

Association Between the Presence of Coronary Artery Disease or Peripheral Artery Disease and Left Ventricular Mass in Patients Who Have Undergone Coronary Computed Tomography Angiography

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Abstract

Background: Left ventricular mass (LVM) is a critical marker of future cardiovascular risk. We determined the association between LVM measured by coronary computed tomography angiography (CCTA) and the presence of coronary artery disease (CAD) or peripheral artery disease (PAD) in patients who had undergone CCTA for screening of CAD.

Methods: We enrolled 1,307 consecutive patients (66 ± 12 years old, 49% males) who underwent CCTA for screening of CAD at the Fukuoka University Hospital (FU-CCTA registry), and either were clinically suspected of having CAD or had at least one cardiovascular risk factor. Patients with coronary stenosis of $\geq 50\%$ by CCTA were diagnosed as CAD. Patients with an ankle brachial pressure index < 0.9 or who had already been diagnosed with PAD were considered to have PAD. Left ventricular mass index (LVMI), left ventricular ejection fraction (LVEF), end-diastolic volume (EDV) and end-systolic volume (ESV) were measured. The patients were divided into CAD (-) and CAD (+) or PAD (-) and PAD (+) groups.

Results: The prevalences of CAD and PAD in all patients were 50% and 4.8%, respectively. Age, %males, %hypertension (HTN), %dyslipidemia (DL), %diabetes mellitus (DM), %smoking and %chronic kidney disease in the CAD (+) group were significantly higher than those in the CAD (-) group. Age, %males, %HTN, %DM and %smoking in the PAD (+) group were significantly higher than those in the PAD (-) group. CAD was independently associated with LVMI (odds

ratio (OR): 1.01, 95% confidence interval (CI): 1.01 - 1.02, $P < 0.01$) in addition to age, male, HTN, DL, DM, and smoking. PAD was also independently associated with LVMI (OR: 1.01, 95% CI: 1.0 - 1.02, $P = 0.018$) in addition to age, DM, and smoking.

Conclusions: LVMI determined by CCTA may be useful for predicting atherosclerotic cardiovascular diseases including both CAD and PAD, although there were considerable differences between %CAD and %PAD in all patients.

Keywords: Left ventricular hypertrophy; Computed tomography angiography; Coronary artery disease; Peripheral artery disease

Introduction

Coronary computed tomography angiography (CCTA) is widely available in hospitals in Japan, allowing the noninvasive assessment of coronary artery stenosis, calcification, and plaque [1, 2]. CCTA can also be used to measure left ventricular ejection fraction (LVEF) and left ventricular mass (LVM) with the use of software [3, 4].

Left ventricular hypertrophy (LVH) is a predictor of cardiovascular morbidity and mortality [5-12], and the improvement of LVH reduces the subsequent cardiovascular risk [10, 11]. The Framingham study showed that an increase in LVM of 50 g per height, as measured by echocardiography, was associated with an increased risk of coronary artery disease (CAD) [9].

The ankle brachial index (ABI) is an indicator of generalized vascular atherosclerosis because lower levels have been associated with higher rates of concomitant coronary and cerebrovascular diseases, and with the presence of cardiovascular risk factors [13-18]. A low ABI has been related to an increased incidence of mortality (total and cardiovascular), myocardial infarction and stroke. These increased relative risks have been shown to be independent of baseline cardiovascular disease and risk factors, suggesting that ABI might play an independent role in predicting cardiovascular events. Some studies have

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shown a high prevalence of LVH in patients at their first diagnosis of peripheral artery disease (PAD) [19]. The prevalence of echo LVH when LVM was indexed to body surface area (BSA) was 50%. There is already a high prevalence of LVH in patients at their first diagnosis of PAD [20]. Therefore, LVH is common enough in PAD patients to potentially make a major contribution to cardiac death.

