Influence of myocardial bridge on atherosclerotic plaque distribution and characteristics evaluated by near-infrared spectroscopy intravascular ultrasound

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Abstract

Background This study aims to clarify whether myocardial bridge (MB) could influence atherosclerotic plaque characteristics assessed using near-infrared spectroscopy-intravascular ultrasound (NIRS-IVUS) imaging.

Methods One hundred and sixteen patients who underwent percutaneous coronary intervention (PCI) using NIRS-IVUS imaging were included. MB was defined as an echo-lucent band surrounding left anterior descending artery (LAD). In MB patients, LAD was divided into three segments: proximal, MB, and distal segments. In non-MB patients, corresponding three segments were defined based on the average length of the above segments. Segmental maximum plaque burden and lipid content derived from NIRS-IVUS imaging in the section of maximum plaque burden were evaluated in each segment. Lipid content of atherosclerotic plaque was evaluated as lipid core burden index (LCBI) and maxLCBI4mm. LCBI is the fraction of pixels indicating lipid within a region multiplied by 1000, and the maximum LCBI in any 4-mm region was defined as maxLCBI4mm.

Results MB was identified in 42 patients. MB was not associated with maximum plaque burden in proximal segment. LCBI and maxLCBI4mm were significantly lower in patients with MB than those without in proximal segment. Multivariable analysis demonstrated both MB and maximum plaque burden in proximal segment to be independent predictors of LCBI in proximal segment.

Conclusions Lipid content of atherosclerotic plaque assessed by NIRS-IVUS imaging was significantly smaller in patients with MB than those without. MB could be considered as a predictor of lipid content of atherosclerotic plaque when assessed by NIRS-IVUS imaging.

Keywords Myocardial bridge · Near-infrared spectroscopy intravascular ultrasound imaging · Atherosclerosis

Introduction

Myocardial bridge (MB) is a common anatomic variant of a muscle bundle overlying the intramyocardial segment of an epicardial coronary artery. The prevalence of MB varies based on the detection methods, such as autopsy, angiography, computed tomography, and intravascular imaging, and its location is mostly in the left anterior descending artery (LAD) [1–3]. It has been previously reported that MB accelerates coronary atherosclerosis in the segment proximal to MB, whereas the development of atherosclerosis is significantly suppressed in LAD intima beneath MB [2]. On the other hand, several studies with multi-slice computed tomography have demonstrated that MB has no relationship with the progression of atherosclerotic plaque [4, 5]. Thus, controversy about the association between MB and atherosclerotic plaque formation exists. Moreover, it is unclear whether MB has an impact on plaque characteristics in the segments proximal to, beneath, and distal to MB.

Intravascular imaging, such as intravascular ultrasound (IVUS), can directly observe atherosclerotic plaque and estimate plaque vulnerability in vivo [6–8], and there are a few studies investigating the association of MB with plaque...
distribution and characteristics [9, 10]. However, these studies utilized only gray-scale IVUS imaging. Near-infrared spectroscopy (NIRS)-IVUS consists of an ultrasound transducer and a scanning near-infrared laser to detect the lipid content of atherosclerotic plaque. Previous studies have shown that a large amount of lipid core plaque (LCP) detected by NIRS-IVUS can predict future adverse cardiovascular events [11, 12]. Thus, the aim of this study was to clarify whether MB could influence atherosclerotic plaque characteristics as assessed using NIRS-IVUS.

Materials and methods

Study design and population

The present study was a single-center, retrospective observational analysis, conducted between March 2017 and May 2019 at Chiba University Hospital. Patients who underwent percutaneous coronary intervention (PCI) for any coronary artery disease with NIRS-IVUS imaging of LAD were considered eligible for the present study. In patients who underwent PCI, all participants with significant visual stenosis (> 50% on angiography) but no symptoms related to myocardial ischemia (e.g., exertional chest pain and shortness of breath) received some kinds of ischemic tests. Those who did not receive any ischemic tests had symptoms related to myocardial ischemia with significant visual stenosis or had no symptoms with severe visual stenosis (> 90% on angiography). To keep the accuracy of the evaluation for lipid accumulation detected by NIRS, patients were excluded if they had pre-existing coronary artery stent or severe calcification in the segment proximal to MB. Written informed consent for examination was obtained from all patients, and the ethical committee of Chiba University approved this study.

