra; Philips Healthcare) with a trigger threshold of 110 Hounsfield units in the ascending aorta. Approximately 50–70 mL of contrast agent (Iomeron 400, 400 mg iodine/mL; Bracco Imaging SpA, Milan, Italy) was injected through the antecubital vein at a rate of 4.5–5 mL/s. This was followed by a 50 mL 1:1 mixed contrast saline chaser delivered at 4 mL/s using a dual-head power injector (Stellant; Medrad, Pittsburgh, PA, USA).

Measurements

Quantitative CCTA was conducted independently by two

 https://doi.org/10.3348/kjr.2024.1143

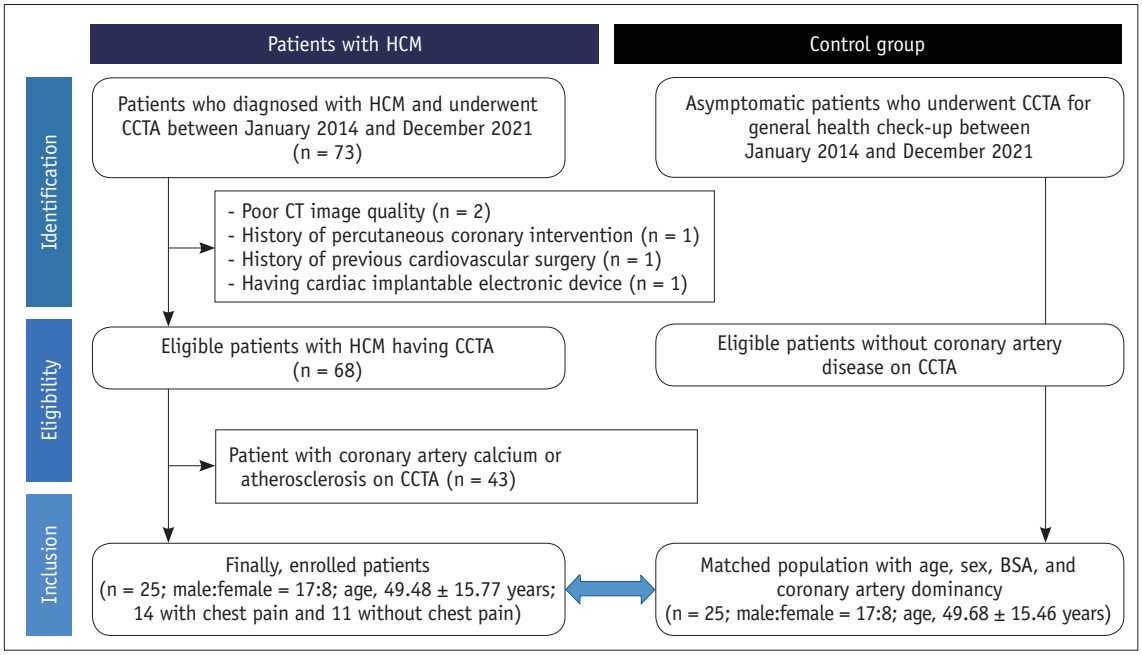

Fig. 1. Flowchart of study population selection. HCM = hypertrophic cardiomyopathy, CCTA = coronary computed tomography angiography, BSA = body surface area

observers: a board-certified radiologist and a technician with 11 and 5 years of experience in cardiac multimodality imaging, respectively. The analysis was conducted using a dedicated workstation (iNtuition, Terarecon, Foster City, CA, USA). Two readers independently analyzed the images, with one measurement obtained after a comprehensive joint review of the cases. The coronary artery diameter and lumen area were measured using an automatically generated multiplanar reconstruction, which was reviewed and adjusted as needed. All measurements were performed during the mid-diastolic phase (RR interval, 75%–78%).

