Remnant lipoproteins play an important role of in-stent restenosis in type 2 diabetes undergoing percutaneous coronary intervention: a single-centre observational cohort study

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Abstract
Background: Increasing evidence has suggested that the presence of remnant lipoproteins is a significant risk factor for atherosclerosis. Remnant lipoproteins are lipoproteins that are rich in triglycerides (TGs), and the main components include very-low-density lipoprotein (VLDL) in the fasting state. Diabetic patients often have hypertriglyceridemia with elevated levels of VLDL cholesterol but normal levels of low-density lipoprotein cholesterol (LDL-C). The aim of the present study was to elucidate the potential role of remnant lipoproteins-induced atherosclerosis in the occurrence and development of in-stent restenosis (ISR) in diabetic patients with coronary artery disease.

Methods: The present study enrolled 2312 patients with type 2 diabetes mellitus who underwent percutaneous coronary intervention from January 2013 to December 2014 and who were followed up by angiography. Patients were divided into two groups based on the presence or absence of ISR, and multivariate Cox's proportional hazards regression modelling showed that remnant-like particle cholesterol (RLP-C) was an independent risk factor for ISR. According to the receiver operating characteristic curve, the optimal cutoff point of the RLP-C was identified, and the patients were further divided into 2 groups. Propensity score matching analysis was performed, and 762 pairs were successfully matched. Log-rank tests were used to compare Kaplan–Meier curves for overall follow-up to assess ISR.

Results: The multivariate Cox’s proportional hazards regression analysis showed that RLP-C was independently associated with ISR, and the baseline RLP-C level at 0.505 mmol/L was identified as the optimal cutoff point to predict ISR. Patients were divided into 2 groups by RLP levels. After propensity score matching analysis, a total of 762 pairs matched patients were generated. Kaplan–Meier curves showed that the estimated cumulative rate of ISR was significantly higher in patients with RLP-C levels ≥ 0.505 mmol/L (log-rank P < 0.001; HR equal to 4.175, 95% CI = 3.045–5.723, P < 0.001) compared to patients with RLP-C levels < 0.505 mmol/L.
**Background**

In-stent restenosis is an important factor for successful coronary intervention (PCI). Several large-scale clinical trials have confirmed that the occurrence rate of in-stent restenosis (ISR) ranges from 3 to 20% after coronary stenting implantation, even in the drug-eluting stents (DES) era [1]. Patients with diabetes mellitus (DM) have a 2–4 times higher risk of developing ISR after PCI compared with non-diabetic patients [2, 3]. The poor prognosis [4, 5] of this particular population deserves additional attention.

The high rate of ISR may be related to dyslipidaemia in DM. A recent multicentre study has shown that the prevalence of dyslipidaemia has reached up to 67.1% among diabetic patients in China [6] and is uniquely manifested by high levels of triglycerides (TGs) and very-low-density lipoprotein cholesterol (VLDL-C) but normal levels of low-density lipoprotein cholesterol (LDL-C) [7]. Remnant lipoproteins are lipoproteins that are rich in triglycerides (TGs), and the main components include VLDL in the fasting state [8]. Therefore, diabetic patients have abnormal lipid metabolism mainly due to increased remnant-like particle cholesterol (RLP-C). Recent studies have shown that high levels of remnant lipoproteins can predict coronary events in diabetic patients independent of the degree of coronary stenosis, age, gender, hypercholesterolaemia, low-density lipoprotein, hypertriglyceridaemia and other risk factors [9]. Therefore, it is of great significance to elucidate the role of RLP-induced atherosclerosis in diabetic patients.

**Methods**

**Study patients**

The present study is a retrospective cohort study of 2701 coronary artery disease (CAD) patients with Type 2 diabetes mellitus (T2DM) who underwent successful coronary second-generation drug-eluting stents (G2-DESs) implantation at Beijing Anzhen Hospital (Beijing, China) from January 2013 to December 2014 and were followed up by angiography. Patients who died in the hospital after baseline PCI or without sufficient clinical and angiographic data at baseline and follow up were excluded. Of these patients, 2312 patients who met the inclusion and exclusion criteria were analysed in the present study. Multivariate Cox’s proportional hazards regression modelling showed that RLP-C was an independent risk factor for ISR. According to the receiver operating characteristic (ROC), the optimal cutoff point of the RLP-C was identified, and the patients were divided into the following 2 groups: low RLP-C group (n = 1072) and high RLP-C group (n = 1240). Propensity score matching analysis was performed in the two groups with a proportion of 1:1, including baseline data (age, gender, BMI, duration of diabetes mellitus, symptom-driven hospitalization and SYNTAX score). Finally, 762 pairs of DM patients were successfully matched. Log-rank tests were used to compare Kaplan–Meier curves for overall follow-up to assess ISR between the two groups.

