Comparison Of Apo B/A1, Lipoprotein (A) And LDL-C/Apo B For Predicting Coronary Atherosclerosis In Patients ≤ 65 Years With Low LDL-C

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Abstract

Background: High low-density lipoprotein cholesterol (LDL-C) and old age are major risk factors for coronary artery disease. However, the risk factors for coronary artery disease in relatively young patients with low LDL-C have not been elucidated. Therefore, we performed this study to evaluate the risk factors of coronary artery disease, in patients under 65 years with low LDL-C.

Methods: This study included 622 lipid-lowering agent naive patients ≤ 65 years with LDL-C < 127.5 mg/dL who underwent coronary computed tomographic angiography. We performed multivariate logistic regression analysis to evaluate the risk factors of coronary atherosclerosis, defined as the presence of coronary plaque. In addition to traditional cardiovascular risk factors, the covariate included novel lipid profiles (apolipoprotein (Apo) B/Apo A1) ratio, LDL-C/Apo B ratio, and lipoprotein (a)).

Results: Of the total 622 patients, the mean age was 54.4 ± 8.9 and the mean LDL-C was 91.0 ± 23.1 mg/dL. Multivariate regression analysis showed that age, male, diabetes mellitus, and hypertension were independent predictors of coronary atherosclerosis. A high LDL-C/Apo B ratio was a negative predictor of coronary atherosclerosis.

Conclusions: A low LDL-C/Apo B ratio is associated with the development of coronary atherosclerosis in patients ≤ 65 years with low LDL-C levels.

Introduction

High low-density lipoprotein cholesterol (LDL-C) and old age are major risk factors for coronary artery disease [1–4]. Won et al. reported that age ≥ 65 years, LDL-C level ≥ 127.5 mg/dL, and male sex were the major risk factors for subclinical coronary atherosclerosis in patients without traditional cardiovascular risk factors [1]. However, risk factors for coronary artery disease in relatively young patients and low LDL-C have not been elucidated. In addition, the apolipoprotein (Apo) B/Apo A1 ratio, lipoprotein (a) (Lp (a)) level, and LDL-C/Apo B ratio have been useful for predicting atherosclerotic cardiovascular disease (ASCVD) and mortality [5–10]. However, it is not known whether these novel lipid profiles are better predictors of coronary artery disease in relatively young patients with low LDL-C compared to the conventional lipid profiles. Therefore, we performed this study to evaluate the risk factors of coronary artery disease, including traditional cardiovascular risk factors, and novel lipid profiles, in patients under 65 years with LDL-C < 127.5 mg/dL.

Methods

Study population and data collection

The study population was selected from the coronary computed tomographic angiography (CCTA) registry of the Daegu Catholic Medical Center (Daegu, Korea). Between January 2013 and September 2020, 3,696 patients who visited our hospital for chest discomfort underwent CCTA and lipid profile

evaluation (total cholesterol (TC), LDL-C, high-density lipoprotein cholesterol (HDL-C), triglyceride (TG), APO A1, APO B, and Lp (a)). Among the 3,696 patients, 1,162 patients were ≤ 65 years and had LDL-C levels < 127.5 mg/dL. Of the 1,162 patients, 298 patients who underwent percutaneous coronary intervention (PCI) or cardiac surgery, 217 patients who were maintained on lipid-lowering drugs, 20 patients with end-stage renal disease, and 5 patients with motion artifacts on CCTA were excluded. Finally, 622 patients were included in the analysis. The inclusion and exclusion criteria are shown in a flow diagram (Figure 1).

