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Longitudinal quantitative assessment of coronary plaque progression related to body mass index using serial coronary computed tomography angiography

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Aims This study explored the coronary plaque volume change (PVC) according to the change of percent body mass index (BMI) and categorical BMI group using serial coronary computed tomography angiography (CCTA).

Methods and results A total of 1568 subjects who underwent serial CCTA with available BMI at baseline (CCTA1) and follow-up (CCTA2) were included. Median inter-scan period was 3.3 (interquartile range: 2.6–4.6) years. Quantitative assessment of coronary plaque was performed at both scans. All participants were categorized into three BMI (kg/m^2) groups: normal: <25.0; overweight: 25.0–29.9; and obesity: ≥30.0. During follow-up, there were no significant differences in annualized PVC according to the 5% change of BMI in all BMI groups. Among 1424 (90.8%) subjects in the same BMI group at CCTA1 and CCTA2, a significant difference in annualized (PVC) was observed among the three groups. In 144 (9.2%) subjects with the change in their BMI group at CCTA2 compared their results at CCTA1, annualized PVC was not different compared with subjects in the same BMI group during follow-up. The percent change of BMI was not significantly related to the annualized PVC after adjusting confounding factors.

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Male gender [odds ratio (OR): 1.38; 95% confidence interval (CI): 1.05–1.81; $P = 0.022$], baseline plaque volume (OR: 1.07; 95% CI: 1.05–1.09; $P < 0.001$), and baseline overweight or obesity (OR: 1.35; 95% CI: 1.04–1.77; $P = 0.027$) were independently associated with coronary plaque progression.

Conclusion Over the near term, longitudinal small changes in BMI were not associated with changes in coronary plaque volume although baseline BMI was.

Clinical trial registration ClinicalTrials.gov NCT02803411.

Keywords atherosclerosis • coronary computed tomography angiography • body mass index • obesity

Introduction

The prevalence of obesity has rapidly increased over the past few decades worldwide.[1,2] In recent years, obesity has become an established risk factor for coronary artery disease (CAD), which is the most important cause of morbidity and mortality.[3,4] Prior cross-sectional investigations using coronary computed tomography angiography (CCTA) reported that an increased body mass index (BMI) is associated with an increased prevalence of CAD.[5,6] Thus, recent guidelines have strongly recommended both modest weight reduction, 5–10% of initial body weight, and lifestyle modifications for the overweight or obese populations.[7] However, the coronary atherosclerosis related to the percent change of BMI has not been established in general population. Thus, the present study explored both coronary atherosclerosis along the full length of the coronary vascular tree and the changes in coronary plaque volume according to the percent change of BMI in subjects with low-to-intermediate cardiovascular risk.

Methods

Design overview, setting, and participants

The overall study design of the Progression of AtheRosclerotic PlAque DetermIned by Computed TomoGraphic Angiography IMaging (PARADIGM) registry has been previously described.[8] Briefly, the PARADIGM is a prospective, open-label, international, multicentre dynamic observational registry designed to evaluate associations between changes in serial CCTA imaging findings and clinical presentation. Between 2003 and 2015, a total of 2252 consecutive subjects underwent CCTA at 13 centres in seven countries (Supplementary data online, *Table S1*). The inclusion criteria of the PARADIGM registry were as follows: (i) subjects who underwent two or more clinically indicated CCTAs with 64-detector rows or greater for CAD evaluation, (ii) at least a 2-year interval between the baseline (CCTA1) and follow-up CCTA (CCTA2), and (iii) prospective data collection for CAD risk factors. Exclusion criteria included the following: (i) unavailable clinical or laboratory data within 1 month from CCTA1 or CCTA2 and (ii) uninterpretable CCTA. Among 2252 consecutive participants, 1568 subjects with available BMI data at CCTA1 and CCTA2 were included in the present study. All participants were categorized into three groups according to BMI (kg/m^2) value: normal: <25.0 ($n = 752$); overweight: 25.0–29.9 ($n = 675$); and obesity: ≥30.0 ($n = 141$). The main objective of this study was (i) to compare the change of plaque volume according to the 5% of BMI change and the categorical BMI group change and (ii) to identify the independent predictors for coronary plaque progression. The study protocol was approved by the institutional review boards of all centres, and, when required, all patients provided written informed consent.