NIRS-IVUS imaging and analysis

Systematic NIRS-IVUS imaging of the LAD was performed using the Makoto™ Intravascular Imaging System (NIPRO Corporation, Osaka, Japan), and was acquired from as distal as possible, with motorized transducer pullback speed of 0.5 mm/second. In cases where PCI for LAD was indicated, NIRS-IVUS imaging was acquired before any intervention if the severity of lesion allowed crossing of the imaging catheter or after balloon dilatation if needed. MB was defined as an echo-lucent band partially surrounding the LAD. In MB patients, LAD was divided into three segments: proximal segment (LAD ostium to MB entrance), MB segment, and distal segment. In non-MB patients, corresponding three segments were defined based on the average length of the above segments. To quantify the amount of lipid content of atherosclerotic plaque, lipid core burden index (LCBI) was evaluated in each segment. LCBI is the fraction of pixels indicating lipid within a region multiplied by 1000 and LAD was scanned for the maximum LCBI in any 4-mm region, which was defined as maxLCBI_{4mm}.

Grayscale IVUS analysis

All grayscale IVUS images were analyzed using echo Plaque 3.0 (INDEC Systems, Los Altos, CA, USA) by experienced investigators, and all measurements were conducted in each of the three segments. Vessel and lumen areas were measured at the leading edge of the external elastic membrane (EEM) and luminal borders. Plaque area was calculated as vessel area minus lumen area. Plaque burden was calculated as plaque area divided by vessel area. Maximum plaque burden, and vessel and lumen areas in the cross-section of maximum plaque burden were obtained. In patients with MB, thickness of MB halo and maximum arterial compression were evaluated. Arterial compression was calculated as a decrease in EEM-cross sectional area (CSA) at systole divided by EEM-CSA at end-diastole, which was represented as a percentage. Atherosclerotic plaque calcification severity was assessed as the largest arc of calcium in the cross-section of maximum plaque burden, then further classified as calcification score: zero-point (0°), one-point (1° to 90°), two-point (91° to 180°), three-point (181° to 270°) or four-point (271° to 360°).

Statistical analysis

Statistical analysis was performed with JMP® 13.2.0 (SAS Institute Inc., Cary, NC, USA). Data are expressed as mean±SD or frequency (%). Difference between baseline characteristics was assessed by Student t test or Wilcoxon rank-sum test and Fisher’s Exact test or Pearson’s X^2 test in terms of continuous variables and categorical variables, respectively. Univariable analysis was performed using linear regression analysis of rank-transformed outcomes. The associated variables on univariable analysis (p < 0.10) were included in multivariable analysis. Multiple linear regression analysis of rank-transformed outcomes was used for multivariable analysis. The partial regression coefficient (β) was used to express the contribution of each variable included in the multivariable analysis. A value of p < 0.05 was considered statistically significant.

Results

Baseline characteristics

A total of 136 patients underwent PCI using NIRS-IVUS imaging during the study period. Among these, NIRS-IVUS
 imaging of the proximal segment was not available in 20 patients because of the severe calcification and pre-existing coronary artery stent. Thus, 116 patients were included in this study (male, 80%; age, 69.4 ± 10.9 years). MB was identified in 42 out of 116 patients (36%). Baseline characteristics are shown in Table 1. Of these 116 patients, 54 (47%), 32 (27%), and 30 (26%) patients had silent myocardial ischemia (SMI), stable angina pectoris (SAP), and acute coronary syndrome (ACS), respectively. The presentation of coronary artery disease (CAD) did not significantly differ between the patients with and without MB. Patients with MB were significantly younger and had less NIRS-IVUS imaging for LAD as a culprit vessel of PCI performed. Other characteristics were similar between patients with and without MB.