Acquisition and analysis of CCTA images

CT scans were performed with a 256-slice CT (Definition Flash; Siemens Healthineers AG) or a 512-slice CT (Revolution CT; GE Healthcare). All patients with an initial heart rate ≥ 60 beats/min were given an oral beta-blocker (propranolol 20 mg) to achieve a target heart rate of 50 to 60 beats/min. Sublingual nitroglycerin was administered immediately before scanning. An iodine contrast agent (60–70 mL) was administered into the antecubital vein within 10 seconds followed by 25 mL of saline solution injected at 5.0 mL/second. The CT-reconstructed imaging data were transferred to a GE Centricity system (GE Healthcare Bio-Sciences Corp) for postprocessing and subsequent image analysis. A radiologist read each scan independently at a central reading center. Atherosclerosis was defined as the presence of coronary plaque. Plaques were defined as structures ≥ 1 mm² within and/or adjacent to the vessel lumen and were clearly distinguishable from the lumen and the surrounding pericardial tissue [1]. Stenosis of 50% or more in one vessel was defined as 1 vessel disease, and stenosis of 50% or more in two or more vessels was defined as multivessel disease. The coronary artery calcium score (CACS) was acquired with the Agatston method using a commercially available reconstruction program for three-dimensional reconstruction and measurement (Aquarius iNtuition TM Ver.4.4.12 TeraRecon) [11, 12]. CACS > 0 was defined as having a detectable coronary artery calcium [13]. After CCTA evaluation, coronary revascularization (PCI or coronary artery bypass graft (CABG)) was performed according to the judgment of the attending physician. The coronary revascularization rate was investigated. All methods were performed in accordance with the relevant guidelines and regulations.

Statistical analysis

Data were expressed as number (%) and mean $\pm$ standard deviation. Categorical data were compared using the Chi-square test or Fisher's exact test. Continuous variables were compared using the Student's t-test and Kruskal–Wallis H test when they were normally and non-normally distributed, respectively. Univariate analysis using logistic regression was performed to identify potential independent predictors for coronary atherosclerosis. Age, sex, and variables with a p-value < 0.05 in the univariate analysis were included in the multivariate analysis to determine the independent predictors for coronary atherosclerosis. Diagnostic accuracy of lipid profiles on a continuous scale in the prediction of coronary atherosclerosis was estimated as the area under the curve (AUC) from receiver operating characteristic (ROC) curves. Univariate analysis using logistic regression was performed to identify potential independent predictors of the lowest tertile of the LDL-C/Apo B ratio. Age, sex, and variables with a p-value < 0.05 were included in

the multivariate logistic regression analysis. Pearson's correlation analysis was used to assess the correlations between various parameters and the LDL-C/Apo B ratio. A p-value<0.05 was considered statistically significant. Statistical analyses were performed using SPSS version 25.0 (IBM, Armonk, NY, USA).

Results

Of the total 622 patients, the mean age was 54.4 ± 8.9 years and the mean LDL-C level was 91.0 ± 23.1 mg/dL. 63.2% were male and 23.2% had diabetes. Coronary revascularization was performed in 13%. Atherosclerosis, defined as the presence of coronary plaque, was present in 61.1% of the total patients. The clinical characteristics of the patients according to presence of plaque are shown in **Table 1**. In the plaque group compared to the no plaque group, age and fasting blood glucose were significantly higher, and estimated glomerular filtration rate and body mass index were significantly lower. The proportion of male, diabetes mellitus, hypertension, and smoking patients were significantly higher in the plaque group compared to the no plaque group. In the comparison of lipid profiles, the plaque group showed significantly lower TC, HDL-C, Apo A1, and LDL-C/ Apo B ratio compared to the no plaque group. Lp (a) was significantly higher in the plaque group compared to the no plaque group.