Acquisition and interpretation of CCTA

All testing, data acquisition, and image post-processing for CCTA in the PARADIGM cohort were in accordance with Society of Cardiovascular Computed Tomography guidelines.[9,10] CCTA was conducted using a scanner with ≥64-detector rows in all centres. Radiation dose according to BMI group was present in Supplementary data online, *Table S2*. Baseline and follow-up datasets from each centre were transferred to an offline workstation for analysis using semi-automated plaque analysis software (QAngioCT Research Edition v2.1.9.1; Medis Medical Imaging Systems, Leiden, the Netherlands) with manual correction. Independent level III-experienced readers who were masked to the clinical and test results analysed all CCTAs. Segments with a diameter ≥2 mm were evaluated using a modified 17-segment American Heart Association model.[10,11] Plaque volumes (mm^3) of every coronary segments, regardless of the presence of atherosclerotic plaque, were obtained and then summated to generate the total plaque volume on a per-patient level. Atherosclerotic plaque volume was further sub-classified by composition, employing predefined intensity cut-off values in Hounsfield units (HU) for necrotic core (-30 to 30 HU), fibro-fatty plaque (31–130 HU), fibrous plaque (131–350 HU), and calcified plaque (≥351 HU) that had been validated against intravascular ultrasound.[12,13] Non-calcified plaque volume was defined as the sum of the fibrous, fibro-fatty, and necrotic core plaque volumes. For longitudinal comparisons of CCTAs, both baseline and follow-up coronary segments were co-registered using fiduciary landmarks, including distance from ostia or branch vessels takeoffs. Plaque volume change (PVC) was defined as plaque volume at CCTA2 minus plaque volume at CCTA1 in per-patient level. Annualized PVC was defined as PVC divided by inter-scan period. Coronary plaque progression was defined as the difference in plaque volume between follow-up and baseline CCTA >0. Representative CCTA imaging is presented in *Figure 1*.

Statistical analysis

Continuous variables are expressed as mean ± standard deviation or median and interquartile range. Categorical variables are presented as absolute values and proportions. The characteristics of participants across the three BMI groups were compared using one-way analysis of variance with Bonferroni's *post hoc* test or the Kruskal–Wallis test for continuous variables, as appropriate, and a χ^2 test for categorical variables. In a subgroup analysis, continuous variables were compared using the independent *t*-test or Mann–Whitney *U* test, as appropriate, and

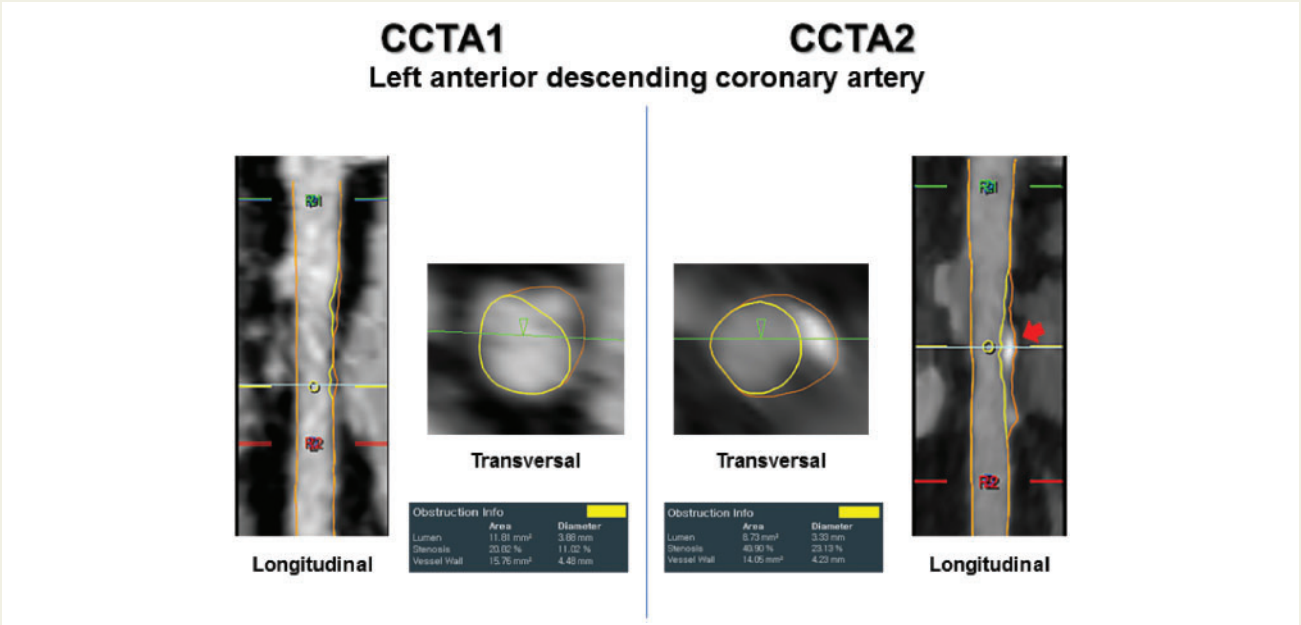

Figure 1 Representative CCTA image. A 66-year-old man underwent CCTA1 and CCTA2 with 7.3 years of inter-scan period. At CCTA1, the patient's BMI was 28.0 kg/m^2 and total plaque volume was 108.0 mm^3. During follow-up, the percent change of BMI was -6.1% but coronary plaque progression was observed. Both PVC and annualized PVC were 164.9 mm^3 and 22.5 mm^3, respectively. Red arrow in CCTA2 shows an increased plaque volume of left anterior descending coronary artery compared with CCTA1.