The LV myocardium was automatically extracted through basic cardiac function analysis, and myocardial mass was semi-automatically calculated by delineating the outer and inner myocardial contours. The software integrated the two datasets three-dimensionally and quantified the LV territories distal to any point on CCTA [15] (Fig. 2).

The study measured the minimum coronary artery diameter, MLA, and the myocardial volume (territory) supplied by the left main (LM), proximal left anterior descending (pLAD), proximal left circumflex (pLCx), and proximal right coronary artery (pRCA). Additionally, researchers calculated an index of the myocardial mass supplied, adjusted for MLA, as V_{sub}/MLA^2, as reported in previous studies [13,16].

Statistical Analysis

Data are presented as mean ± standard deviation (SD), numbers, or percentages. Baseline characteristics and CCTA measurements between patients with HCM and controls were compared using paired Student's *t*-tests for continuous variables and the Wilcoxon signed-rank test for ordinal variables. CCTA measurements were also compared between the patients with and without chest pain using an independent Student's *t*-test for continuous variables and the Mann–Whitney U test for ordinal variables. Inter-observer agreement was assessed using intraclass correlation coefficients (ICC). A *P*-value <0.05 was considered statistically significant. Statistical analyses were conducted using SPSS Statistics for Windows version 20.0 (IBM Corp., Armonk, NY, USA).

RESULTS

Baseline Characteristics

Baseline characteristics of the study population are summarized in Table 1. Twenty-two pairs (88%) demonstrated right coronary artery dominance, while three pairs (12%) exhibited left coronary artery dominance. Patients with HCM had a notably higher prevalence of hypertension (40% vs. 8%, *P* = 0.008) and atrial fibrillation (15% vs. 0%, *P* = 0.037) compared to the control group.

https://doi.org/10.3348/kjr.2024.1143

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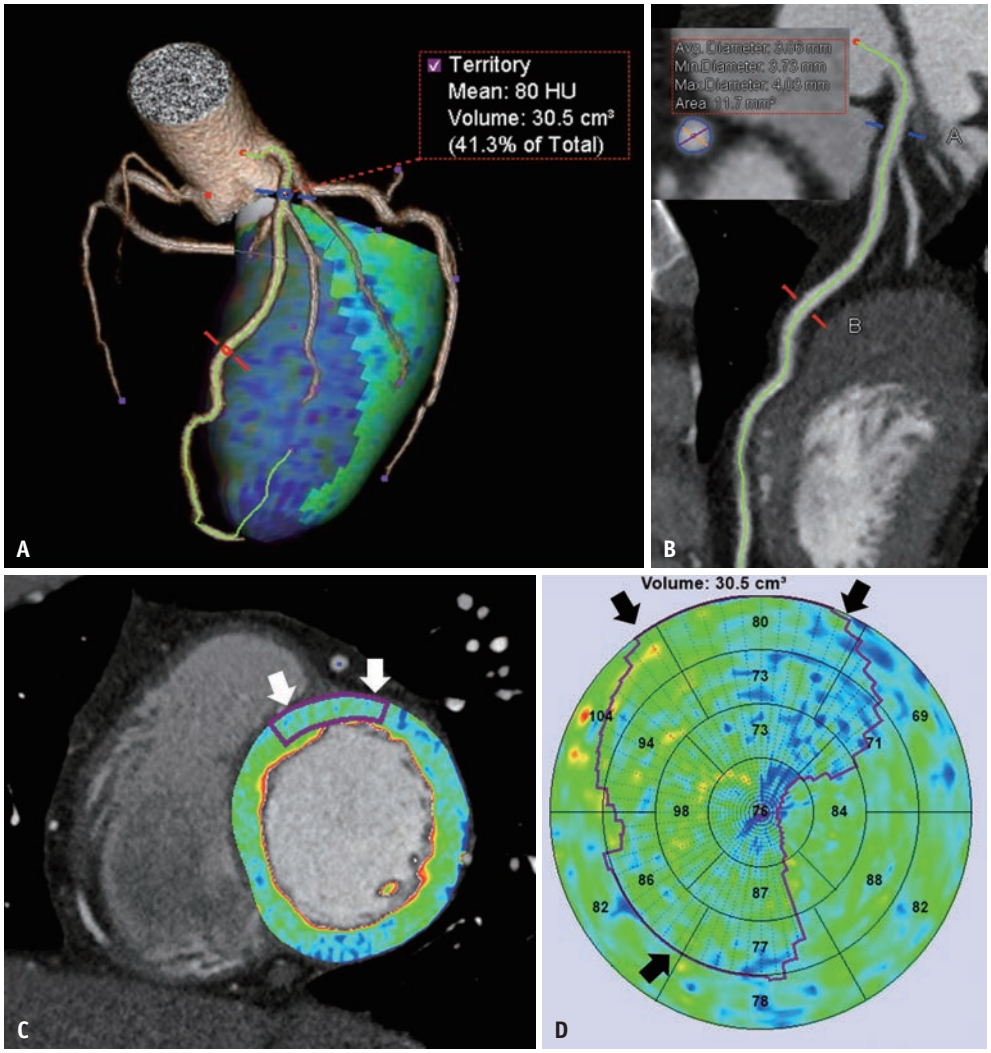