Independent predictors of atherosclerosis

According to the multivariate logistic regression analysis, age (odds ratio (OR): 1.108; 95% confidence interval (CI): 1.076–1.140, $p<0.001$), male sex (OR: 3.912; 95% CI: 2.443–6.264, $p<0.001$), diabetes mellitus (OR: 2.354; 95% CI: 1.369–4.048, $p=0.002$) and hypertension (OR: 2.260; 95% CI: 1.446–3.532, $p<0.001$) were significantly associated with coronary atherosclerosis. Meanwhile, a high LDL-C/Apo B ratio was a negative predictor of coronary atherosclerosis (OR: 0.277; 95% CI: 0.077–0.994, $p=0.049$) (**Table 2**). According to the ROC analyses, the best predictive value for coronary atherosclerosis was an LDL-C/Apo B ratio ≤ 1.106 (AUC=0.598 $p=0.000$; sensitivity, 57.0%; specificity, 57.4%). The AUC for the LDL-C/Apo B ratio was larger than that of the Apo B/Apo A1 ratio and HDL-C, LDL-C, Lp (a), and TG (Apo B/Apo A1 ratio: AUC=0.540, $p=0.096$; HDL-C: AUC=0.583, $p<0.001$; LDL-C: AUC=0.455, $p=0.057$; Lp (a): AUC=0.522, $p=0.351$; TG: AUC=0.565, $p=0.007$)

Characteristics according to tertiles of the LDL-C/Apo B ratio

The clinical characteristics of the patients stratified by tertiles of the LDL-C/Apo B ratio are shown in **Table 3**. The cut-off points between tertiles were 1.0275 (between the first and second tertile) and 1.1666 (between the second and third tertile). The averages of the LDL-C/Apo B ratio of tertiles 1, 2, and 3 was 0.908 ± 0.126 , 1.099 ± 0.039 , and 1.268 ± 0.095 , respectively. In tertile 1, there was significantly more male and diabetic patients than in the other 2 tertiles. Fasting blood glucose was significantly higher in tertile 1 than in the other 2 tertiles. In terms of CCTA characteristics, the proportion of patients with CAC exceeding 0, plaque, and multivessel disease were significantly higher in tertile 1 than in the other 2 tertiles. After CCTA, more coronary revascularization was performed in tertile 1 compared to the other 2 tertiles.

Independent predictors of the lowest tertile for the LDL-C/Apo B ratio

In the multivariate logistic regression analysis, diabetes mellitus (OR: 1.816; 95% CI: 1.196–2.756, $p=0.005$) and hypertriglyceridemia (OR: 1.006; 95% CI: 1.003–1.008, $p<0.001$) were the independent predictors of the lowest tertile for the LDL-C/Apo B ratio (**Table 4**).

Correlation analysis between variables and the LDL-C/Apo B ratio

The results of Pearson's correlation analysis between the LDL-C/ Apo B ratio and other clinical factors are presented in **Supplementary Table 1**. TG and HDL levels had the strongest correlation with the LDL-C/Apo B ratio (TG: correlation coefficient = -0.291, $p<0.001$; HDL-C: correlation coefficient = 0.291, $p<0.001$) as depicted in **Figure 2**.

Discussion

The primary findings of present study were as follows: 1) A low LDL-C/Apo B ratio was an independent predictor of coronary atherosclerosis in patients ≤ 65 years with low LDL cholesterol; 2) The LDL-C/Apo B ratio demonstrated a greater predictive accuracy for coronary atherosclerosis compared with other lipid profiles, including Apo B/Apo A1 ratio and Lp (a), in patients < 65 years with low LDL cholesterol; 3) Diabetes mellitus and hypertriglyceridemia were significantly associated with a low LDL-C/Apo B ratio.