categorical variables were compared using a χ^2 test. Comparison of continuous variables between CCTA1 and CCTA2 was performed using a paired *t*-test. Generalized linear model was used to identify the association between clinical variables and annualized PVC. Logistic regression analysis was performed to identify the independent predictors of coronary plaque progression. Independent variables included traditional cardiovascular risk factors including age, gender, hypertension, diabetes, dyslipidaemia, and current smoking, baseline plaque volume, baseline categorical BMI group, and statin use. Variables with $P < 0.05$ in the univariate analysis were considered confounding variables and entered into multivariate regression analysis. All statistical analyses were performed using the Statistical Package for the Social Sciences version 19 (SPSS, Chicago, IL, USA) and SAS (version 9.1.3; SAS Institute Inc., Cary, NC, USA). A *P*-value <0.05 was considered significant for all analyses.

Results

Baseline characteristics of study population and coronary arteries

The overall mean age was 61 ± 9 years, 62.4% were male, and the median inter-scan period was 3.3 years (2.6–4.6). Hypertension, diabetes mellitus, and dyslipidaemia were present in 54.6%, 21.4%, and 43.9% of the subjects, respectively. In addition, 17.8% of the patients were current smokers. Baseline characteristics according to BMI groups are described in *Table 1*. The prevalence of male gender, hypertension, and dyslipidaemia were significantly different in each BMI group. However, there were significant differences in the vessel volume, lumen volume, and plaque volume among the three BMI groups.

Clinical and laboratory variables at CCTA1 and CCTA2

Changes in clinical and laboratory variables during the follow-up periods are presented in *Table 2*. There were significant differences in systolic and diastolic blood pressure and the levels of triglyceride (TG) and high-density lipoprotein cholesterol (HDL-C) according to the three BMI groups at CCTA1 and CCTA2. However, the levels of total cholesterol (T-chol) and low-density lipoprotein cholesterol (LDL-C) were not different between each of the BMI groups at both CCTAs. During the inter-scan period, the use of statins increased considerably after CCTA1 (45.2% vs. 57.4%). Compared with CCTA1, systolic and diastolic blood pressure and the levels of T-chol, TG, HDL-C, and LDL-C were significantly decreased in all subjects at CCTA2. Shifts from one BMI group to another between CCTA1 and CCTA2 were observed in only 9.2% of the subjects, with 4.4% shifting into a decreased BMI group and 4.8% shifting to an increased BMI group. Subjects with change in BMI more than 5% between CCTA1 and CCTA2 were observed in 15.5%; subjects with decrease and increase more than 5% were 7.0% and 8.5%, respectively.

Change of plaque volume according to the 5% percent change of BMI

There were no significant differences in either PVC or annualized PVC between subjects with a change in BMI of less than 5%,

Table 1 Baseline characteristics

	Normal ($n = 752$)	Overweight ($n = 675$)	Obese ($n = 141$)	*P*-value
Age (years)	62 (55–67)	61 (55–67)	61 (54–67)	0.468
Male	430 (57.2)	453 (67.1)	95 (67.4)	<0.001
Initial BMI (kg/m^2)	23.3 (22.0–24.2)	26.8 (26.0–27.9)	31.3 (30.5–33.0)	<0.001
Hypertension	367 (49.1)	395 (59.5)	94 (67.1)	<0.001
Diabetes mellitus	162 (21.6)	143 (21.5)	30 (21.3)	0.994
Dyslipidaemia	299 (40.0)	316 (47.7)	74 (52.9)	0.002
Current smoking	137 (18.3)	116 (17.5)	26 (18.7)	0.894
Coronary artery				
Segment length (mm)	379.1 (270.9–464.7)	389.2 (272.7–473.0)	379.4 (274.9–456.1)	0.816
Vessel volume (mm^3)	1890.0 (1277.1–2549.5)	2098.9 (1449.4–2946.6)	2391.8 (1714.3–3127.8)	<0.001
Lumen volume (mm^3)	1783.3 (1212.1–2438.6)	1976.2 (1353.0–2826.3)	2256.4 (1533.6–2995.7)	<0.001
Plaque volume (mm^3)	45.5 (3.2–127.6)	58.7 (9.2–150.4)	80.8 (14.9–190.3)	0.004
Fibrous	18.8 (0.4–53.2)	26.0 (4.9–66.4)	37.6 (6.3–82.8)	<0.001
Fibrous fatty	1.9 (0–15.9)	5.2 (0.0–22.5)	7.8 (0.4–31.5)	<0.001
Necrotic core	0.0 (0.0–0.6)	0.0 (0.0–1.5)	0.2 (0.0–2.3)	<0.001
Dense calcium	8.3 (0.0–45.4)	10.0 (0.0–41.5)	16.7 (0.4–53.8)	0.187
Inter-scan period (years)	3.3 (2.6–4.6)	3.4 (2.6–4.6)	3.3 (2.7–4.8)	0.813