Fig. 2. Measurement of coronary artery and myocardial volume using coronary computed tomography angiography. **A-D:** Illustration of computed tomography measurements using curved multiplanar reconstructions of the coronary arteries and myocardial territory analysis through myocardial segmentation and integration on the dedicated workstation. Subtended myocardial volume (cm^3) (**A**, arrows in **C**, **D**), and minimal luminal diameter (mm) **(B)** and minimal luminal area (mm^2).

The current smoking rate was also significantly elevated in patients with HCM (18% vs. 8%, $P = 0.005$). Clinical symptoms were prevalent in the HCM cohort; 56% (14/25) reported chest pain, 44% (11/25) reported dyspnea, and 20% (5/25) reported syncope. In contrast, none of the control group participants reported any symptoms.

Comparison of CCTA Measurements Between Patients With HCM and Normal Control Group

LV Maximal Wall Thickness, Myocardial Volume, and Ejection Fraction

Patients with HCM showed significantly greater LV maximal wall thickness compared to the control group (21.48 ± 5.64 mm vs. 8.44 ± 1.53 mm, $P < 0.001$). Similarly, end-diastolic myocardial mass (230.87 ± 104.59 g vs. 119.16 ± 29.08 g, $P < 0.001$) and myocardial volume (226.70 ± 98.07 cm^3 vs. 113.45 ± 27.71 cm^3, $P < 0.001$) were notably higher in patients with HCM. Despite these structural differences, the ejection fraction did not significantly differ between the two groups (60.89% ± 9.21% vs. 57.68% ± 7.05%, $P = 0.242$) (Table 2).

The phenotypic distribution of HCM in the study population was as follows: 13 (52%) patients had asymmetric septal hypertrophy, 10 (40%) had apical hypertrophy, and two (8%) had concentric hypertrophy. Importantly, the type of HCM did not show a significant association with variations in the coronary artery measurements among patients with HCM.

