Exploring the link between myocardial bridging and left ventricular hypertrophy: Congenital factors or remodeling?

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DOI: 10.33963/v.phj.102044

Received:
May 27, 2024

Accepted:
August 12, 2024

Early publication date:
August 12, 2024

ABSTRACT

Background: Myocardial bridging (MB) was considered a congenital anomaly and was found with increased frequency in coronary computed tomography angiography. Some case studies reported an association of MB with various cardiomyopathies. However, the association between MB severity and left ventricular hypertrophy remains unclear.

Aims: This cross-sectional study aimed to evaluate whether myocardial bridge is related to left ventricular hypertrophy in patients referred for coronary computed tomography angiography (CCTA).

Methods: This cross-sectional study included 227 patients (age 53.2 [11.1] years, 48% female) who underwent 640-slice CCTA and were diagnosed with MB. MB severity was measured as MB muscle index (MMI) (MB length × MB thickness), and left ventricular hypertrophy (LVH) was assessed with transthoracic echocardiography.

Results: MB segments were detected in all patients on the left anterior descending artery. CCTA was performed to exclude coronary artery disease in most patients (90%; n = 206). Eighty-two (36.1 %) had LVH, and MMI was significantly higher in patients with LVH than those without LVH (27.3 [19.5–38.9] vs. 24 [13.8–37.1]; $P = 0.022$, respectively). There was a positive correlation between the left ventricular mass index and myocardial bridge length (r = 0.414; $P = 0.001$), MB index (r = 0.310; $P < 0.001$), and the age of the patients (r = 0.191; $P = 0.004$). MB thickness and MMI were also positively correlated with relative wall thickness.

Conclusion: MB is a common finding, and its severity is associated with left ventricular hypertrophy in patients undergoing CCTA.

Key words: coronary computed tomography angiography, echocardiography, left ventricular hypertrophy, myocardial bridge muscle index, myocardial bridging

INTRODUCTION

Myocardial bridging (MB) is a congenital anomaly characterized by the intramyocardial course of major epicardial coronary arteries [1]. The left anterior descending coronary artery (LAD) is most frequently involved [2]. The prevalence reported is highly variable; some studies have reported ~2% in angiography, but it can be as high as 80% in autopsy series [2]; with the increased use of coronary computed tomography angiography (CCTA) in recent years, the number of MB patients reported has been increasing. Advanced multi-slice scanners became a useful non-invasive method to identify MB, and the reports have shown a higher prevalence of 18%–58% [3–5]. Until recently, MB has been considered a benign condition. However, it is potentially associated with various clinical findings such as myocardial ischemia, arrhythmia, acute myocardial infarction, coronary spasm, and sudden death [6–8]. Previous studies have reported that MB was more common in cardiac diseases associated with left ventricular hypertrophy (LVH), such as aortic stenosis or hypertrophic cardiomyopathy (HCM) [9, 10]. Especially, HCM patients have a higher prevalence than the general population, with rates up to 30% [11].

WHAT'S NEW?

This cross-sectional study investigated the relationship between myocardial bridge (MB) and left ventricular hypertrophy. It included 227 patients who underwent 640-slice coronary computed tomography angiography and transthoracic echocardiography. Myocardial severity was measured as the myocardial bridge muscle index with the guidance of autopsy studies. Despite the lack of significant differences in MB length and thickness between the groups, it is noteworthy that the MB index was higher in patients with left ventricular hypertrophy. Furthermore, the left ventricular mass index positively correlated with the patients' MB length, myocardial bridge index, and age. Relative wall thickness was positively correlated with MB thickness and myocardial bridge index. These findings are essential for evaluating patients with myocardial bridges undergoing coronary computed tomography angiography.

The MB muscle index (MMI [the Myocardial Bridge Muscle Index] = MB length × MB depth) was introduced by Ishikawa et al. [12] from autopsy studies as a new concept to assess the significance of MBs. They found that the MMI was greater in patients with higher LAD plaque burden, and they suggested that a larger MB mass likely resulted in greater hemodynamic compromise. The length and depth of MB can be easily measured by CCTA [13]. Forsdahl et al. [13] showed that the MMI provided a non-invasive insight into the hemodynamic significance of the bridge. This study aimed to assess the association between MB severity and LVH in adult patients. The MMI was determined with 640-slice CCTA, and the degree of LVH was measured using transthoracic echocardiography (TTE).