Values are expressed as median (interquartile range) or *n* (%).
BMI, body mass index.

those with decreases in BMI of more than 5%, and those with increases in BMI of more than 5% at CCTA2 compared with CCTA1 in all BMI groups (*Figure 2*). Coronary artery characteristics at CCTA2 according to BMI groups are described in Supplementary data online, *Table S3*.

Change of plaque volume according to the categorical BMI group change

The results of changes in coronary plaque volume according to BMI group are presented in *Figure 3*. In subjects without any change in BMI group between CCTA1 and CCTA2, a significant difference in PVC was observed among the three groups (normal: 49.1 ± 83.0 mm^3 vs. overweight: 59.0 ± 89.3 mm^3 vs. obesity: 70.8 ± 87.8 mm^3; $P = 0.014$). Similarly, the annualized PVC was significantly different among the three groups (normal: 14.6 ± 24.0 mm^3 vs. overweight: 17.0 ± 24.7 mm^3 vs. obesity: 21.7 ± 29.4 mm^3; $P = 0.009$) (*Figure 3A*). In subjects who shifted to an increased or decreased BMI group between CCTA1 and CCTA2, both PVC and annualized PVC were not significantly different compared to subjects with no change in BMI group at both CCTA1 and CCTA2 (*Figure 3B–D*).

Association between clinical variables and change of plaque volume

The results of generalized linear model for analysing the association between clinical variables and annualized PVC were present in *Table 3*. Hypertension, diabetes mellitus, and baseline plaque volume were independently associated with annualized PVC. However, the percentage change of BMI did not have significant association with annualized PVC.

Independent predictors for coronary plaque progression

The results of univariate and multivariate regression analyses to identify the independent predictors for coronary plaque progression were present in *Table 4*. In the univariate regression analysis, age [odds ratio (OR): 1.03; 95% confidence interval (CI): 1.02–1.04], male gender (OR: 1.76; 95% CI: 1.37–2.25), hypertension (OR: 1.72; 95% CI: 1.34–2.20), diabetes mellitus (OR: 1.71; 95% CI: 1.22–2.38), dyslipidaemia (OR: 1.43; 95% CI: 1.11–1.84), current smoking (OR: 1.43; 95% CI: 1.01–2.02), baseline plaque volume (OR: 1.08; 95% CI: 1.06–1.10), baseline overweight or obesity (OR: 1.54; 95% CI: 1.21–1.98), and statin use (OR: 1.77; 95% CI: 1.37–2.30) were significantly associated with plaque progression (all $P < 0.05$). Multivariate regression analysis showed that male (OR: 1.38; 95% CI: 1.05–1.81), baseline coronary plaque volume (OR: 1.07; 95% CI: 1.05–1.09), and baseline overweight or obesity (OR: 1.35; 95% CI: 1.04–1.77) were independently associated with coronary plaque progression (All $P < 0.05$).

Discussion

The results of this prospective, multicentre, international study demonstrated that both 5% change of BMI and categorical BMI group change were not related to the coronary PVC during the near-term period. This study could identify that male gender, baseline plaque volume, and baseline overweight or obesity were independently associated with coronary plaque progression.