Currently, lowering the LDL-C is the main treatment strategy to prevent ASCVD [14-15]; however, ASCVD still occurs in patients with low LDL-C. Therefore, studies on lipoproteins to evaluate residual cardiovascular risk in patients with low LDL-C are ongoing. In this study, the LDL-C/Apo B ratio was an independent predictor of coronary atherosclerosis. On the other hand, Apo B/Apo A1 and Lp (a) did not predict coronary atherosclerosis and showed a lesser predictive accuracy for coronary atherosclerosis compared with the LDL-C/Apo B ratio. In previous studies, the LDL-C/Apo B ratio has been identified as a marker of small dense low-density lipoprotein (sdLDL). Several papers have reported that sdLDL increases the risk of ASCVD [16-18]. Increased atherogenesis of sdLDL is associated with specific biochemical properties of these particles. The small size makes penetration into the arterial wall easier, and smaller LDL particles have decreased receptor mediated uptake. Therefore, the half-life of sdLDL is longer than that of large LDL particles. A longer circulation time increases the possibility of atherogenic modifications of sdLDL in the blood [19, 20]. Hirano et al. found that an LDL-C/Apo B ratio of 1.2 corresponds to an LDL diameter of 25.5 nm, which has been proposed as a cut-off value between sdLDL and large buoyant LDL [21]. In our study, the best predictive value for coronary atherosclerosis was an LDL-C/Apo B ratio ≤ 1.106 . This difference seems to be due to the difference between the biochemical and clinical values.

A low LDL-C/Apo B ratio has been associated with increased cardiovascular events [9,10]. In our study, the proportion of patients with CAC exceeding 0, with multivessel disease, and who underwent revascularization were highest in the lowest tertile of the LDL-C/Apo B ratio. These results suggest that

sdLDL affects not only the occurrence of plaque but also its severity which are consistent with the findings of Koba et al [22].

This study focused on the role of sdLDL in coronary atherosclerosis in patients with LDL-C <127.5 mg/dL. In Heinz's paper, subgroup analysis according to the LDL-C level showed that sdLDL was an independent predictor for major cardiovascular events only in the group with LDL-C below 121 mg/dL but not above 121 mg/dL [10]. These findings suggest that the impact of sdLDL on coronary atherosclerosis is greater in patients with low LDL-C than in patients with high LDL-C. Meanwhile, previous articles revealed that the ASCVD risk associations with Lp (a) are attenuated with low LDL-C [7,23]. Thus, different types of lipid profiles may better represent residual cardiovascular risk depending on a patient's LDL-C level.

The LDL-C/Apo B ratio cutoff value of 1.106 in this study is much lower than the cutoff value of 1.46 suggested by Heinz et al. [10], indicating that the primary endpoints in our study were less sensitively affected by the LDL-C/Apo B ratio as compared to their study. The difference in the cutoff values of the LDL-C/Apo B ratio between the two studies could be due to the following reasons. First, Heinz et al. conducted a study on patients with coronary artery disease and peripheral artery disease; in contrast, our study was for patients who had never been diagnosed with coronary artery disease. Second, there were no restrictions on the age or LDL-C in the study of Heinz et al. The cut-off value of the LDL-C/Apo B ratio may vary depending on the target patient group. A larger study is necessary to determine a universal clinical cut-off value of the LDL-C/Apo B ratio.

Previous studies have demonstrated a high sdLDL level is associated with hypertriglyceridemia, metabolic syndrome, and diabetes [24-27]. As in previous studies, in our study, diabetes mellitus (OR: 1.816; 95% CI: 1.196–2.756, $p=0.005$) and hypertriglyceridemia (OR: 1.006; 95% CI: 1.003–1.008, $p<0.001$) were independent predictors of lowest tertile of the LDL-C/Apo B ratio. Treatment criteria for sdLDL are not yet clear. However, in patients with diabetes or hypertriglyceridemia, clinicians can additionally measure the LDL-C/Apo B ratio to further reduce the cardiovascular risk of patients even after satisfying the LDL-C target. Medications to reduce sdLDL have already been reported. Bahadir et al. reported significant reduction of the sdLDL after statin in hypercholesterolemic patients with metabolic syndrome [28]. In addition to statin, triglyceride-lowering treatments such as fibrates and omega-3 fatty acids, can be considered in patients with the high sdLDL since the formation of sdLDL is associated with hypertriglyceridemia [29,30].