METHODS

Study population

This cross-sectional study included patient data from two years (January 2021–December 2022). The patients were referred for 640 CCTA slices and had already undergone TTE in the previous six months. The recruitment scheme of the study population is shown in Figure 1. Altogether, 2133 patients who underwent CCTA were screened for initial work. Of the 369 patients, 102 did not have recent TTE, and 7 CCTA scans could not be analyzed due to poor image quality. Lastly, 33 patients were excluded due to valvular heart disease (intermediate or severe valvular disease) or cardiomyopathy, including HCM with or without outflow tract obstruction, restrictive-dilated cardiomyopathy, infiltrative cardiac processes (e.g., amyloidosis, Fabry disease, Danon disease). Thus, a total of 227 patients with MB were available for the analysis. The study was conducted following the Helsinki Declaration and was approved by the Institutional Review Board.

Coronary computed tomography angiography

CCTA of coronary arteries was performed using a 640-slice scanner of Aquilion ONE Genesis Edition (Canon Medical Systems, Otawara, Japan). The scan was performed at

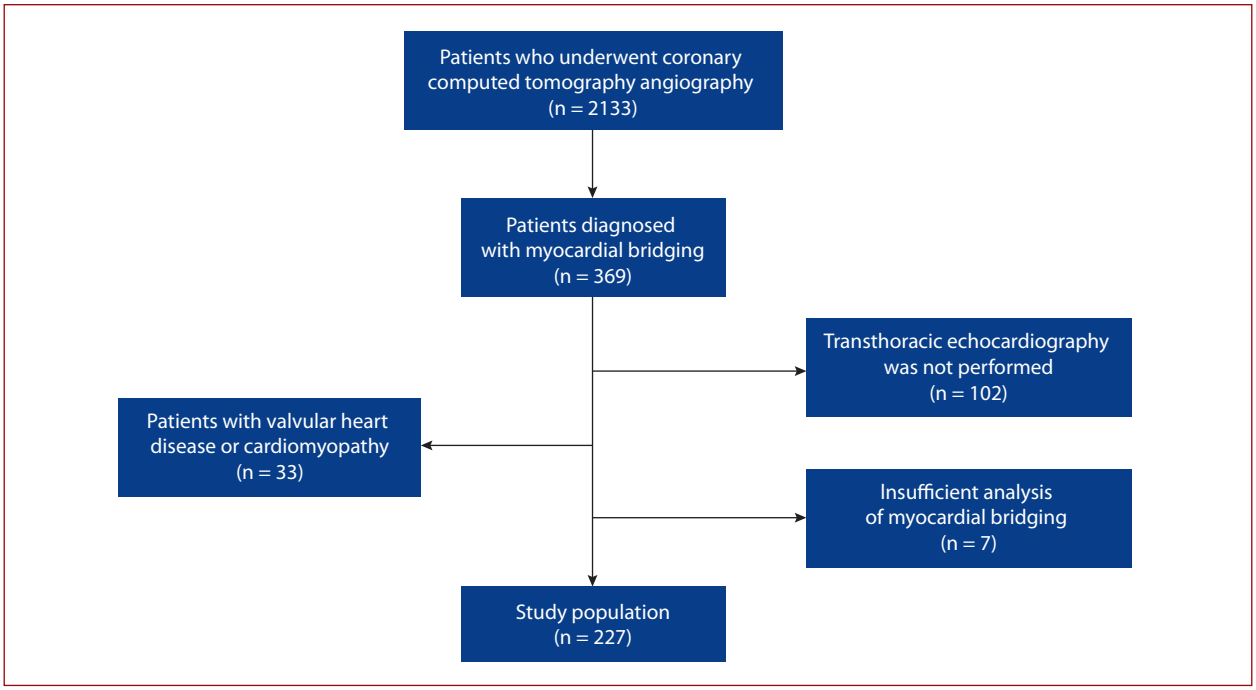

Figure 1. Flow chart of the study

100 kV-120 kV (adapted to body weight) and 250–500 mA (automatically determined based on the patient's scanogram data). The gantry rotation time was 275 ms, and the scan collimation was in the range of 0.5 × 100 mm to 0.5 × 160 mm (manually planned, based on the size and location of the heart area to be scanned). The field of view was set between 200–270 mm, covering the area from the tracheal bifurcation to the base of the heart. Before scanning, the patients whose heart rate was >100 beats/min were given oral or intravenous beta-blockers or calcium channel blockers.