We have been studying the risk factors for CAD at the Fukuoka University Hospital for the primary prevention of CAD using the Fukuoka University CCTA registry (FU-CCTA registry) [21-24]. Therefore, we determined the association between LVM measured by CCTA and the presence of CAD or PAD in patients who have undergone CCTA for screening of CAD.

Materials and Methods

Study subjects

We performed a cross-sectional study and enrolled 1,307 consecutive patients who underwent CCTA for screening of CAD at the Fukuoka University Hospital (FU-CCTA registry) and either were clinically suspected of having CAD or had at least one cardiovascular risk factor. This study was conducted in compliance with the ethical standards of the responsible institution on human subjects as well as with the Helsinki Declaration. The study was approved by the Fukuoka University Ethics Committee and was conducted after written consent was obtained from the subjects (#09-10-02).

Evaluation of coronary artery stenosis and PAD

Coronary artery stenosis was evaluated by constructing a volume-rendered image (Aquilion ONE, Canon Medical Systems USA, Inc., Tustin, CA) and evaluating the degree of lumen stenosis with multiplanar images. Coronary stenosis of $\geq 50\%$ by CCTA was considered to be CAD. The severity of coronary artery stenosis was determined by the number of branches with significant coronary stenosis, the coronary artery calcification (CAC) score, and the Gensini score. An analysis of cardiac function using the Aquilion ONE was used to automatically trace both diastolic and systolic cardiac function, and LVM, LVEF, left ventricular end-diastolic volume (LVEDV), and left ventricular end-systolic volume (LVESV) were measured. The left ventricular mass index (LVMI) was calculated by dividing LVM by the BSA in each patient. Patients with ABI < 0.9 or who had already been diagnosed with PAD were considered to have PAD.

Assessment of cardiovascular risks

All patients were evaluated with regard to body mass index (BMI), BSA, family history of CAD, smoking history, blood pressure (BP), hypertension (HTN), diabetes mellitus (DM), hemoglobin A1c (HbA1c), and fasting blood glucose (FBG).

In addition, the presence of dyslipidemia (DL), triglyceride (TG), high-density lipoprotein cholesterol (HDL-C), low-density lipoprotein cholesterol (LDL-C), non-HDL-C, chronic kidney disease (CKD), and estimated glomerular filtration rate (eGFR) were assessed.

Medications

Patients were being treated with an angiotensin-converting enzyme inhibitor/angiotensin II receptor blocker (ACEI/ARB), β -blocker, calcium channel blocker (CCB), and/or diuretic (DU) for HTN. They were taking statins or eicosapentaenoic acid (EPA) for DL, and an α -glucosidase inhibitor (α -GI), biguanide, dipeptidyl peptidase-4 inhibitor (DPP4I), insulin, sulfonylurea (SU) or thiazolidine for DM.

Statistical analysis

Statistical analyses were performed using Excel (SSRI, Tokyo, Japan) at Fukuoka University (Fukuoka, Japan), and EZR (Saitama Medical Center, Jichi Medical University, Saitama, Japan), which is a graphical user interface for R (The R Foundation for Statistical Computing, Vienna, Austria). Continuous variables are shown as the mean \pm standard deviation (SD). Categorical and continuous variables were compared between groups using a Chi-squared analysis and *t*-tests, respectively. Univariable and multivariable analyses of predictors for the presence of CAD or PAD were used to identify independent variables. We used a logistic regression model to examine multiple independent variables with the presence of CAD or PAD as the dependent variable. For the selection of independent variables, we selected from items that are believed to have a clinical impact in the presence of CAD or PAD (risk factors for atherosclerotic cardiovascular diseases) and added LVMI and some medications (ACEI/ARB and β -blocker) that affect LVMI.