The advancements in CT technology enable evaluations of coronary atherosclerosis including plaque morphology, quantification of plaque volume, and stenosis severity.[14–16] In addition, the semi-

Table 2 Changes in clinical and laboratory variables between CCTA1 and CCTA2

	Total ($n = 1568$)	Normal ($n = 752$)	Overweight ($n = 675$)	Obese ($n = 141$)	*P*-value
CCTA1					
Systolic blood pressure (mmHg)	129 ± 18	126 ± 17	131 ± 18*	138 ± 20*,**	<0.001
Diastolic blood pressure (mmHg)	78 ± 11	76 ± 10	79 ± 11*	81 ± 12*	<0.001
Fasting glucose (mg/dL)	110 ± 34	108 ± 36	111 ± 32	113 ± 32	0.235
T-chol (mg/dL)	187 ± 40	185 ± 40	189 ± 40	190 ± 42	0.213
TG (mg/dL)	143 ± 85	130 ± 80	155 ± 90*	161 ± 75*	<0.001
HDL-C (mg/dL)	51 ± 14	53 ± 15	49 ± 12*	48 ± 11*	<0.001
LDL-C (mg/dL)	114 ± 34	113 ± 34	115 ± 34	114 ± 41	0.467
Statin use	708 (45.2)	334 (46.8)	307 (49.4)	67 (53.6)	0.306
CCTA2					
Systolic blood pressure (mmHg)	127 ± 17***	125 ± 16	128 ± 17*,***	137 ± 20*,**	<0.001
Diastolic blood pressure (mmHg)	76 ± 10***	75 ± 10***	77 ± 10*,***	79 ± 12*,**	<0.001
Fasting glucose (mg/dL)	110 ± 28	108 ± 28	110 ± 29	115 ± 25	0.162
T-chol (mg/dL)	171 ± 38***	170 ± 37***	173 ± 39***	178 ± 41***	0.118
TG (mg/dL)	129 ± 77***	118 ± 71***	139 ± 82*,***	138 ± 80***	<0.001
HDL-C (mg/dL)	50 ± 13***	52 ± 14***	48 ± 12*,***	49 ± 12	<0.001
LDL-C (mg/dL)	100 ± 33***	98 ± 32***	101 ± 34***	106 ± 34	0.134
Statin use	900 (57.4)	442 (63.5)	384 (62.3)	74 (62.2)	0.896
Changed obesity group between CCTA1 and CCTA2					<0.001
No change	1424 (90.8)	701 (93.2)	606 (89.8)	117 (83.0)	
Decreased	68 (4.4)	0 (0.0)	44 (6.5)	24 (17.0)	
Increased	76 (4.8)	51 (6.8)	25 (3.7)	0 (0.0)	
Cut-offs of 5% BMI change between CCTA1 and CCTA2					<0.001
Within 5% BMI change	1325 (84.5)	655 (87.1)	570 (84.4)	100 (70.9)	
Decrease more than 5%	110 (7.0)	34 (4.5)	52 (7.7)	24 (17.0)	
Increased more than 5%	133 (8.5)	63 (8.4)	53 (7.9)	17 (12.1)	
Change of BMI (kg/m^2)	0.03 ± 1.43	0.15 ± 1.21	0.00 ± 1.08	-0.52 ± 3.02*	<0.001

Values are expressed as mean ± standard deviation or *n* (%).
*$P < 0.05$ vs. normal.
**$P < 0.05$ vs. overweight.
***$P < 0.05$ vs. index CCTA.
BMI, body mass index; CCTA, coronary computed tomography angiography; CCTA1, baseline CCTA; CCTA2, follow-up CCTA; HDL-C, high-density lipoprotein cholesterol; LDL-C, low-density lipoprotein cholesterol; T-chol, total cholesterol; TG, triglyceride.

automated plaque quantification methods allow for the faster measurement of plaque volumes with only minor modifications compared to the traditional manual assessment methods. Plaque volumes measured using the semi-automated methods are highly reproducible and well correlated with those measured by invasive intravascular ultrasound (IVUS).[17,18] Therefore, CCTA can be considered a useful non-invasive tool for identifying the natural course of coronary atherosclerosis in subjects with low-to-intermediate cardiovascular risk.

Obesity is an important public health issue, and the prevalence of obesity is rapidly increasing. Clearly, obesity is strongly associated with adverse CAD events.[19,20] In the MESA (multi-ethnic study of atherosclerosis) study, asymptomatic obese subjects had an increased prevalence of CAD as defined by the coronary artery calcium score.[21] Moreover, subjects with increased BMI showed a greater prevalence, extent, and severity of CAD among subjects with suspected CAD who were referred for CCTA in the CONFIRM (COronary CT Angiography EvaluatioN For Clinical Outcomes: An InteRnational Multicentre) study.[5] However, these studies evaluated only the association between baseline CCTA findings and clinical outcome according to obese status. The PROSPECT (Providing Regional Observations to Study Predictors of Events in the Coronary Tree) study evaluated the natural history of coronary atherosclerosis using CCTA, but the study cohort was very small and all subjects were evaluated after acute coronary syndrome.[12] Thus, data on the change in patients' lifestyles after performing CCTA and the progression of coronary atherosclerosis according to BMI status in subjects with low-to-intermediate risk have been scarce in real clinical practice. The recent guidelines have strongly recommended both modest weight reduction of 5–10% of initial body weight, not absolute body weight, for the overweight or obese populations. It is well-known that the absolute body weight could be significantly influenced on the temporal lifestyle change (i.e. voiding, defecation, dehydration, and so on). To minimize these confounding effects, the present study focused on evaluating the PVC and annualized PVC according to the cut-off of 5% BMI change and categorical BMI group change together.