A non-contrast-enhanced coronary tomography was performed on each patient to assess Agatston calcium score [14]. The coronary artery calcium score was calculated by multiplying the area of lesions with a density of ≥130 Hounsfield units (HU) with a density factor derived from the maximum density of each lesion (1 for 130–199 HU, 2 for 200–299 HU, 3 for 300–399 HU, and 4 for lesions ≥400 HU). The total score was calculated by summing up the scores of each lesion.

We injected 50–80 milliliters of iodinated contrast medium (350 mgI/ml) intravenously at 5 ml/sec, followed by 20 ml of saline. The reconstruction of images was performed in a synchronized manner with an electrocardiogram in prospective (40–70 beats per minute [BPM]), modulated (70–90 BPM), and retrospective (90–120 BPM) scans. Images at the 75% phase of the RR interval and the best phase automatically generated by the software were provided automatically by the device. In cases where the optimal phase was not achieved, the operator utilized the software to create the most appropriate phase, beat, and functional information.

The images were transferred to a workstation (Vitrea Advanced, US) for image reconstruction. Axial images, multiplanar reconstruction, three-dimensional volume rendering, curved planar reformat, cinematic rendering using Global Illumination Rendering, and three-dimensional maximum intensity projection images were used to assess the properties of myocardial bridging.

We accepted an intramyocardial segment of the coronary artery when a segment of the coronary artery was covered by the myocardium and the complete myocardial encasement when defining MB on axial multiplanar reconstruction. We measured cross-sectional images for each tunneled segment of bridging length (at best projection view, from the entrance point to the exit point) and depth (maximal thickness of the myocardial layer from the epicardial surface) (Figure 2). MMI was calculated as MB length × MB thickness [12]. The segment with MB, atherosclerotic plaque proximal to MB, and plaque at the site of MB was also noted. The interpretations of each patient's CCTA images were performed by a radiologist experienced in cardiovascular imaging.