Results

Patient characteristics in the non-CAD and CAD groups

Patient characteristics in the non-CAD and CAD groups are shown in Table 1. A total of 1,308 patients were enrolled: 658 in the non-CAD group and 650 in the CAD group. Age was 62 ± 13 years in the non-CAD group and 69 ± 10 years in the CAD group, and this difference was significant. The gender difference was also significant; 41% of the non-CAD group were male, compared to 57.2% of the CAD group. Similarly, there were significant differences in the prevalences of smoking (29.6% vs. 38.8%, $P < 0.05$), HTN (55% vs. 75.7%, $P < 0.05$), DM (18.5% vs. 32.2%, $P < 0.05$), DL (62.2% vs. 73.3%, $P < 0.05$) and CKD (22.9% vs. 32%, $P < 0.05$). In the evaluation of coronary artery, significant differences were found in the number of diseased branches, the CAC score, and the Gensini score. In terms of treatment, there were significant dif-

Table 1. Patient Characteristics in the Non-CAD and CAD Groups

	Non-CAD group N = 658	CAD group N = 650	Non-CAD vs. CAD group P value
Age (years)	62 ± 13	69 ± 10	< 0.001
Gender (male) (%)	41	57.2	< 0.001
BMI (kg/m ²)	24.0 ± 4.0	23.8 ± 3.7	0.348
BSA (m ²)	1.6 ± 0.21	1.6 ± 0.19	0.438
Family history (%)	23.3	21.5	0.457
Smoking (%)	29.6	38.8	< 0.001
HTN (%)	55	75.7	< 0.001
SBP (mm Hg)	133 ± 18	139 ± 21	< 0.001
DBP (mm Hg)	78 ± 13	79 ± 13	0.28
DM (%)	18.5	32.2	< 0.001
HbA1c (%)	5.9 ± 0.9	6.1 ± 0.9	< 0.001
FBG (mg/dL)	105 ± 43	113 ± 50	< 0.001
DL (%)	62.2	73.7	< 0.001
TG (mg/dL)	128 ± 100	141 ± 90	0.027
HDL-C (mg/dL)	60 ± 16	54 ± 15	< 0.001
LDL-C (mg/dL)	117 ± 32	113 ± 32	0.015
L/H	2.25	2.23	0.894
Non-HDL-C (mg/dL)	145 ± 38	144 ± 69	0.70
CKD (%)	22.9	32	< 0.001
eGFR (mL/min/1.73 m ²)	70 ± 15.2	66 ± 16.7	< 0.001
MetS (%)	19.4	31.1	< 0.001
VD	0 ± 0.12	1.88 ± 0.82	< 0.001
CAC score	35 ± 142	457 ± 986	< 0.001
Gensini score	2.5 ± 3.7	20.7 ± 19.7	< 0.001
Medications			
ACEI/ARB (%)	29.5	43.2	< 0.001
CCB (%)	31	46.8	< 0.001
β-blocker (%)	6.4	11.4	0.001
DU (%)	7.1	9.8	0.079
Statin (%)	24.5	38.8	< 0.001
EPA (%)	2.4	3.2	0.383
SU (%)	4.1	8.6	< 0.001
α-GI (%)	1.4	3.2	0.024
Biguanide (%)	4.7	8	0.015
Thiazolidine (%)	2.1	2.8	0.453
DPP4I (%)	7.8	13.1	< 0.001
Insulin (%)	2.4	2.8	0.701

Continuous variables are expressed as mean ± SD. CAD: coronary artery disease; BMI: body mass index; BSA: body surface area; HTN: hypertension; SBP: systolic blood pressure; DBP: diastolic blood pressure; DM: diabetes mellitus; HbA1c: hemoglobin A1c; FBG: fasting blood glucose; DL: dyslipidemia; TG: triglyceride; HDL-C: high-density lipoprotein cholesterol; LDL-C: low-density lipoprotein cholesterol; L/H-C: a ratio of LDL-C to HDL-C; Non-HDL-C: total cholesterol minus HDL-C; CKD: chronic kidney disease; eGFR: estimated glomerular filtration rate; MetS: metabolic syndrome; VD: vessel disease; CAC: coronary artery calcium; ACEI/ARB: angiotensin-converting enzyme inhibitor/angiotensin II receptor blocker; CCB: calcium channel blocker; DU: diuretic; EPA: eicosapentaenoic acid; SU: sulfonylurea; α-GI: α-glucosidase inhibitor; DPP4I: dipeptidyl peptidase-4 inhibitor.

ferences in the use of ACEI/ARB, β -blocker, CCB, statin, SU, α -GI, biguanide, and DPP4I.