This study has strengths. First, this study is the first to compare the effects of Apo B/Apo A1, Lp (a), and LDL-C/Apo B on coronary atherosclerosis in patients with low LDL-C. Second, unlike previous studies that reported the relationship between the LDL-C/Apo B ratio and cardiovascular events [9,10], our study reports the relationship between the LDL-C/Apo B ratio and the coronary plaque. Third, the present study was conducted on relatively young patients. Therefore, we investigated the role of the LDL-C/Apo B ratio in the early stages of atherosclerosis. However, this study has limitation. First, this was a single-center study. Second, the study population were comprised of Korean; studies with other races are necessary to

confirm and generalize our findings. Third, because our study targeted patients who underwent CCTA for chest discomfort, the plaque incidence was high. Therefore, in order to apply the results of this study to the general population, a large-scale study including asymptomatic patients should be conducted.

Declarations

Ethics approval and consent to participate.

The protocol of the present study was approved by the institutional review board of daegu catholic university medical center. The institutional review board of daegu catholic university medical center waived the requirement for patient informed consent because of the study's retrospective nature.

Data availability

All data generated or analyzed during this study are included in this published article

Author contributions

All authors have made substantial contributions. H.W.J. and H.J.B. performed the statistical analysis. H.W.J. and H.J.B. contributed to data acquisition. H.W.J. and H.J.B. contributed to the interpretation of data. H.W.J. drafted the manuscript. S.P.H. critically revised the manuscript. All authors read and approved the final manuscript. All authors also gave final approval and agree to be accountable for all aspects of work ensuring integrity and accuracy.

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Competing interests

The authors declare no competing interests.

Conclusion

A low LDL-C/Apo B ratio is significantly associated with the development of coronary atherosclerosis in patients ≤ 65 years with low LDL-C levels. These findings support the need for detection and treatment of a low LDL-C/Apo B ratio for preventing the development of coronary atherosclerosis in relatively young patients with low LDL-C.

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Tables

Table 1. Characteristics of individuals according to presence of plaque.

Variables	Total patients (n=622)	No plaque (n=242)	Plaque (n=380)	p-value
Clinical characteristics				
Age, years	54.4 ± 8.9	49.9 ± 10.5	57.2 ± 6.3	<0.001
Male	393 (63.2)	107 (44.2)	286 (75.3)	<0.001
Diabetes	144 (23.2)	27 (11.2)	117 (30.8)	<0.001
Hypertension	240 (38.6)	61 (25.2)	179 (47.1)	<0.001
Smoking	208 (33.4)	64 (26.4)	144 (37.9)	0.003
SBP (mmHg)	126.1 ± 18.0	124.9 ± 18.2	126.8 ± 17.8	0.189
DBP (mmHg)	75.6 ± 12.1	75.2 ± 13.0	75.8 ± 11.6	0.534
BMI (kg/m ²)	24.4 ± 3.64	24.9 ± 4.23	24.1 ± 3.19	0.017
eGFR (ml/min/1.73 m ²)	94.1 ± 18.9	99.8 ± 17.7	90.5 ± 18.8	<0.001
EF (%)	56.3 ± 12.2	56.7 ± 12.1	56.1 ± 12.3	0.560
Fasting blood glucose	114.4 ± 44.5	107.3 ± 44.6	118.8 ± 44.0	0.002
Lipid profile				
TC (mg/dL)	150.6 ± 27.0	153.7 ± 26.1	148.7 ± 27.4	0.028
TG (mg/dL)	121.1 ± 92.5	112.4 ± 89.5	126.6 ± 94.1	0.069
HDL-C (mg/dL)	46.9 ± 15.2	49.3 ± 15.7	45.3 ± 14.6	0.001
LDL-C (mg/dL)	91.0 ± 23.1	93.2 ± 21.9	89.6 ± 23.7	0.056
Lp (a) (mg/dL)	20.8 ± 24.8	18.3 ± 20.0	22.4 ± 27.3	0.046
Apo B (mg/dL)	83.5 ± 18.5	83.4 ± 17.2	83.7 ± 19.4	0.835
Apo A1 (mg/dL)	128.4 ± 30.1	132.2 ± 28.8	126.0 ± 30.7	0.012
Apo B/Apo A1	0.695 ± 0.267	0.665 ± 0.216	0.714 ± 0.294	0.026
LDL-C/Apo B	1.091 ± 0.174	1.12 ± 0.178	1.07 ± 0.169	0.001