Clinical data in patients who underwent PCI were shown in Table S1. Among the 78 patients who underwent PCI to LAD, 8 (43%) and 14 (25%) of patients with and without MB received some kinds of ischemic tests. No significant difference was found in the portion of patients who received each ischemic test and the location of culprit lesion between the two groups. During the PCI procedure, 9 (43%) and 29 (51%) patients with and without MB needed balloon dilatation before IVUS. The balloon size and length used in the procedure were similar between the two groups, whereas total balloon inflation time was significantly shorter in patients with MB than those without.

**Grayscale IVUS analysis**

IVUS findings are shown in Table 2. Maximum plaque burden was significantly greater in the proximal segment than in other segments. In comparison between patients with and without MB, maximum plaque burden was similar in the proximal and distal segments, whereas it was significantly smaller in the MB segment compared with the corresponding mid segment in patients without MB (Fig. 1).

### Table 1 Baseline characteristics

|                | MB n=42 | Non-MB n=74 | P value |
|----------------|---------|-------------|---------|
| Age (years)    | 66.8±11.4 | 70.9±10.5  | 0.051   |
| Male           | 37 (88%) | 56 (76%)   | 0.107   |
| BMI            | 25.0±3.9 | 24.0±3.7   | 0.177   |
| Hypertension   | 32 (76%) | 45 (61%)   | 0.092   |
| Diabetes mellitus | 16 (38%) | 33 (45%)  | 0.496   |
| Dyslipidemia   | 33 (79%) | 49 (66%)   | 0.160   |
| Current smoker | 5 (12%)  | 14 (19%)   | 0.327   |
| Prior myocardial infarction | 8 (19%) | 19 (26%) | 0.417   |
| Prior PCI      | 13 (31%) | 20 (27%)   | 0.653   |
| Prior CABG     | 1 (2%)   | 1 (1%)     | 0.682   |
| eGFR (ml/min/1.73 m2) | 71.5±21.5 | 68.6±21.0 | 0.496   |
| BNP (pg/ml)    | 50.3±99.5 | 156.8±406.1 | 0.098  |
| Admission LVEF (%) | 58.5±10.5 | 56.0±13.7 | 0.333   |
| Stable angina pectoris | 14 (33%) | 18 (24%) | 0.297   |
| Acute coronary syndrome | 9 (22%) | 21 (29%) | 0.411   |
| Target vessel for PCI | 21 (50%) | 57 (77%) | 0.003   |

**Medical treatment at discharge**

| Antiplatelet | 42 (100%) | 74 (100%) | 0.682 |
| Anticoagulant | 4 (10%)  | 8 (11%)   | 0.827 |
| Statin        | 38 (90%) | 68 (92%)  | 0.794 |
| ACE-I or ARB  | 26 (62%) | 40 (54%)  | 0.412 |
| β blocker     | 19 (45%) | 24 (32%)  | 0.170 |
| Calcium channel blocker | 21 (50%) | 35 (47%) | 0.780 |
| Diuretic      | 2 (5%)   | 13 (18%)  | 0.048 |
| MRA           | 1 (2%)   | 6 (8%)    | 0.213 |
| OHA           | 17 (40%) | 31 (42%)  | 0.563 |

Values are mean ± standard deviation or n (%)

**ACE-I** angiotensin converting enzyme inhibitor, **ARB** angiotensin receptor blocker, **BNP** brain natriuretic peptide, **LVEF** left ventricular ejection fraction, **MRA** mineralocorticoid receptor antagonist, **OHA** oral hypoglycemic agent, **PCI** percutaneous coronary intervention