Patient characteristics in the non-PAD and PAD groups

Patient characteristics in the non-PAD and PAD groups are shown in Table 2. Age was 65 ± 12 years in the non-PAD group and 70 ± 10 years in the PAD group, and this difference was significant. The gender difference was also significant: 61.9% of the PAD group were male, compared to 48.6% of the non-PAD group. Similarly, there were significant differences in the prevalences of smoking (33.5% vs. 52.4%, $P < 0.05$), HTN (64.5% vs. 79.4%, $P < 0.05$) and DM (24.4% vs. 46%, $P < 0.05$). In the evaluation of coronary arteries, significant differences were observed in the number of diseased vessels, the CAC score, and the Gensini score. Significant differences were also observed in treatment with SU, DPP4I, and insulin.

LV profiles in the non-CAD and CAD groups

LV function on CT was examined in the non-CAD and CAD groups (Fig. 1). Although LVEF and LVESV were not significantly different between the two groups, LVMI in the CAD group was significantly increased (71.6 ± 22.6 g/m² vs. 65.0 ± 18.2 g/m², $P < 0.05$). In addition, LVEDV in the non-CAD group was significantly lower than that in the CAD group (111.7 ± 34.0 mL vs. 116.7 ± 31.3 mL, $P < 0.05$).

LV profiles in the non-PAD and PAD groups

LV function was examined by CT in the non-PAD and PAD groups (Fig. 2). There were no significant differences between the two groups in LV profiles, although LVMI in the PAD (+) group tended to be higher than that in the PAD (-) group (75.9 ± 35.1 g/m² vs. 67.8 ± 19.6 g/m², $P = 0.074$).

LVMI in the non-CAD/non-PAD, CAD/non-PAD, non-CAD/PAD and CAD/ PAD groups

LVMI in the CAD/non-PAD and CAD/PAD groups were significantly higher than that in the non-CAD/non-PAD group (Table 3).

Univariable and multivariable analyses of predictors for the presence of CAD

A univariable analysis was performed on predictors for the presence of CAD (Table 4). Age, male, HTN, ACEI/ARB, β -blocker, DL, DM, smoking and CKD in addition to LVMI were significantly associated with the presence of CAD. A multivariable analysis of predictors for CAD is also shown in Table 4. The presence of CAD was independently associated with LVMI (odds ratio (OR): 1.01, 95% confidence interval (CI): 1.01 - 1.02, $P < 0.01$) in addition to age, male, HTN, DL, DM and smoking.

Univariable and multivariable analyses of predictors for the presence of PAD

A univariable analysis was performed with predictors for PAD (Table 5). The results showed associations between the presence of PAD and age, male, HTN, DM, smoking or LVMI. The presence of PAD was independently associated with LVMI (OR: 1.01, 95% CI: 1.0 - 1.02, $P = 0.018$) in addition to age, DM and smoking by a multivariable analysis (Table 5).

Discussion

In this study, we analyzed the association between LVMI and the presence of CAD or PAD. An increased LVMI is associated with all-cause mortality, arrhythmic death, and sudden death in patients with CAD [25]. In addition, the Framingham study showed that an increase in LVM over time by echocardiography in a healthy population is a risk factor for major adverse cardiovascular events (MACEs) [7, 9]. These previous studies suggest that LVH may be useful for predicting subsequent MACEs and all-cause mortality. Thus, we hypothesized that LVMI obtained at the time of CCTA was associated with CAD or PAD because atherosclerotic cardiovascular diseases such as CAD and PAD are MACEs. Finally, we found that the presence of CAD or PAD was associated with LVMI in patients with suspected CAD or at least one cardiovascular risk at the time of CCTA for screening of CAD.