Data are given as mean ± SD, or as number (%). Apo A1; apolipoprotein A1, Apo B; apolipoprotein B, BMI; body mass index, CACS; coronary artery calcium score, CCTA; coronary computed tomographic angiography, EF; ejection fraction, eGFR; estimated glomerular filtration rate, HDL-C; high-density lipoprotein cholesterol, LDL-C; low-density lipoprotein cholesterol, Lp (a); lipoprotein (a), TC; total cholesterol, TG; triglyceride

Table 2. Independent predictors for coronary atherosclerosis.

Variables	Univariate analysis			Multivariate analysis		
	OR	95% CI	p-value	OR	95% CI	p-value
Age	1.112	1.086–1.138	<0.001	1.108	1.076–1.140	<0.001
Male	3.839	2.720–5.417	<0.001	3.912	2.443–6.264	<0.001
Diabetes	3.542	2.246–5.587	<0.001	2.354	1.369–4.048	0.002
Hypertension	2.642	1.856–3.762	<0.001	2.260	1.446–3.532	<0.001
Smoking hx	1.697	1.192–2.415	0.003	0.980	0.603–1.593	0.935
BMI	0.948	0.906–0.991	0.018	0.946	0.889–1.006	0.077
SBP	1.006	0.997–1.015	0.189			
DBP	1.004	0.991–1.018	0.534			
eGFR	0.969	0.959–0.980	<0.001	0.990	0.978–1.003	0.139
LDL-C	0.993	0.986–1.000	0.056			
HDL-C	0.982	0.972–0.993	0.001	0.993	0.975–1.012	0.464
TG	1.002	1.000–1.004	0.073			
Apo B/Apo A1	2.089	1.085–4.022	0.028	1.093	0.387–3.086	0.867
Lp (a)	1.007	1.000–1.014	0.048	1.007	0.998–1.016	0.147
LDL-C/Apo B	0.184	0.068–0.496	0.001	0.277	0.077–0.994	0.049

Apo A1; apolipoprotein A1, Apo B; apolipoprotein B, BMI; body mass index, CI; confidence interval, DBP: diastolic blood pressure, eGFR: estimated glomerular filtration rate, HDL-C; high-density lipoprotein cholesterol, HR; hazard ratio, LDL-C; low-density lipoprotein cholesterol, Lp (a); lipoprotein (a), OR; odd ratio, SBP: systolic blood pressure, TG; triglyceride.

Table 3. Characteristics of individuals stratified by tertiles of the LDL-C/ Apo B ratio.

Data are given as mean $\pm$ SD, or as number (%). Apo A1; apolipoprotein A1, Apo B; apolipoprotein B, BMI; body mass index, CACS; coronary artery calcium score, CCTA; coronary computed tomographic angiography, EF; ejection fraction, eGFR; estimated glomerular filtration rate, HDL-C; high-density lipoprotein cholesterol, LDL-C; low-density lipoprotein cholesterol, Lp (a); lipoprotein (a), TC; total cholesterol, TG; triglyceride

Table 4. Independent predictors for lowest tertile of the LDL-C/ Apo B ratio.