https://doi.org/10.3348/kjr.2024.1143

Coronary Artery Diameter, Lumen Area, and Subtended Myocardial Volume

Regarding the coronary artery measurements, patients with HCM had significantly larger minimal lumen diameters, areas, and subtended myocardial volumes than those in the control group for all proximal coronary segments (Table 3, Fig. 3). The MLAs were significantly larger in patients with HCM for the LM (20.93 ± 6.31 mm^2 vs. 15.24 ± 3.90 mm^2, P < 0.001), pLAD (14.28 ± 3.55 mm^2 vs. 11.36 ± 2.07 mm^2, P = 0.001), pLCx (10.94 ± 3.60 mm^2 vs. 9.15 ± 2.93 mm^2, P = 0.045), and pRCA (13.41 ± 4.85 mm^2 vs. 11.22 ± 3.20 mm^2, P = 0.018). The subtended myocardial territory supplied at the point of coronary MLA was significantly larger for all proximal coronary arteries in patients with HCM, including LM (127.94 ± 66.81 cm^3 vs. 56.51 ± 20.12 cm^3, P < 0.001), pLAD (79.68 ± 39.05 cm^3 vs. 29.90 ± 10.80 cm^3, P < 0.001), pLCx (44.35 ± 38.13 cm^3 vs. 22.28 ± 14.90 cm^3, P = 0.002), and pRCA (30.64 ± 21.43 cm^3 vs. 15.90 ± 7.56 cm^3, P = 0.003). The interobserver agreement for the minimal lumen diameter and lumen area was good (ICC range: 0.80–0.98).

Table 1. Baseline characteristics of patients with HCM and control group

Characteristic	HCM (n = 25)	Control (n = 25)	*P*
Age, yrs	49.5 ± 15.8	49.7 ± 15.5	0.288
Sex, male:female	17:8	17:8	1.000
Height, m	166.2 ± 11.4	169.4 ± 11.2	0.190
Weight, kg	74.1 ± 14.0	71.9 ± 14.9	0.081
Heart rate, bpm	58.8 ± 9.8	54.7 ± 6.0	0.060
Body surface area	1.8 ± 0.2	1.8 ± 0.2	0.161
Coronary dominancy			1.000
Right	22 (88)	22 (88)	
Left	3 (12)	3 (12)	
Current smoking	9 (18)	2 (8)	0.005
Comorbidities			
Diabetes mellitus	1 (4)	1 (4)	1.000
Hypertension	10 (40)	2 (8)	0.008
Dyslipidemia	3 (12)	0 (0)	0.074
Atrial fibrillation	4 (15)	0 (0)	0.037
Chronic kidney disease	0 (0)	0 (0)	
Symptom presentation			
Chest pain	14 (56)	0 (0)	<0.001
Dyspnea	11 (44)	0 (0)	<0.001
Syncope	5 (20)	0 (0)	0.018

Data are mean ± standard deviation or number of patients (%).
HCM = hypertrophic cardiomyopathy

Coronary Artery Dimension-Adjusted Subtended Myocardial Volume (V_{sub}/MLA^2)

Despite increases in both the coronary luminal area and subtended myocardial volume, patients with HCM exhibited a higher V_{sub}/MLA^2 ratio compared to the control group. The difference in V_{sub}/MLA^2 was statistically significant for the pLAD, with values of 403.56 ± 200.35 mm^{-1} vs. 241.70 ± 85.87 mm^{-1} (P < 0.001), and for the pRCA, with values of 186.06 ± 95.07 mm^{-1} vs. 125.07 ± 70.18 mm^{-1} (P = 0.007).

Comparison of CCTA Measurements Between Patients With HCM Based on the Presence of Chest Pain

Table 4 summarizes the comparison of the MLA, subtended myocardial volume, and V_{sub}/MLA^2 ratio between individuals with and without chest pain. The V_{sub}/MLA^2 ratios of the pLAD (473.75 ± 227.38 mm^{-1} vs. 314.24 ± 110.74 mm^{-1}, P = 0.018) and pLCx (417.04 ± 182.65 mm^{-1} vs. 275.29 ± 112.97 mm^{-1}, P = 0.044) were significantly higher in those experiencing chest pain compared to those without chest pain (Fig. 4).