Another important issue in this study was the association between PAD and LVMI. PAD was also independently associated with LVMI by a multivariable analysis. It has been shown that, among patients with CKD, those with higher BMI and LVMI tend to have an abnormal ABI [26]. It has also been suggested that adding ABI to the Framingham risk score may provide a more accurate assessment of all-cause mortality, including cardiovascular death [27]. Several studies have shown that LVH is strongly associated with cardiovascular risk and mortality [9, 28]. Furthermore, it has been suggested that the presence of LVH is associated with the risk of cardiovascular death in patients with PAD, and that the prevalence of LVH is increased by 29% when patients have PAD in addition to HTN and angina pectoris [29]. A previous study showed that 50% of patients with PAD had LVH, and that LVH is common in patients with PAD [24]. Our study in patients who underwent CCTA for screening of CAD is also consistent with the results reported previously.

Since DM, HTN, and DL are already known risk factors for CAD, we were also able to confirm that DM, HTN, and DL were associated with CAD. Therefore, it is assumed that the subjects of this study are not a special population, and we believe that this is worth analyzing. HTN, DL, smoking, and DM are also well-known risk factors for PAD [30]. However, in this study, while smoking and DM were associated with PAD, HTN and DL were not. In a previous study [30], the study population consisted of subjects from the healthy general population. In contrast, in this study, the population consisted of patients with cardiovascular risk or suspected CAD. The proportion of patients with cardiovascular risks in their background in the present study was relatively high. Therefore, it is possible that

Table 2. Patient Characteristics in the Non-PAD and PAD Groups

	Non-PAD group N = 1,245	PAD group N = 63	Non-PAD vs. PAD group P value
Age (years)	65 ± 12	70 ± 10	< 0.001
Gender (male) (%)	48.6	61.9	0.04
BMI (kg/m ²)	24.0 ± 4.0	23.8 ± 4.5	0.86
BSA (m ²)	1.6 ± 0.2	1.6 ± 0.19	0.94
Family history (%)	22.7	12.7	0.06
Smoking (%)	33.5	52.4	0.002
HTN (%)	64.5	79.4	0.016
SBP (mm Hg)	136 ± 19	141 ± 20	0.04
DBP (mm Hg)	78 ± 13	75 ± 13	0.06
DM (%)	24.4	46	< 0.001
HbA1c (%)	6.0 ± 1.0	6.5 ± 1.1	< 0.001
FBG (mg/dL)	108 ± 47	122 ± 37	< 0.001
DL (%)	67.9	69.8	0.74
TG (mg/dL)	135 ± 96	118 ± 56	0.024
HDL-C (mg/dL)	57 ± 16	53 ± 15	0.051
LDL-C (mg/dL)	115 ± 32	104 ± 33	0.01
L/H	2.25	2.23	0.231
Non-HDL-C (mg/dL)	146 ± 56	130 ± 36	< 0.001
CKD (%)	27	36.5	0.097
eGFR (mL/min/1.73 m ²)	68.3 ± 16.0	65.9 ± 18.2	0.296
MetS (%)	24.7	31.7	0.225
VD	0.91 ± 1.09	1.60 ± 1.19	< 0.001
CAC score	228 ± 700	695 ± 1,364	0.009
Gensini score	10.9 ± 15.9	22.9 ± 26.2	0.002
Medications			
ACEI/ARB (%)	35.8	47.6	0.057
CCB (%)	38.4	47.6	0.148
β-blocker (%)	8.7	11.1	0.492
DU (%)	8.3	12.7	0.242
Statin (%)	31.3	39.7	0.167
EPA (%)	2.9	1.6	1.00
SU (%)	5.7	20.6	< 0.001
α-GI (%)	2.2	4.8	0.17
Biguanide (%)	6.1	12.7	0.058
Thiazolidine (%)	2.3	6.3	0.069
DPP4I (%)	9.7	23.8	0.002
Insulin (%)	2.3	9.5	0.005