### Table 2 NIRS-IVUS findings

|                | MB n=42 | Non-MB n=74 | P value |
|----------------|---------|-------------|---------|
| Proximal segment |         |             |         |
| Vessel area (mm²) | 13.9±4.5 | 13.8±4.5  | 0.940   |
| Lumen area (mm²)  | 4.5±2.3  | 4.2±2.3    | 0.462   |
| Maximum plaque burden (%) | 67.4±11.4 | 69.5±11.9 | 0.364   |
| LCBI             | 101.5±81.6 | 151.8±103.7 | 0.009   |
| MaxLCBI_{mm}     | 338.1±231.1 | 429.1±212.6 | 0.034   |
| Calcification Score | 1.0±0.8 | 1.3±1.0 | 0.130   |
| MB segment       |         |             |         |
| Vessel area (mm²) | 8.6±2.9  | 9.0±2.8    | 0.504   |
| Lumen area (mm²)  | 5.0±2.0  | 4.5±2.7    | 0.288   |
| Maximum plaque burden (%) | 40.6±12.9 | 51.4±18.1 | 0.001   |
| LCBI             | 19.1±44.1 | 87.7±97.1 | <0.001  |
| MaxLCBI_{mm}     | 44.1±98.5 | 219.2±203.7 | 0.001   |
| Calcification Score | 0.4±0.6 | 0.6±0.7 | 0.296   |
| Distal segment   |         |             |         |
| Vessel area (mm²) | 8.4±2.5  | 7.2±2.1    | 0.039   |
| Lumen area (mm²)  | 4.5±1.9  | 3.7±1.7    | 0.085   |
| Maximum plaque burden (%) | 45.9±16.3 | 47.5±19.8 | 0.737   |
| LCBI             | 42.1±70.8 | 70.1±87.9 | 0.165   |
| MaxLCBI_{mm}     | 108.7±177.3 | 172.0±208.9 | 0.202   |
| Calcification Score | 0.2±0.4 | 0.5±0.7 | 0.012   |

Values are mean ± standard deviation

**LCBI** lipid core burden index, **MB** myocardial bridge
In patients with MB, the distance from LAD ostium to MB entrance was 39.0 ± 9.8 mm and the length and maximum thickness of MB halo were 19.7 ± 10.5 mm and 0.54 ± 0.24 mm, respectively. Arterial compression of the MB segment was 18.6% on average. There were no significant correlations between maximum plaque burden in the proximal segment and MB parameters evaluated by grayscale IVUS, such as MB length ($r = -0.24$, $p = 0.12$), halo thickness ($r = -0.23$, $p = 0.15$), arterial compression ($r = -0.08$, $p = 0.62$), and the distance from LAD ostium ($r = 0.05$, $p = 0.73$). Calcification scores in the cross-section of maximum plaque burden in the proximal and MB segments were similar between patients with and without MB, but it was significantly lower in the distal segment in patients with MB compared to those without.

**NIRS-IVUS analysis**

In patients with MB, LCBI and maxLCBI$_{4\text{mm}}$ were significantly lower in the proximal segment ($p = 0.0088$ and 0.034, respectively. Representative cases were shown in Fig. 2) and in the MB segment ($p < 0.0001$ and $< 0.0001$, respectively), compared with the corresponding segments in non-MB patients (Table 2). In the distal segment, on the other hand, LCBI and maxLCBI$_{4\text{mm}}$ were similar between the two groups.

In the univariable analysis, target vessel for PCI, lumen area, maximum plaque burden, and the presence of MB were significantly associated with LCBI in the proximal segment (Table 3). Multivariable analysis demonstrated maximum plaque burden and MB to be independent predictors of LCBI in the proximal segment. Regarding maxLCBI$_{4\text{mm}}$ in the proximal segment, significant associations were shown in current smoking, target vessel for PCI, lumen area, maximum plaque burden, and the presence of MB in the univariable analysis. Multivariable analysis demonstrated a tendency with MB to predict maxLCBI$_{4\text{mm}}$ and current smoking and maximum plaque burden as independent predictors for maxLCBI$_{4\text{mm}}$.

With regard to the MB segment, target vessel for PCI, lumen area, calcification score, maximum plaque burden, the presence of MB, and use of beta-blockers were significantly associated with LCBI and maxLCBI$_{4\text{mm}}$, in the univariable analysis (Table 4). Multivariable analysis revealed maximum plaque burden and the presence of MB as independent predictors for both LCBI and maxLCBI$_{4\text{mm}}$. In the distal segment, MB was non-significantly associated with lower LCBI and maxLCBI$_{4\text{mm}}$ compared to those without.