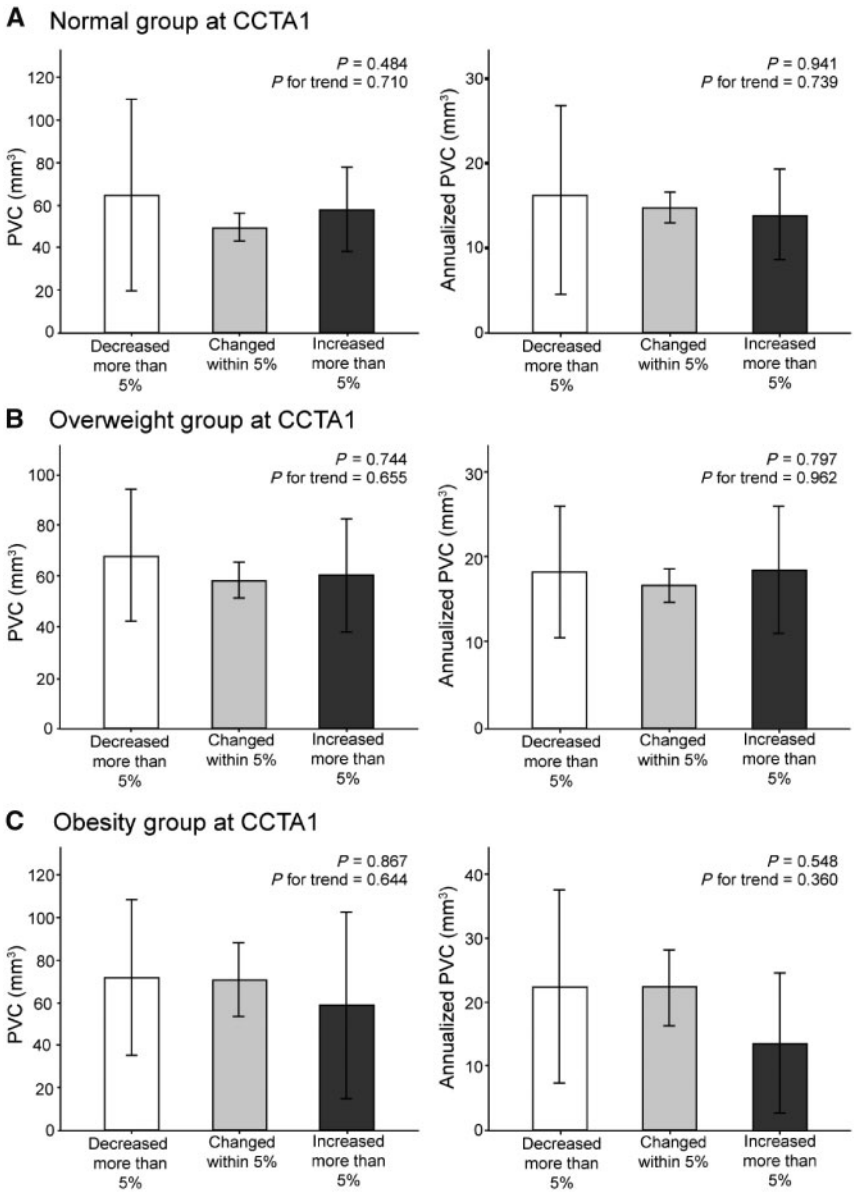

Figure 2 Coronary plaque progression according to the 5% of BMI change at CCTA2. (*A*) Normal, (*B*) overweight, and (*C*) obesity at CCTA1.