Continuous variables are expressed as mean ± SD. PAD: peripheral artery disease; BMI: body mass index; BSA: body surface area; HTN: hypertension; SBP: systolic blood pressure; DBP: diastolic blood pressure; DM: diabetes mellitus; HbA1c: hemoglobin A1c; FBG: fasting blood glucose; DL: dyslipidemia; TG: triglyceride; HDL-C: high-density lipoprotein cholesterol; LDL-C: low-density lipoprotein cholesterol; L/H-C: a ratio of LDL-C to HDL-C; Non-HDL-C: total cholesterol minus HDL-C; CKD: chronic kidney disease; eGFR: estimated glomerular filtration rate; MetS: metabolic syndrome; VD: vessel disease; CACS: coronary artery calcium score; ACEI/ARB: angiotensin-converting enzyme inhibitor/angiotensin II receptor blocker; CCB: calcium channel blocker; DU: diuretic; EPA: eicosapentaenoic acid; SU: sulfonylurea; α-GI: α-glucosidase inhibitor; DPP4I: dipeptidyl peptidase-4 inhibitor.

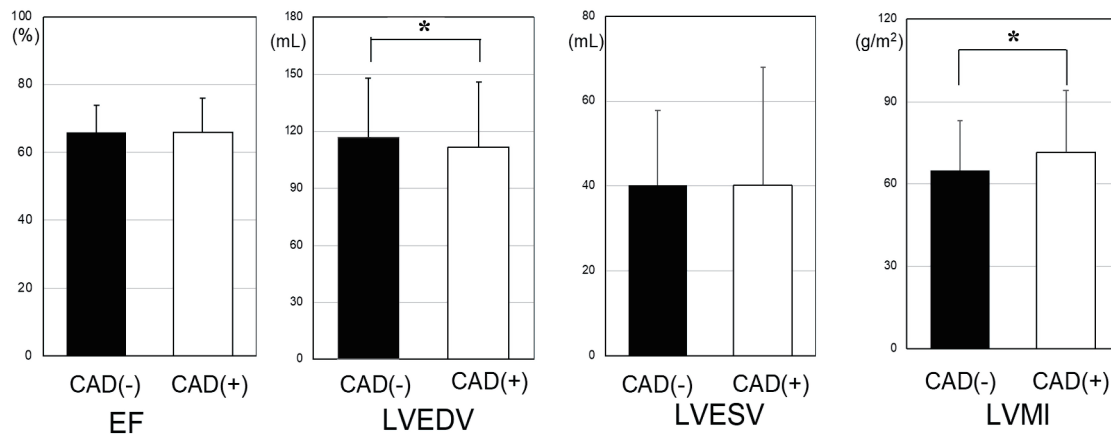


Figure 1. LV profiles in the non-CAD and CAD groups. * $P < 0.05$. CAD: coronary artery disease; LV: left ventricular; LVMI: left ventricular mass index; LVEF: left ventricular ejection fraction; LVEDV: left ventricular end-diastolic volume; LVESV: left ventricular end-systolic volume.

some risk factors were not associated with the presence of PAD. On the other hand, older age, DM, and smoking might be more strongly associated with PAD than other risk factors. The 2007 TASC II consensus document lists the OR for various risk factors of symptomatic PAD [31]. The OR of DM and smoking are roughly between 3 and 4, whereas those for HTN and DL are between approximately 1.5 and 2. There have been no reports of OR for risk factors related to asymptomatic PAD. While our study includes patients with asymptomatic conditions and the patient backgrounds differ, which makes direct comparisons challenging; HTN and DL can be considered to have a lower attributable risk for PAD compared to DM and smoking. In addition, the PAD group consisted of only 63 individuals, suggesting low statistical power.