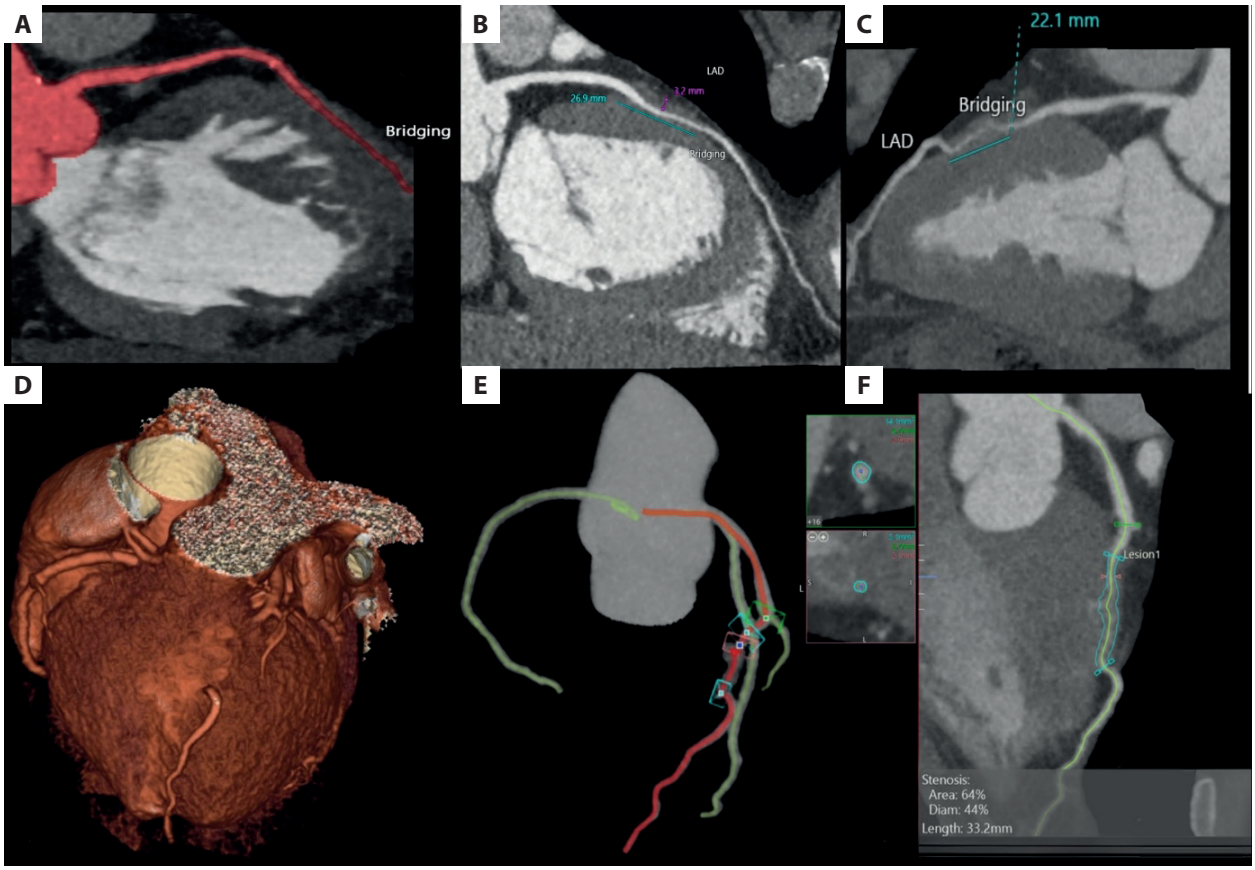

Figure 2. Coronary computed tomography angiography showing myocardial bridging (**A**), measurement length and thickness of the myocardial bridging segment in various computed tomography images (**B**, **C**), 3-dimensional volume rendering image of myocardial bridging (**D**), assessment of the bridging stenosis and length (**E**, **F**)

Echocardiography

TTE was performed on each participant with a commercially available Doppler echocardiograph (VIVID 7, General Electric-Vingmed Ultrasound, Horten, Norway) using a 3.0-MHz transducer. The echocardiographic examination was conducted and examined by a sonographer (B.A.) blinded for CCTA results. Echocardiographic imaging was performed following standardized procedures outlined by the American Society of Echocardiography and the European Association of Cardiovascular Imaging [15]. Measurements were obtained from a parasternal long-axis view with 2D or M-mode (preferably) recordings approximately at the mitral valve leaflet tips. Left ventricular internal dimension diastole (LVIDd), inter-ventricular septum thickness in diastole (IVSd), posterior wall thickness in diastole, left ventricular internal dimension in systole, inter-ventricular septum thickness in systole, posterior wall thickness in systole, left atrial diameter, and left ventricular ejection fraction were calculated. Left ventricular mass (LVM) (g) was calculated as: 0.8 (1.04 [(LVIDd + PW + IVSd)^3 (LVIDd)^3]) + 0.6 [16, 17]. Body surface area (BSA) calculated according to the DuBois Formula; BSA (m^2) = 0.007184 × height (cm) 0.725 × weight (kg) 0.425. The left ventricular mass index (LVMi) was defined by indexation of LVM to BSA. Relative wall thickness (RWT) was calculated two times posterior wall thickness divided by the left ventricular diastolic diameter. LVH was described as an increased LVMi greater than 95 g/m^2 in women and 115 g/m^2 in men [16, 17].

Data collection and definitions

The patient's clinical information was collected upon admission, including demographic data, biochemical data, lifestyle, medical history, use of medications, and exercise test results. Diabetes at baseline was defined as fasting blood glucose levels of ≥126 mg/dl and/or a history of diabetes, while hypertension was defined as blood pressure levels of ≥140/90 mm Hg on admission and/or a previous diagnosis of hypertension. Dyslipidemia was determined according to the latest guidelines or treatment with lipid-lowering drugs, considering patient risk factors. Additionally, a family history of coronary artery disease (CAD) was determined when CAD was found in first-degree relatives aged <55 (male) or <65 (female) years.

Statistical analysis

All statistical analyses were performed using the SPSS 22.0 software package (IBM Corp, Armonk, NY, US). Categorical variables were presented as frequencies and percentages and were compared using the χ^2 or Fisher's exact test. Continuous variables with normal distribution were presented as means (standard deviations) and compared using the two-sample t-test; skewed data were presented as medians and ranges (IQR), and the Mann–Whitney U test was used for analysis. The Spearman correlation was used to analyze the relationship between LVMi, RWT, MB thickness, MB length, MMI, and age. Statistical significance was set as a 2-tailed *P*-value of <0.05.