In the present study, the levels of LDL at CCTA2 were lower than those at CCTA1 without significant difference according to obese status. Likewise, the incidence of statin use increased considerably after the CCTAs in all groups. This might have been influenced by the results of previous studies demonstrating that strict LDL control had a beneficial effect on the coronary atherosclerosis.[22–24] During the median 3.3 years of follow-up, the change in BMI between CCTA1 and CCTA2 was observed in approximately 9% of the overall subjects. However, 17% of obese subjects had shifted to a decreased BMI group between CCTA1 and CCTA2. In addition, 17% of obese subjects had achieved more than 5% reduction of BMI at CCTA2 compared with the baseline BMI at CCTA1. Considering the negligible weight loss over 1 year in the overweight or obese survivors after acute myocardial infarction in the PREMIER (Prospective Registry Evaluating Myocardial Infarction: Event and Recovery) study,[25] this proportion of obese subjects might be meaningful in the present study. Notably, baseline overweight or obesity was independently related to the coronary plaque progression. However, no significant difference in the annualized PVC according to 5% of BMI change was observed in normal, overweight, and obesity group. In addition, subgroup analysis for subjects with obesity at CCTA1 showed that there were no significant differences in either PVC or annualized PVC between subjects with decreases in BMI of more than 10%, those with a change in BMI of less than 10%, and those with increases in BMI of more than 10% at CCTA2 compared to CCTA1 (Supplementary data online, *Figure S1*). This finding could be said to indicate that the beneficial effect of body weight reduction on coronary