There was a gender difference in the association between CAD and LVMI. It has been shown that males are at higher risk for CAD compared to females [32] even after adjustment for cardiovascular risk factors. Although females are more

likely to develop PAD than males in all age groups [33], the present study found no gender association by a multiple logistic analysis in patients with PAD. Additional studies will be needed to resolve this issue.

Study limitations

First, patients who had at least one cardiovascular risk factor or who were clinically suspected of having CAD were enrolled in this study. Although the number of cases of CAD was sufficient, the number of cases of PAD was small. This may have contributed to the difficulty of a statistical comparison. Second, it was not possible to evaluate patients over time, and the influence of changes in LVH could not be examined as that in a prospective study, because CAD and PAD were evaluated at the time of CCTA. Third, PAD was evaluated by ABI and not by angiography or contrast CT. Therefore, the lesion site could

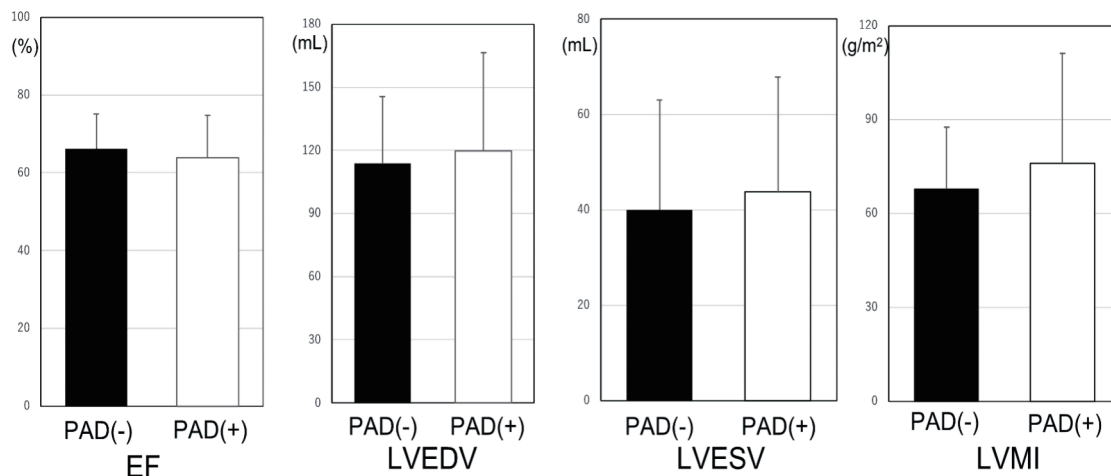


Figure 2. LV profiles in the non-PAD and PAD groups. PAD: peripheral artery disease; LV: left ventricular; LVMI: left ventricular mass index; LVEF: left ventricular ejection fraction; LVEDV: left ventricular end-diastolic volume; LVESV: left ventricular end-systolic volume.

Table 3. LVMI in the Non-CAD/Non-PAD, CAD/Non-PAD, Non-CAD/PAD and CAD/PAD Groups

Group	LVMI
Non-CAD/non-PAD	64.8 ± 17.9
CAD/non-PAD	71.1 ± 20.9**
Non-CAD/PAD	71.7 ± 26.7
CAD/PAD	77.3 ± 37.7*

Continuous variables are expressed as mean ± SD. **P < 0.01, *P < 0.05 vs. non-CAD/non-PAD group. CAD: coronary artery disease; PAD: peripheral artery disease; LVMI: left ventricular mass index.