RESULTS

The mean age of the patients was 53.2 (11.1) years, and 48 % (n = 118) were female. Two hundred and twenty-seven patients with myocardial bridging were included in the study. The prevalence of atherosclerotic plaques proximal to the MB of the LAD was 49% (20/41) in patients with atherosclerosis. Of the 227 patients, 82 (36.1%) had LVH, and 90.7%) of patients underwent CCTA due to the exclusion of CAD. MB segments were detected on the LADs in all patients. Baseline patient characteristics are presented in Table 1. No significant differences were found in sex, BSA, diabetes, CAD, hypertension, dyslipidemia, family history for CAD, chronic kidney disease, smoking status, treadmill exercise test results, anterior ischemia in myocardial perfusion imaging, and exercise status in a week. Use of beta-blockers (36.6% vs. 20.0%; *P* = 0.006), statins (15.9% vs. 5.5%; *P* = 0.010), and angiotensin-converting enzyme/ /angiotensin receptor blockers (39.0% vs. 23.4%; *P* = 0.013) were higher in LVH patients. LVH patients were older than those without LVH (56.3 [11.5] vs. 51.5 [10.5] years; *P* = 0.002, respectively), and the body mass index was higher in LVH patients than in those without LVH (30.7 [5.3] vs. 27.9 [4.4] kg/m^2; *P* <0.001, respectively).

CCTA findings and anatomic properties of MB are shown in Table 2. There was no difference in MB length and thickness. MMI was higher in LVH patients than in those without LVH (27.3 [19.5–38.9] vs. 24 [13.8–37.1]; *P* = 0.022, respectively). The Agatson score, ratio of plaque proximal to MB, and calcific plaque ratio at MB did not differ between the groups. CCTA was performed to exclude CAD in most patients (n = 206, 90%). The location of the bridging segment was similar between the groups. Almost all the tunneled segments were in the mid and distal part of the LAD.

The echocardiographic findings of the study subjects are shown in Table 3. Left atrial diameter, LVIDd, IVSd, posterior wall thickness in diastole, posterior wall thickness in systole, and left ventricular internal dimension in systole were higher in LVH patients (*P* <0.005). Left ventricular mass (242.5 [19.4] vs. 147.9 [39.7] g; *P* <0.001) and left ventricular mass index (125.8 [26.6] vs. 78.7 [18.4] g/m^2; *P* = 0.001) were higher in LVH patients than in those without LVH. RWT was higher in patients with LVH than those without LVH (0.51 [0.09] vs. 0.46 [0.09] cm; *P* <0.001). Left ventricular ejection fraction and inter-ventricular septum thickness in systole were not different between the groups.

There were positive correlations between the LVMi and MB length (r = 0.414; *P* = 0.001), MMI (r = 0.310; *P* <0.001), and the age of the patients (r = 0.191; *P* = 0.004) (Figure 3). No significant correlation was found between the left ventricular mass index and myocardial bridge thickness. There was also a positive correlation of RWT with MMI (r = 0.241; *P* = 0.001), and myocardial bridge thickness (r = 0.156; *P* = 0.028) (Table 4)

Table 1. Baseline characteristics and admission properties of the patients with myocardial bridging