Variables	Total patients (n=622)	Tertile 1 of the LDL-C/ Apo B ≤ 1.0275 (n=207)	Tertile 2 of the LDL-C/ Apo B 1.0275–1.1666 (n=210)	Tertile 3 of the LDL-C/ Apo B >1.1666 (n=205)	p-value
Clinical characteristics					
Age, years	54.4 $\pm$ 8.9	54.2 $\pm$ 8.1	54.1 $\pm$ 9.6	54.8 $\pm$ 9.1	0.686
Male	393 (63.2)	145 (70.0)	138 (65.7)	110 (53.7)	0.002
Diabetes	144 (23.2)	68 (32.9)	48 (22.9)	28 (13.7)	<0.001
Hypertension	240 (38.6)	89 (43.0)	79 (37.6)	72 (35.1)	0.244
Smoking	208 (33.4)	77 (37.2)	70 (33.3)	61 (29.8)	0.277
SBP (mmHg)	126.1 $\pm$ 18.0	127.3 $\pm$ 18.9	126.8 $\pm$ 18.4	124.1 $\pm$ 16.4	0.152
DBP (mmHg)	75.6 $\pm$ 12.1	76.3 $\pm$ 12.9	76.2 $\pm$ 12.0	74.3 $\pm$ 11.4	0.180
BMI (kg/m ²)	24.4 $\pm$ 3.64	24.6 $\pm$ 3.87	24.6 $\pm$ 3.63	24.0 $\pm$ 3.40	0.216
eGFR, (ml/min/1.73 m ²)	94.1 $\pm$ 18.9	94.5 $\pm$ 21.4	94.1 $\pm$ 17.7	93.7 $\pm$ 17.4	0.904
EF (%)	56.3 $\pm$ 12.2	55.0 $\pm$ 12.9	55.3 $\pm$ 12.7	58.7 $\pm$ 10.5	0.004
Fasting blood glucose	114.4 $\pm$ 44.5	121.6 $\pm$ 58.3	114.6 $\pm$ 34.7	106.8 $\pm$ 35.3	0.004
CCTA characteristics					
CACS	198.8 $\pm$ 580.1	254.9 $\pm$ 678.5	165.9 $\pm$ 495.4	175.9 $\pm$ 550.8	0.060
CAC> 0	305 (49.0)	121 (58.5)	103 (49.0)	81 (39.5)	0.001
plaque	380 (61.1)	148 (71.5)	129 (61.4)	103 (50.2)	<0.001
1 vessel disease	107 (17.2)	37 (17.9)	37 (17.6)	33 (16.1)	0.875
Multivessel disease	60 (9.6)	29 (14.0)	15 (7.1)	16 (7.8)	0.033
Revascularization	81 (13.0)	37 (17.9)	22 (10.5)	22 (10.7)	0.040

Variables	Univariate analysis			Multivariate analysis		
	OR	95% CI	p-value	OR	95% CI	p-value
Age	0.998	0.979–1.017	0.823	0.992	0.973–1.013	0.465
Male	1.575	1.103–2.249	0.012	1.196	0.806–1.775	0.375
Diabetes	2.182	1.489–3.198	<0.001	1.816	1.196–2.756	0.005
Hypertension	1.319	0.938–1.853	0.111			
Smoking hx	1.284	0.905–1.822	0.161			
BMI	1.021	0.976–1.069	0.368			
eGFR	1.002	0.993–1.011	0.686			
HDL-C	0.965	0.953–0.978	<0.001	0.994	0.969–1.012	0.672
TG	1.006	1.004–1.008	<0.001	1.006	1.003–1.008	<0.001
Apo A1	0.989	0.983–0.995	<0.001	0.990	0.978–1.002	0.106
Lp (a)	1.002	0.996–1.009	0.531			

Apo A1; apolipoprotein A1, Apo B; apolipoprotein B, BMI; body mass index, CI; confidence interval, eGFR: estimated glomerular filtration rate, HDL-C; high-density lipoprotein cholesterol, HR; hazard ratio, LDL-C; low-density lipoprotein cholesterol, Lp (a); lipoprotein (a), OR; odd ratio, TG; triglyceride.

Figures

Figure 1

Enrollment flow chart for analysis. CCTA; coronary computed tomographic angiography, DCMC; Daegu catholic medical center, ESRD; end-stage renal disease, LDL-C; low-density lipoprotein cholesterol, PCI; percutaneous coronary intervention.

Figure 2

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