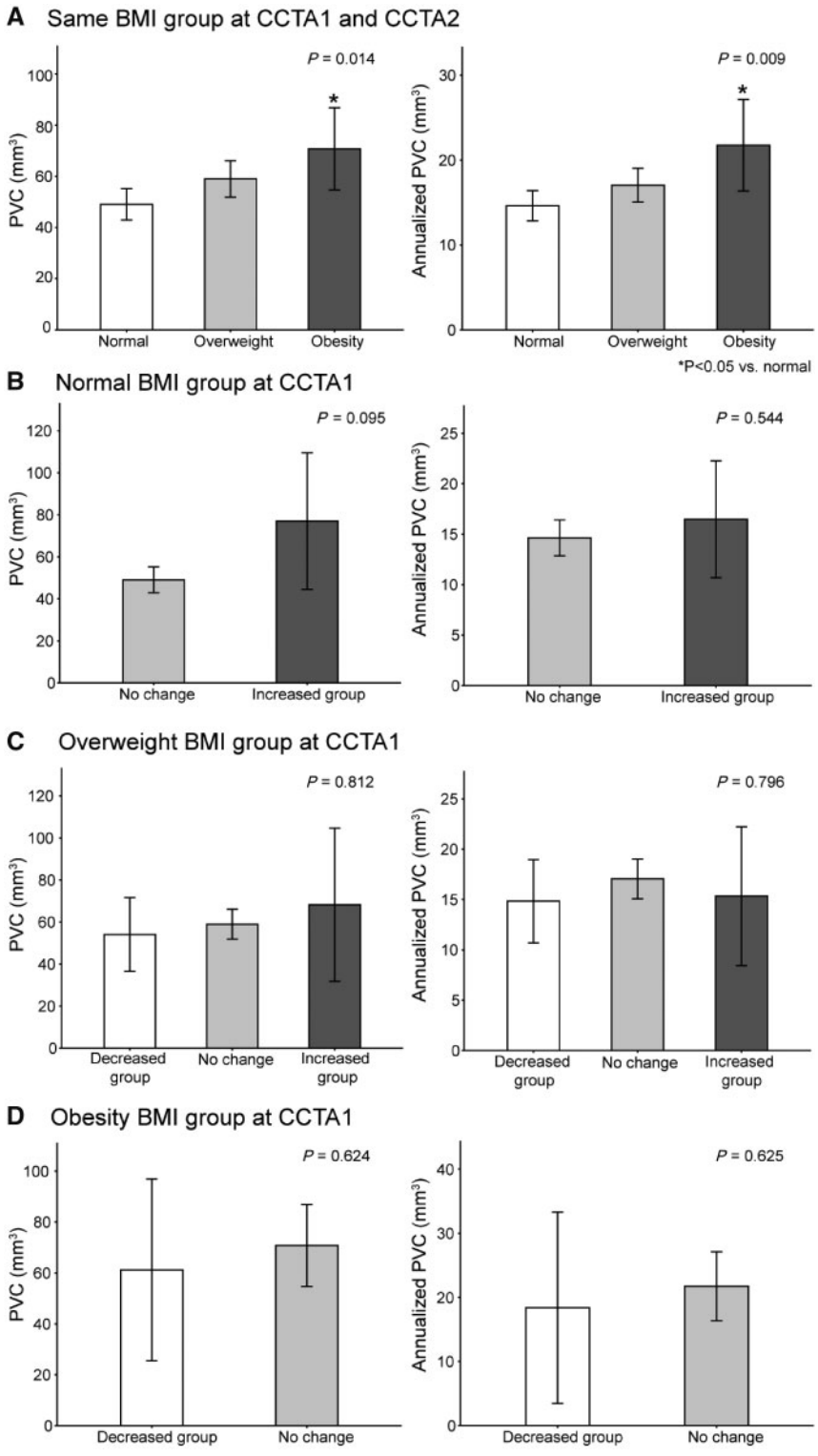

Figure 3 Comparison of coronary plaque progression based on BMI group at CCTA1 and CCTA2. (*A*) Same BMI group at CCTA1 and CCTA2; normal vs. overweight vs. obesity at CCTA2. (*B*) Normal at CCTA1; normal vs. overweight or obesity at CCTA2. (*C*) Overweight at CCTA1; overweight vs. normal vs. obesity at CCTA2. (*D*) Obesity at CCTA1; obesity vs. normal or overweight at CCTA2.

atherosclerosis was not fully reflected during the near-term period. To attenuate coronary plaque volume progression, medical therapy for traditional cardiovascular risk factors should be emphasized in overweight and obese subjects with low-to-intermediate risk in clinical practice.

The present study has some limitations. First, only baseline and final BMI data were available; therefore, longitudinal changes in BMI between CCTA could not be confirmed. Second, larger plaque volumes at baseline might lead to larger increases in plaque volume. However, when the baseline plaque volume was adjusted for, we were able to identify that baseline overweight or obesity markedly influenced plaque progression in addition to the baseline plaque volume. Third, despite the application of strict and standardized criteria for the CCTA assessment, atherosclerosis findings might be influenced by the Hounsfield unit density, which is affected by a myriad of factors. In addition, the different kVp setting according to obesity status could potentially have a confounding effect. Fourth, this study was performed in multi-ethnic population but participants were overwhelmingly composed of Caucasian and East Asian subjects. Finally, there was minimal change in BMI because of the observational design of the present study. Thus, further prospective and randomized studies with larger sample sizes are necessary to confirm the results of the present study. Despite these limitations, this study used CCTA to estimate plaque volume in a large cohort and identified the progression of coronary atherosclerosis according to the percent BMI change and categorical BMI group change in subjects with low-to-intermediate cardiovascular risk.

Table 3 Generalized linear model for the association between clinical variables and annualized PVC

	Multivariate beta	*P*-value
Age (years)	0.59	0.442
Male	0.10	0.749
Percentage change of BMI	3.25	0.072
Hypertension	4.10	0.043
Diabetes mellitus	4.53	0.034
Current smoking	0.39	0.531
Baseline plaque volume (mm^3)	419.48	<0.001
Statin use	0.63	0.426

BMI, body mass index; PVC, plaque volume change.

Conclusions

The present study demonstrated that neither 5% change of BMI nor categorical BMI group change were associated with coronary PVC based on serial quantitative assessment by CCTA during near-term period. This study could identify that male gender, baseline plaque volume, and baseline overweight or obesity had independent impact on coronary plaque progression. Further large prospective and randomized studies with longer follow-up duration might be necessary to confirm the results of present study.

Supplementary data

Supplementary data are available at *European Heart Journal - Cardiovascular Imaging* online.

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Conflict of interest: none declared.

Table 4 Independent predictors for coronary plaque progression

	Univariate		Multivariate	
	OR (95% CI)	*P*-value	OR (95% CI)	*P*-value
Age (years)	1.03 (1.02 - 1.04)	<0.001	1.01 (1.00 - 1.03)	0.099
Male	1.76 (1.37 - 2.25)	<0.001	1.38 (1.05 - 1.81)	0.022
Hypertension	1.72 (1.34 - 2.20)	<0.001	1.23 (0.94 - 1.63)	0.138
Diabetes mellitus	1.71 (1.22 - 2.38)	0.002	1.32 (0.93 - 1.88)	0.126
Dyslipidaemia	1.43 (1.11 - 1.84)	0.005	1.00 (0.738 - 1.35)	0.979
Current smoking	1.43 (1.01 - 2.02)	0.044	1.24 (0.85 - 1.81)	0.258
Baseline plaque volume (per 10 mm^3)	1.08 (1.06 - 1.10)	<0.001	1.07 (1.05 - 1.09)	<0.001
Baseline categorical BMI group				
Normal	1		1	
Overweight or obesity	1.54 (1.21 - 1.98)	0.001	1.35 (1.04 - 1.77)	0.027
Statin use	1.77 (1.37 - 2.30)	<0.001	1.34 (0.99 - 1.81)	0.054

BMI, body mass index; CI, confidence interval; OR, odds ratio.

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Corrigendum

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In the original article, the affiliations of authors F. Cademartiri and E. Maffei were incorrect. These have now been corrected.