Parameters	LVH (+) (n = 82)	LVH (–) (n = 145)	*P*-value
Age, years, mean (SD)	56.3 (11.5)	51.5 (10.5)	**0.002**
Male, n (%)	33 (40.2)	76 (52.4)	0.078
Body surface area, m^2, mean (SD)	1.92 (0.18)	1.88 (0.20)	0.092
Body mass index, kg/m^2, mean (SD)	30.7 (5.3)	27.9 (4.4)	**<0.001**
Diabetes, n (%)	29 (35.4)	47 (32.4)	0.651
Coronary artery disease, n (%)	2 (2.4)	4 (2.8)	0.885
Hypertension, n (%)	45 (54.9)	66 (45.5)	0.175
Dyslipidemia, n (%)	27 (32.9)	36 (24.8)	0.191
Family history for CAD, n (%)	25 (30.5)	51 (35.2)	0.472
Chronic kidney disease, n (%)	2 (2.4)	4 (2.8)	0.885
Smoking, n (%)			0.139
Never	42 (51.2)	56 (38.6)	
Current	20 (24.4)	51 (35.2)	
Former	20 (24.4)	38 (26.2)	
Treadmill exercise test, n (%)			0.810
Positive	3 (3.7)	9 (6.2)	
Negative	15 (18.3)	29 (20.0)	
Non-diagnostic	7 (8.5)	10 (6.9)	
MPS anterior ischemia (number)[a]	4/8	5/10	0.126
Drugs, n (%)			
Beta blocker	30 (36.6)	29 (20.0)	**0.006**
Calcium channel blocker	14 (17.1)	21 (14.5)	0.604
Nitrates	1 (1.2)	1 (0.7)	0.682
Acetylsalicylic acid	22 (26.8)	25 (17.2)	0.217
Trimetazidine	2 (2.4)	1 (0.7)	0.268
ACEI/ARB	32 (39.0)	34 (23.4)	**0.013**
Statins	13 (15.9)	8 (5.5)	**0.010**
Exercise status in a week, n (%)			0.370
None	55 (67.1)	83 (57.2)	
Only one day	6 (7.3)	12 (8.3)	
Two-three day	14 (17.1)	24 (16.6)	
Five-day	4 (4.9)	12 (8.3)	
All days	3 (3.7)	14 (9.7)	

[a]Number means the patient was performed MPS and positive anterior ischemia was detected

Abbreviations: ACE, angiotensin converting enzyme; ARB, angiotensin receptor blocker; CAD, coronary artery disease; MPS, myocardial perfusion imaging; SD, standard deviation

Table 2. Coronary computed tomography findings and anatomic properties of myocardial bridges

Parameters	LVH (+) (n = 82)	LVH (–) (n = 145)	*P*-value
MB length, mm, mean (SD)	15.9 (4.3)	14.7 (6.1)	0.142
MB thickness, mm, mean (SD)	1.8 (0.7)	1.9 (0.9)	0.420
MB muscle index, median (IQR)	27.3 (19.5–38.9)	24 (13.8–37.1)	0.022
Agatson score, median (IQR)	0 (0–10)	0 (0–27.5)	0.119
Plaque proximal to MB, n (%)	28 (26.2)	38 (34.1)	0.206
Calcific plaque at MB, n (%)	3	–	–
Indication of CCTA, n (%)			0.459
Exclusion of CAD	76 (92.7)	130 (89.7)	
Myocarditis/pericarditis	2 (2.4)	2 (1.4)	
Arrythmia	4 (4.9)	13 (9.0)	
Location of MB			0.753
Proximal LAD	–	1 (0.7)	–
Mid-LAD	40 (48.8)	70 (48.3)	
Distal LAD	42 (51.2)	74 (51.0)	

Abbreviations: CAD, coronary artery disease; CCTA, coronary computed tomography angiography; LAD, left anterior descending artery; MB, myocardial bridging

Table 3. Echocardiographic findings of the study subjects and assessment of left ventricular hypertrophy with left ventricular mass index

Parameters	LVH (+) (n = 82)	LVH (–) (n = 145)	*P*-value
Left ventricular ejection fraction, median (IQR)	65 (60–72)	68 (65–71)	0.119
Left atrial diameter, cm	4.1 (0.5)	3.6 (0.6)	<0.001
LVIDd, cm	4.89 (0.56)	4.45 (0.42)	<0.001
IVSd, cm	1.25 (0.20)	1.06 (0.19)	0.005
PWd, cm	1.23 (0.17)	1.10 (0.27)	0.004
LVIDs, cm	3.02 (0.65)	2.58 (0.47)	<0.001
IVSs, cm	1.78 (0.22)	1.74 (0.61)	0.544
PWs, cm	1.76 (0.21)	1.54 (0.22)	0.008
Left ventricular mass, g	242.5 (19.4)	147.9 (39.7)	<0.001
Left ventricular mass index, g/m^2	125.8 (26.6)	78.7 (18.4)	0.001
Relative wall thickness, cm	0.51 (0.09)	0.46 (0.09)	<0.001

Data expressed as mean (standard deviation)

Abbreviations: IVSd, interventricular septum in diastole; IVSs, interventricular thickness in systole; LVIDd, left ventricular internal diameter in diastole; LVIDs, left ventricular internal dimension in systole; PWd, left ventricular posterior wall thickness in diastole; PWs, posterior wall thickness in systole