DISCUSSION

Our study showed that patients with HCM have significantly larger proximal coronary artery diameters and luminal areas compared to healthy controls. Despite the increased coronary artery dimensions, the subtended myocardial volume corrected with the square of the MLA (V_{sub}/MLA^2) was higher in patients with HCM, particularly in those with the pLAD and pRCA. Additionally, the V_{sub}/MLA^2 ratios were higher in the pLAD and pLCx in patients with HCM experiencing chest pain compared to asymptomatic patients. Given the distribution of HCM types in our study

Table 2. Comparison of cardiac CT measurements between HCM patients and control group

Parameter	HCM (n = 25)	Control (n = 25)	*P*
Maximal myocardial thickness, mm	21.48 ± 5.64	8.44 ± 1.53	<0.001
Ejection fraction, %	60.89 ± 9.21	57.68 ± 7.05	0.242
End-diastolic myocardial mass, g	230.87 ± 104.59	119.16 ± 29.08	<0.001
End-diastolic myocardial volume, cm^3	226.70 ± 98.07	113.45 ± 27.71	<0.001

Data are mean ± standard deviation.
HCM = hypertrophic cardiomyopathy

https://doi.org/10.3348/kjr.2024.1143

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Table 3. Coronary artery measurements on coronary computed tomography angiography in HCM patients and control group

	HCM (n = 25)	Control (n = 25)	*P*
Left main			
Minimal lumen diameter, mm	5.11 ± 0.72	4.37 ± 0.52	<0.001
MLA, mm^2	20.93 ± 6.31	15.24 ± 3.90	<0.001
V_{sub}, cm^3	127.94 ± 66.81	56.51 ± 20.12	<0.001
V_{sub}/MLA^2, mm^{-1}	295.75 ± 103.73	260.55 ± 90.52	0.668
Proximal left anterior descending			
Minimal lumen diameter, mm	4.22 ± 0.55	3.79 ± 0.34	0.001
MLA, mm^2	14.28 ± 3.55	11.36 ± 2.07	0.001
V_{sub}, cm^3	79.68 ± 39.05	29.90 ± 10.80	<0.001
V_{sub}/MLA^2, mm^{-1}	403.56 ± 200.35	241.70 ± 85.87	<0.001
Proximal left circumflex			
Minimal lumen diameter, mm	3.68 ± 0.60	3.37 ± 0.57	0.056
MLA, mm^2	10.94 ± 3.60	9.15 ± 2.93	0.045
V_{sub}, cm^3	44.35 ± 38.13	22.28 ± 14.90	0.002
V_{sub}/MLA^2, mm^{-1}	354.67 ± 174.32	255.64 ± 111.67	0.088
Proximal right coronary artery			
Minimal lumen diameter, mm	4.06 ± 0.78	3.74 ± 0.58	0.025
MLA, mm^2	13.41 ± 4.85	11.22 ± 3.20	0.018
V_{sub}, mm^3	30.64 ± 21.43	15.90 ± 7.56	0.003
V_{sub}/MLA^2, mm^{-1}	186.06 ± 95.07	125.07 ± 70.18	0.007

Data are mean ± standard deviation.
HCM = hypertrophic cardiomyopathy, MLA = minimal lumen area, V_{sub} = subtended myocardial volume