**Discussion**

In the present study, MB was identified in 42 out of 116 patients (36%). Maximum plaque burden was not different between patients with and without MB in the proximal and distal segments, whereas significantly smaller in the MB segment compared with the corresponding mid segment in patients without MB. LCBI and maxLCBI$_{4\text{mm}}$ were significantly lower in both the proximal and MB segments in MB patients compared with the corresponding segments in non-MB patients. Multivariable analysis demonstrated MB and maximum plaque burden as independent predictors of LCBI and maxLCBI$_{4\text{mm}}$ in both the proximal and MB segments, except for the tendency that MB predicted maxLCBI$_{4\text{mm}}$ in the proximal segment. In the distal segment, MB was non-significantly associated with lower LCBI and maxLCBI$_{4\text{mm}}$.

MB was previously reported to accelerate atherosclerosis in the segment proximal to MB but not in the segment beneath MB, and to be associated with plaque rupture leading to myocardial infarction, especially in the younger population. These phenomena were supported by autopsy and clinical observational studies [2, 13, 14]. However, several studies using multi-slice computed tomography have demonstrated that MB has no association with the progression of atherosclerotic plaque [4, 5]. In the present study, plaque progression in the proximal segment was not different between patients with and without MB, although it was significantly suppressed in the MB segment compared with the corresponding mid segment in non-MB patients. While MB was defined as any echo-lucent band detected by grayscale IVUS in the present study, it was unclear whether all of MB had a significant effect on coronary blood flow, leading to increased atherosclerosis. In addition, patients in the present study had relatively more comorbidities, such as diabetes (DM) and dyslipidemia (DLP), than those of...
previous studies [15]. Moreover, patients with MB tended to be younger than those without in the present study. These potential factors could make the effect of MB on atherosclerotic plaque formation in the segment proximal to MB equivocal between patients with and without MB.

In the proximal segment of LAD, a smaller amount of lipid content was observed in MB patients compared with non-MB patients whereas plaque burden was not different between the 2 groups. Wall shear stress (WSS) distribution is one of the considerable factors for plaque formation and subsequent vulnerability [16]. In patients with MB, it has been reported that WSS at the segment proximal to MB is lower than that in the segment beneath MB [17, 18]. A previous study has demonstrated the relationship between high WSS and progression of lipid-rich plaque assessed by NIRS-IVUS imaging [19]. Thus, it is possible that low WSS in the segment proximal to MB might be associated with less lipid content observed in MB patients. However, since lower WSS was reported to have an impact on more plaque progression [20], the similar amount of plaque burden between patients with and without MB observed in the present study could not be explained from this point of view. On the other side, patients with MB tended to receive more intensive medical therapy (e.g., renin-angiotensin system inhibitors and beta-blockers) than those without in the present study. Given that renin-angiotensin system inhibitors and beta-blockers have been reported to have plaque-stabilizing effects related or unrelated to the plaque volume [21–24], these differences in pharmacological aspects might account for the discrepancy between plaque progression and instability. Further examination is required to clarify this issue.

In the segment beneath MB, WSS should be higher than in other segments [17, 18], and higher WSS possibly results in higher LCBI [19]. However, from the results of multivariable analysis in the present study, MB was associated with lower LCBI in the segment beneath MB. The
multivariable analysis also demonstrated maximum plaque burden in the MB segment as a significant predictor for LCBI and maxLCBI_{4mm} in the MB segment. Plaque burden was reported to be positively correlated with LCBI in previous studies [25]. Therefore, less plaque burden in the MB segment might cause lower LCBI, despite possibly high WSS in the MB segment. In addition, a previous study with optical coherence tomography showed the absence of adventitial vasa vasorum formation in the segment beneath MB [26], which is involved in atherosclerotic plaque formation. This histological feature of MB may be associated with less plaque burden as well as lipid content in the MB segment.

In the distal segment, MB was non-significantly associated with lower LCBI and maxLCBI_{4mm} compared to those without, whereas maximum plaque burden was similar between the two groups. Although the data regarding the characteristics of atherosclerosis in the segment distal to MB was scarce, there was an observational analysis that demonstrated factors associated with atherosclerosis located distally to MB. Hong et al. indicated cigarettes, angiographical lumen narrowing in the segment proximal to, and under MB as factors associated with atherosclerosis in the segment distal to MB [27]. In the present study, the portion of current smokers, and IVUS-derived maximum plaque burden in the proximal and MB segment were similar between the two groups. Since the measurements derived from quantitative coronary angiography and IVUS analysis were correlated with each other [28], these factors could contribute to the similar plaque burden in the distal segment. In addition, plaque-stabilizing effects caused by renin-angiotensin system inhibitors and beta-blockers could also have some effects on the discrepancy between plaque progression and instability [24] in the distal segment.