Table 4. Univariable and Multivariable Analyses of Predictors for the Presence of CAD

	Univariable regression			Multivariable regression		
	Odds ratio	95% CI	P value	Odds ratio	95% CI	P value
Age	1.05	1.04 - 1.06	< 0.01	1.07	1.05 - 1.08	< 0.01
Male	1.92	1.54 - 2.4	< 0.01	1.97	1.46 - 2.65	< 0.01
BMI	0.986	0.959 - 1.01	0.348	0.979	0.945 - 1.01	0.227
HTN	2.55	2.01 - 3.22	< 0.01	1.59	1.15 - 2.19	< 0.01
ACEI/ARB	1.82	1.45 - 2.29	< 0.01	0.944	0.697 - 1.28	0.708
β-blocker	1.88	1.27 - 2.8	< 0.01	1.55	0.989 - 2.44	0.056
DL	1.71	1.35 - 2.16	< 0.01	1.7	1.29 - 2.24	< 0.01
DM	2.08	1.61 - 2.69	< 0.01	1.56	1.17 - 2.08	< 0.01
Family history of CAD	0.906	0.699 - 1.18	0.457	1.12	0.828 - 1.5	0.473
Smoking	1.5	1.19 - 1.89	< 0.01	1.35	1.01 - 1.8	0.043
CKD	1.58	1.24 - 2.02	< 0.01	0.871	0.651 - 1.17	0.354
LVMI	1.02	1.01 - 1.02	< 0.01	1.01	1.01 - 1.02	< 0.01

CAD: coronary artery disease; BMI: body mass index; HTN: hypertension; ACEI/ARB: angiotensin-converting enzyme inhibitor/angiotensin II receptor blocker; DL: dyslipidemia; DM: diabetes mellitus; CKD: chronic kidney disease; LVMI: left ventricular mass index; CI: confidence interval.

Table 5. Univariable and Multivariable Analyses of Predictors for the Presence of PAD

	Univariable regression			Multivariable regression		
	Odds ratio	95% CI	P value	Odds ratio	95% CI	P value
Age	1.05	1.02 - 1.07	< 0.01	1.05	1.02 - 1.08	< 0.01
Male	1.72	1.02 - 2.9	0.041	1.05	0.556 - 1.97	0.885
BMI	0.993	0.928 - 1.06	0.837	0.997	0.923 - 1.08	0.938
HTN	2.12	1.14 - 3.94	0.018	1.22	0.58 - 2.58	0.597
ACEI/ARB	1.63	0.981 - 2.71	0.059	1.11	0.606 - 2.02	0.741
β-blocker	1.31	0.584 - 2.95	0.51	1.04	0.438 - 2.46	0.933
DL	1.1	0.632 - 1.9	0.743	0.977	0.547 - 1.74	0.937
DM	2.64	1.58 - 4.41	< 0.01	2.15	1.26 - 3.69	< 0.01
Family history of CAD	0.495	0.233 - 1.05	0.067	0.593	0.274 - 1.28	0.185
Smoking	2.18	1.31 - 3.63	< 0.01	2.22	1.21 - 4.09	0.01
CKD	1.56	0.919 - 2.64	0.1	1.01	0.569 - 1.81	0.967
LVMI	1.01	1.0 - 1.02	< 0.01	1.01	1.0 - 1.02	0.018

PAD: peripheral artery disease; BMI: body mass index; HTN: hypertension; ACEI/ARB: angiotensin-converting enzyme inhibitor/angiotensin II receptor blocker; DL: dyslipidemia; DM: diabetes mellitus; CKD: chronic kidney disease; LVMI: left ventricular mass index; CI: confidence interval.

not be evaluated.

Conclusions

LVMI, as measured by CCTA, was associated with atherosclerotic cardiovascular diseases, including CAD and PAD, and may be a useful imaging biomarker.

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Conflict of Interest

The authors have no conflict of interest.

Informed Consent

The study was approved by the Fukuoka University Ethics Committee and was conducted after written informed consent was obtained from the subjects.

Author Contributions

Conceptualization: TT and YS. Methodology: YS and KT. Data collection: TT, YS, KT, SH and Y. Kawahira. Software: YS and KT. Formal analysis: TT and YS. Investigation: TH, TT and Y. Kawahira. Writing - original draft preparation: TT. Writing - review and editing: TH, Y. Kato, and TK. Supervision: MS and SM. All authors have read and agreed to the published version of the manuscript.

Data Availability

The data supporting the findings of this study are available from the corresponding author upon reasonable request.

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