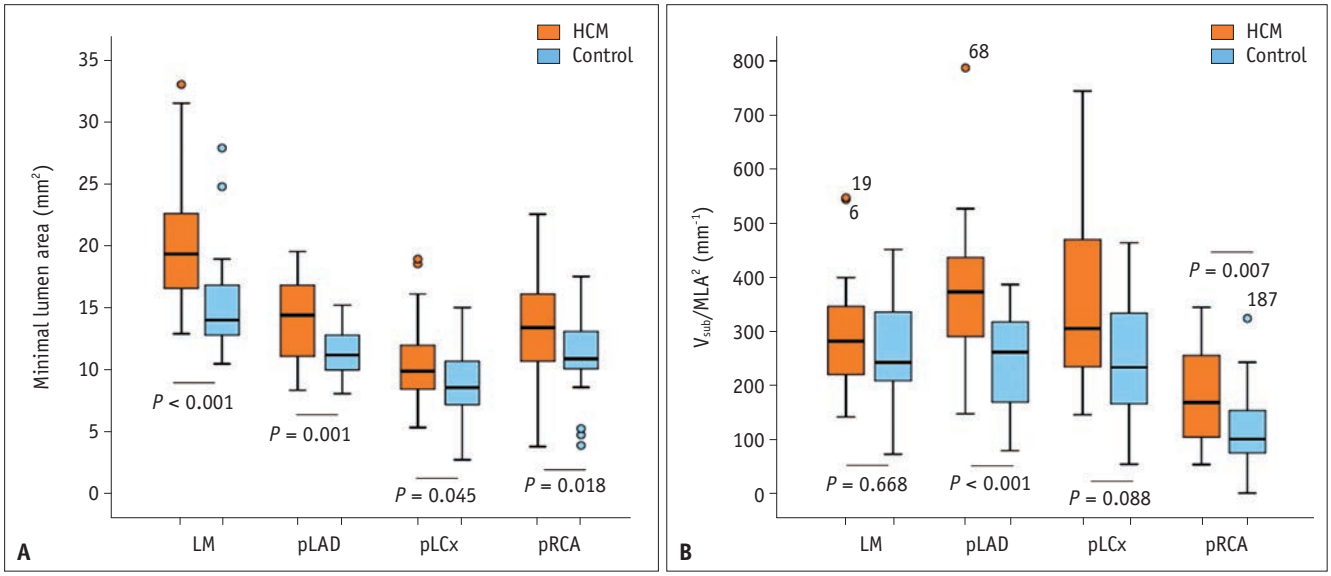

Fig. 3. Comparison of **(A)** MLA and **(B)** coronary artery dimension-adjusted V_{sub} (V_{sub}/MLA^2) between patients with HCM and control groups. MLA = minimal lumen area, V_{sub} = subtended myocardial volume, HCM = hypertrophic cardiomyopathy, LM = left main, pLAD = proximal left anterior descending, pLCx = proximal left circumflex, pRCA = proximal right coronary artery

(52% asymmetric septal type, 40% apical type, and 8% concentric type), we anticipated that the differences would be particularly notable for the pLAD, which supplies the hypertrophied myocardium.

A mathematical index of V_{sub}/MLA^2, as introduced by Yang et al. [13], has been shown to enhance the diagnostic accuracy in predicting an FFR <0.80, comparable to CT-FFR. By applying the concept of V_{sub}/MLA^2 to represent the coronary artery dimension-adjusted subtended myocardial mass, our analysis revealed a discrepancy between the heightened

https://doi.org/10.3348/kjr.2024.1143

Table 4. Comparison of coronary artery measurements in HCM patients with and without chest pain

	HCM without chest pain (n = 11)	HCM with chest pain (n = 14)	*P*
Right dominancy	10 (90.9)	12 (85.7)	0.775
Left main			
Minimal lumen diameter, mm	4.91 ± 0.66	5.26 ± 0.74	0.312
MLA, mm^2	19.26 ± 5.37	22.13 ± 6.84	0.285
V_{sub}, cm^3	107.22 ± 52.89	142.74 ± 71.83	0.312
V_{sub}/MLA^2, mm^{-1}	292.50 ± 75.19	305.21 ± 122.03	0.841
Proximal left anterior descending			
Minimal lumen diameter, mm	4.31 ± 0.52	4.15 ± 0.57	0.501
MLA, mm^2	14.91 ± 3.43	13.78 ± 3.68	0.403
V_{sub}, cm^3	72.87 ± 38.53	85.03 ± 40.05	0.536
V_{sub}/MLA^2, mm^{-1}	314.24 ± 110.74	473.75 ± 227.38	0.018
Proximal left circumflex			
Minimal lumen diameter, mm	3.76 ± 0.72	3.62 ± 0.50	0.936
MLA, mm^2	11.49 ± 4.29	10.51 ± 3.05	0.979
V_{sub}, cm^3	40.15 ± 33.97	47.65 ± 42.07	0.572
V_{sub}/MLA^2, mm^{-1}	275.29 ± 112.97	417.04 ± 182.65	0.044
Proximal right coronary artery			
Minimal lumen diameter, mm	4.18 ± 0.79	3.97 ± 0.80	0.647
MLA, mm^2	14.19 ± 5.22	12.80 ± 4.65	0.647
V_{sub}, mm^3	27.25 ± 15.49	33.31 ± 25.41	0.609
V_{sub}/MLA^2, mm^{-1}	156.42 ± 90.68	209.34 ± 95.05	0.222