Considering that patients with established CAD were included in the present study, it is conceivable that confounding risk factors, such as age, hypertension, DLP, and DM, could affect the lipid content of atherosclerotic plaque. Nevertheless, the multivariable analysis demonstrated only maximum plaque burden (with a positive attitude) and MB (with a negative one) as independent predictors for lipid content in the proximal and mid segments of LAD. Although it is still uncertain whether MB has a significant effect.

### Table 3 Predictors of NIRS findings in proximal segment

| Variable               | LCBI Univariable | LCBI Multivariable | MaxLCBI_{4mm} Univariable | MaxLCBI_{4mm} Multivariable |
|------------------------|------------------|--------------------|---------------------------|----------------------------|
| Age (years)            | 0.133            | 0.159              | 0.128                     | 0.172                      |
| Male                   | 0.044            | 0.646              | -0.065                    | 0.486                      |
| BMI (kg/m²)            | 0.029            | 0.760              | -0.007                    | 0.940                      |
| Hypertension           | 0.018            | 0.850              | 0.055                     | 0.554                      |
| Dyslipidemia           | 0.017            | 0.858              | -0.029                    | 0.757                      |
| Diabetes mellitus      | 0.002            | 0.984              | -0.048                    | 0.611                      |
| Current smoker         | -0.132           | 0.163              | -0.202                    | 0.030 -0.188 0.020         |
| Prior MI               | -0.106           | 0.265              | -0.040                    | 0.668                      |
| UA                     | -0.013           | 0.891              | 0.077                     | 0.412                      |
| eGFR                   | -0.029           | 0.759              | 0.033                     | 0.725                      |
| LDL-C                  | 0.029            | 0.757              | 0.016                     | 0.866                      |
| Initial TnI            | -0.005           | 0.955              | 0.047                     | 0.621                      |
| BNP                    | 0.062            | 0.512              | 0.086                     | 0.356                      |
| ACS                    | 0.120            | 0.206              | 0.134                     | 0.151                      |
| Target vessel          | 0.294            | 0.002              | 0.049 0.628               | 0.314 <0.001 0.055 0.559   |
| Vessel area            | 0.038            | 0.690              | 0.056                     | 0.548                      |
| Lumen area             | -0.311           | 0.001              | 0.064 0.607               | -0.389 <0.001 -0.050 0.681 |
| Max PB in seg P        | 0.455            | <0.001             | 0.465 <0.001              | 0.514 <0.001 0.418 <0.001  |
| Calcification Score    | -0.005           | 0.959              | 0.162                     | 0.082 0.065 0.427          |
| Presence of MB         | -0.245           | 0.009              | -0.203 0.022              | -0.197 0.034 -0.151 0.071  |
| Antiplatelet           | 0.089            | 0.350              | 0.107                     | 0.253                      |
| Beta blocker           | 0.012            | 0.899              | -0.112                    | 0.229                      |
| ACEI/ARB               | 0.024            | 0.800              | 0.100                     | 0.286                      |
| Statin                 | 0.084            | 0.378              | 0.042                     | 0.657                      |

ACS: acute coronary syndrome, BMI: body mass index, LCBI: lipid core burden index, MB: myocardial bridge, PB: plaque burden, seg P: proximal segment.
on atherosclerotic plaque and its lipid content formation, patients with MB would not always have worse prognosis because of MB on atherosclerotic plaque vulnerability. However, further study is warranted to investigate what type of features of MB are associated with plaque stability or vulnerability, since previous reports suggest that MB can cause adverse events, such as arrhythmia, myocardial infarction, and sudden death in some patients with MB [29–31].