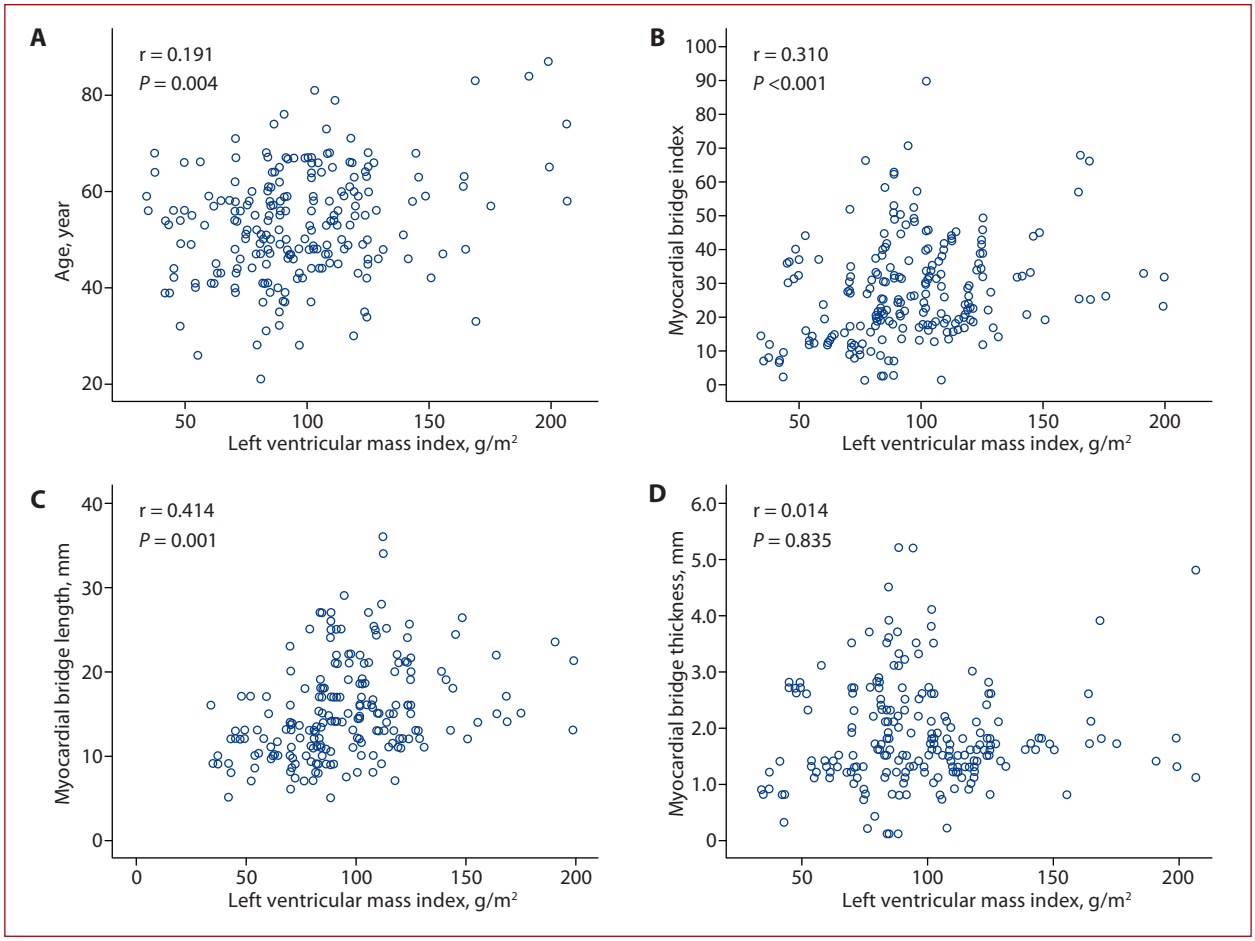

Figure 3. Correlation between and left ventricular mass index and age (**A**), myocardial bridge index (**B**), myocardial bridge length (**C**), and myocardial bridge thickness (**D**)

DISCUSSION

The main finding from this study is that among patients who were referred for CCTA and had myocardial bridging, we observed an association between MMI and LVH. In this study: (1) the MMI was higher in patients with LVH despite the non-significant difference in MB length and thickness between the groups, (2) the LVMi positively correlated with the patients' MB length, MMI, and age, (3) RWT was positively correlated with MB thickness and MMI.