Data are mean ± standard deviation, except for right dominancy expressed as number of patients (%).
HCM = hypertrophic cardiomyopathy, MLA = minimal lumen area, V_{sub} = subtended myocardial volume

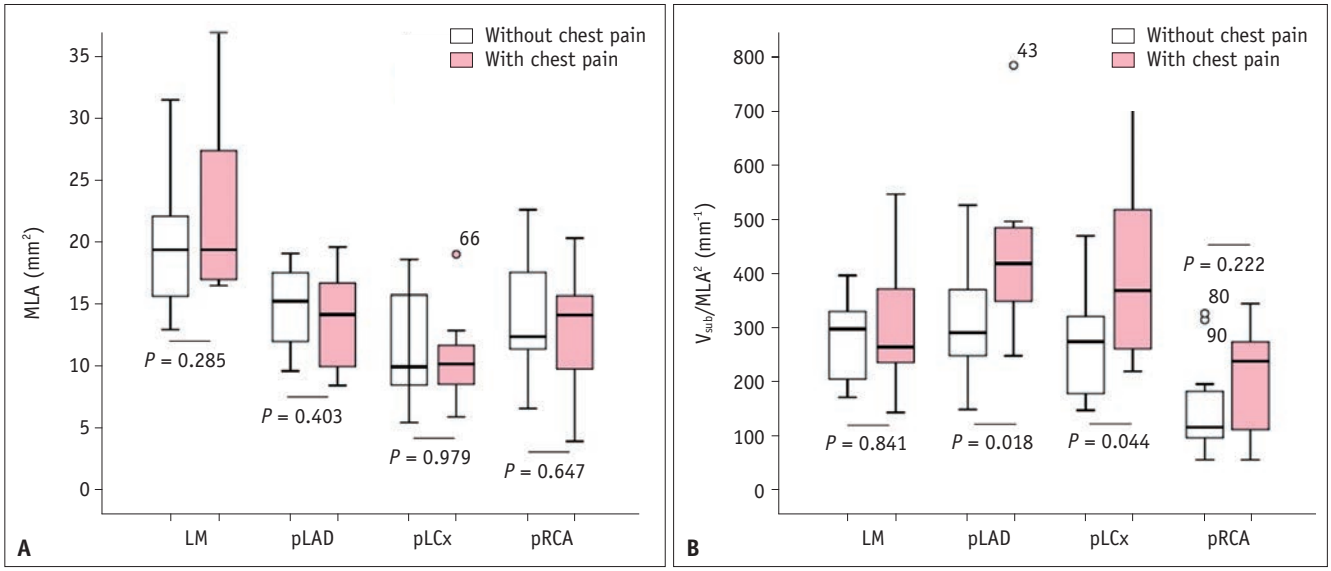

Fig. 4. Comparison of **(A)** MLA and **(B)** coronary artery dimension-adjusted V_{sub} (V_{sub}/MLA^2) in patients with HCM patients based on the presence or absence of chest pain. MLA = minimal lumen area, V_{sub} = subtended myocardial volume, HCM = hypertrophic cardiomyopathy, LM = left main, pLAD = proximal left anterior descending, pLCx = proximal left circumflex, pRCA = proximal right coronary artery

metabolic demand due to increased myocardial mass and the myocardial blood supply in patients with HCM. These findings suggest that V_{sub}/MLA^2 could function as a novel imaging biomarker for HCM, particularly in individuals experiencing chest pain, indicating an imbalance between myocardial blood supply and mass.