There are some limitations of the present study. This was a single-center study, and the number of subjects was small. The present study was a retrospective observational study and patient information was obtained from medical records only. Patients who had pre-existing coronary artery stent or severe calcification in the segment proximal to MB were excluded, which may limit the generalization of the present findings. In addition, all remaining patients were Japanese with 80% being male. Thus, caution is warranted in applying the present study results to different populations. The myocardial bridge could affect the findings of the ischemic tests [32–34], which might result in misunderstandings of the severity of myocardial ischemia in patients who needed PCI. Balloon dilatation before IVUS could affect the plaque volume and the findings derived from NIRS-IVUS in patients who underwent PCI [35–37].

In conclusion, lipid content of atherosclerotic plaque assessed by NIRS-IVUS imaging was significantly smaller in patients with MB than those without. MB could be considered as a predictor negatively associated with plaque vulnerability in the proximal and mid LAD.

### Table 4 Predictors of NIRS findings in MB segment

| Variable                  | LCBI | MaxLCBI<sup>4mm</sup> |
|---------------------------|------|-----------------------|
|                           | Univariable | Multivariable | Univariable | Multivariable |
|                           |    |                      |    |                      |    |                      |    |                      |
|                           | r  | P value               | β  | P value               | r  | P value               | β  | P value               |
| Age (years)               | 0.133 | 0.180             | 0.157 | 0.119             |
| Male                      | 0.099 | 0.318             | −0.055 | 0.587             |
| BMI (kg/m<sup>2</sup>)    | 0.092 | 0.353             | 0.064 | 0.528             |
| Hypertension              | 0.006 | 0.952             | 0.052 | 0.610             |
| Dyslipidemia              | −0.198 | 0.044             | −0.076 | 0.355             | −0.150 | 0.137             |
| Diabetes mellitus         | 0.113 | 0.253             | 0.117 | 0.245             |
| Current smoker            | 0.110 | 0.267             | 0.060 | 0.556             |
| Prior MI                  | 0.119 | 0.228             | 0.086 | 0.394             |
| UA                        | −0.061 | 0.540             | −0.075 | 0.456             |
| eGFR                      | −0.002 | 0.987             | −0.017 | 0.869             |
| LDL-C                     | −0.060 | 0.543             | 0.009 | 0.931             |
| Initial TnI               | −0.077 | 0.446             | −0.097 | 0.344             |
| BNP                       | −0.020 | 0.837             | 0.043 | 0.668             |
| ACS                       | −0.003 | 0.973             | 0.024 | 0.813             |
| Target vessel             | −0.239 | 0.015             | 0.071 | 0.418             | 0.226 | 0.024             | 0.012 | 0.881             |
| Vessel area               | −0.001 | 0.990             | −0.011 | 0.912             |
| Lumen area                | −0.345 | < 0.001           | 0.036 | 0.742             | −0.413 | < 0.001           | 0.021 | 0.833             |
| Max PB in seg M           | 0.578 | < 0.001           | 0.448 | < 0.001           | 0.673 | < 0.001           | 0.523 | < 0.001           |
| Calcification Score       | 0.249 | 0.013             | 0.139 | 0.096             | 0.256 | 0.012             | 0.126 | 0.094             |
| Presence of MB            | −0.390 | < 0.001           | −0.221 | 0.016             | −0.461 | < 0.001           | −0.287 | < 0.001           |
| Antiplatelet              | −0.069 | 0.489             | −0.078 | 0.440             |
| Beta blocker              | 0.197 | 0.045             | 0.110 | 0.200             | 0.251 | 0.012             | 0.136 | 0.081             |
| ACEI/ARB                  | 0.060 | 0.548             | 0.153 | 0.130             |
| Statin                    | 0.018 | 0.859             | −0.019 | 0.848             |

ACS acute coronary syndrome, BMI body mass index, LCBI lipid core burden index, MB myocardial bridge, PB plaque burden, seg M segment beneath the MB

ACS, acute coronary syndrome; BMI, body mass index; LCBI, lipid core burden index; MB, myocardial bridge; PB, plaque burden; seg M, segment beneath the MB

### Supplementary Information

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### Declarations

**Conflict of interest** Author’s disclosure of potential conflicts of interest; the authors declare no conflict of interests.
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