The frequency of MB was found to be 15%–85% in some autopsy series [18]. However, the difference between CCTA and invasive coronary angiography [19] varies significantly. Since it is mainly necessary to see systolic compression to diagnose MB, defined as a milking-like effect, in invasive coronary angiography, the incidence of CCTA is significant [20]. The symptomatic patients referred for CCTA, who had no obstructive CAD, had a relatively high (35%) prevalence of MB [5, 21]. Increased spatial and temporal resolution with

Table 4. Correlation analysis of left ventricular mass index and relative wall thickness with various parameters

Parameters	r-value	*P*-value
Left ventricular mass index		
Myocardial bridge length	0.414	0.001
Myocardial bridge thickness	0.014	0.835
Myocardial bridge muscle index	0.310	<0.001
Age	0.191	0.004
Relative wall thickness		
Myocardial bridge length	0.114	0.115
Myocardial bridge thickness	0.156	0.028
Myocardial bridge muscle index	0.241	0.001
Age	0.191	0.091

a higher detector system might lead to a better diagnosis of MB [22].

MB follow-up and treatment strategies attract more attention due to the increased number of diagnosed patients. As in our study, MB primarily affects the left anterior descending coronary artery [23, 24]. MB causes systolic compression of the coronary artery in various degrees, and it remains intact throughout diastole in most patients. The symptoms of bridging could vary in patients. The standard view on this subject is that these are innocent findings that do not have hemodynamic significance and do not cause ischemia. A delay in diastolic relaxation might cause ischemia and anginal symptoms [25].

Whether MB is congenital or acquired and what factors contribute to its development remains uncertain [26]. Cardiac disorders that are included with LVH are more commonly associated with MB [27], and the occurrence of MB in HCM patients is higher compared to that in the general population [28, 29]. De Gregorio et al. [30] reported a case of hypertensive LVH associated with large MB. In another case report, a patient with hypertension was admitted with myocardial infarction due to MB of the LAD with total systolic compression of the artery. The authors claimed that LVH might contribute to the genesis of ischemia [31]. An autopsy study conducted by Basso et al. [32] reported that MB was more common in HCM than in patients who died from a variety of non-HCM causes. In addition, it has been observed that MBs tend to occur quite frequently among patients with HCM, even those who have experienced sudden death [32]. In contrast to our study, the presence of MB was unrelated to LV wall thickness and age. However, the authors did not assess the association between the degree of MB and LVH.

Patients with septal hypertrophy have deep or extensive MB [29]. Notably, the thickness of the left ventricular wall at the proximal interventricular septum and the extent of asymmetrical septal hypertrophy have been found to be notably greater in pediatric patients with bridging. The correlation between left ventricular outflow tract obstruction and bridging is further evidenced by the significantly elevated left ventricular systolic pressure observed in affected children [10].

The other question is whether MB is an independent risk factor for ischemia and sudden cardiac death or merely an indicator of the severity of LVH, which has been a matter of ongoing debate [26]. Sorajja et al. [33] found that the patients with HCM and concomitant MB do not have an increased risk of cardiac death or SCD. Furthermore, there is strong evidence from small series and case reports suggesting a link between MB and sudden death or myocardial infarction in children and young adults [34]. The frequent reporting of a congenital finding in both autopsy series and adult imaging studies is indeed an intriguing observation. One possible pathophysiological mechanism is that in regions predisposed to myocardial bridging, the growth of cells around the coronary artery may lead to the vessel being encased, which, when subjected to systolic compression, might trigger the development of advanced MB. However, this pathophysiological mechanism remains purely hypothetical. A more accurate explanation might be that MB is an anomaly found incidentally or as a normal variant without a specific pathophysiological mechanism.

This study may have some limitations. First, the study was performed on a relatively small patient group. Long-term changes in the left ventricular or cardiovascular events should be investigated in larger cohorts. Another limitation was the assessment of imaging by a single radiologist.

CONCLUSION

MB can be detected in a substantial proportion of patients undergoing CCTA. This cross-sectional study showed an association between LVH and myocardial bridge severity.

Article information

Conflict of interest: None declared.

Funding: None.

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