The presence of LV myocardial hypertrophy can lead to dynamic LV outflow tract obstruction, linked to increased cardiac morbidity and mortality [17]. The pathologic feature of thickened myocardium in HCM is characterized by

https://doi.org/10.3348/kjr.2024.1143

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myofibrillar disarray and fibrosis, which may contribute to diastolic dysfunction. Late gadolinium enhancement (LGE) on cardiac MRI serves as an imaging biomarker for detecting myocardial fibrosis. As per the AHA/ACC and ESC guidelines [18-20], the presence of LGE is a recognized risk factor warranting ICD insertion. However, unlike CT, MRI availability is limited in many clinical settings owing to cost, scan duration, and contraindications, such as pacemakers or ICDs. In contrast, CT offers greater availability, with shorter scan times, lower costs, and fewer contraindications.

The V_{sub}/MLA^2 index, extensively used in our analysis, has not yet been validated as a practical clinical indicator of ischemia-related symptoms in patients with HCM. Previous studies have established a connection between microvascular disease, the myocardial environment in HCM, and ischemia and fibrosis (scarring) [7-10]. Furthermore, thickening and abnormal function of the intramural coronary arterioles may result in microcirculatory ischemia in HCM, leading to chest pain [21]. Although the V_{sub}/MLA^2 index is based on the epicardial coronary artery lumen and does not directly represent the microvascular environment, it remains clinically valuable as a noninvasive imaging parameter reflecting the overall blood supply to the myocardium in HCM. When used in conjunction with cardiac SPECT or stress-perfusion imaging (CT/MR), this index has significant potential to offer a comprehensive understanding of myocardial blood flow pathophysiology in patients with HCM.

This study has several limitations. First, it was conducted at a single center with a relatively small study population, potentially restricting the generalizability of the findings. Second, the study population comprised individuals of a single ethnicity, possibly not fully representing the diverse global population. Third, functional data, such as cardiac SPECT or treadmill testing, were lacking, which could have facilitated establishing correlations between V_{sub}/MLA^2 and myocardial ischemia symptoms. Further research concentrating on myocardial perfusion reserve could confirm the clinical utility of this novel CCTA parameter, offering a more comprehensive understanding of its role in HCM management. Lastly, all CT measurements were conducted during the mid-diastolic phase (RR interval, 75%–78%), thereby preventing an analysis of how the cardiac cycle affects coronary artery diameter variation.

In conclusion, we have shown the potential clinical utility of CCTA-derived coronary artery dimension-adjusted subtended myocardial mass V_{sub}/MLA^2 in patients with HCM. This measure may more accurately reflect the balance between myocardial blood supply and demand, shedding light on the occurrence of demand angina in HCM patients without obstructive CAD.

Availability of Data and Material

The datasets generated or analyzed during the study are available from the corresponding author on reasonable request.

Conflicts of Interest

The authors have no potential conflicts of interest to disclose.

Author Contributions

Conceptualization: Min Jae Cha. Data curation: Min Jae Cha. Formal analysis: Min Jae Cha. Funding acquisition: Min Jae Cha. Investigation: Min Jae Cha, Hyewon Choi. Methodology: Min Jae Cha. Project administration: Min Jae Cha. Resources: Min Jae Cha, Hyewon Choi. Software: Min Jae Cha. Supervision: Min Jae Cha. Validation: Min Jae Cha. Visualization: Min Jae Cha. Writing—original draft: Min Jae Cha, Jung Han Woo. Writing—review & editing: Min Jae Cha, Jung Han Woo.

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Funding Statement

Bracco is providing the funding for this study.

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https://doi.org/10.3348/kjr.2024.1143