**Stent implantation**

All enrolled patients received G2-DESs implantations in the catheterization centre. The type of G2-DESs included zotarolimus-eluting stents (Endeavor and Endeavor Resolute; Medtronic Vascular, USA), domestic sirolimus-eluting stents (Firebird2; MicroPort Medical, China), everolimus-eluting stents (Xience V and Xience Prime; Abbott Vascular, USA, Promus and Promus Element; Boston Scientific, USA). Stent implantation was performed according to current practice guidelines, and stents were selected by experienced interventional cardiologists. During the procedure, patients received a bolus of 100 IU/kg heparin with a repeated bolus of 2000 IU heparin to maintain the activated clotting time of ≥ 300 s. All patients received aspirin (100 mg/day was administered) and clopidogrel (300 mg loading dose followed by 75 mg/day for at least 12 months). When ISR was diagnosed, patients were treated with re-DES implantation. Procedural success was defined as follows: reduction of stenosis to less than 10% residual narrowing; thrombolysis in myocardial infarction (TIMI) flow grade III; improvement in ischaemic symptoms; and no major procedure related complications [7].

**Data collection**

A standard case report form (CRF) was used to collect patients’ demographic and clinical characteristics, including age, gender, smoking, drinking, CAD risk factors, family history, life style, medical history and coronary angiographic information at baseline PCI and follow-up angiography. During a physical examination,
anthropometric indices, such as weight, height and blood pressure (BP), were measured. Body mass index (BMI) was calculated as weight in kilograms divided by the square of the height in metres.

Coronary angiogram data, such as minimal stent diameter, average stent length and stenosis percent, were also recorded by two experienced investigators at baseline and follow-up for coronary angiography analysis.

**Laboratory analysis**

Venous blood samples were collected after an overnight fast for testing lipid profiles, HbA1c, fasting blood glucose (FBG), high-sensitivity C-reactive protein (hs-CRP) and uric acid (UA) levels using standard laboratory methods at baseline PCI and follow-up angiography.

The HbA1c was tested using ion exchange high-performance liquid chromatograph (HPLC) method. Blood samples for lipid profiles were collected from patients taking statin for more than 2 weeks. The total cholesterol (TC), TG, FBG and UA levels were determined according to enzymatic methods. LDL-C and high-density lipoprotein cholesterol (HDL-C) levels were measured by homogeneous assays. RLP-C levels were calculated as TC minus LDL-C and HDL-C according to the recommendation of dyslipidaemia guidelines [10, 11].

**Disease definitions**

The primary end point of the present study was the occurrence of ISR. ISR was defined as a diameter stenosis of ≥ 50% occurring in the segment inside the stent, 5 mm proximal to the stent or 5 mm distal to the stent at follow-up angiography [12]. The target lesion was considered as the most severe narrowing vessel identified by angiographic appearance with electrocardiograph (ECG) changes. Multivessel disease (MVD) was defined as a diameter stenosis of ≥ 50% occurring in 2 or more vessels.

Diabetes mellitus was defined as either a previous diagnosis of DM (treated with diet, oral agents or insulin) or a new diagnosis of DM (FBG ≥ 7.0 mmol/L on 2 occasions during hospitalization) [13]. Hypertension was defined by systolic blood pressure (SBP) ≥ 140 mmHg, diastolic blood pressure (DBP) ≥ 90 mmHg and/or the use of antihypertensive treatment in the past 2 weeks [14]. The severity of coronary artery lesions was quantified by angiographic appearance with electrocardiograph changes. Multivessel disease (MVD) was defined as a diameter stenosis of ≥ 50% occurring in 2 or more vessels.

**Statistical analysis**

Continuous variables were expressed as the mean (X̄) ± standard deviation (SD) in the case of normal distribution, and differences between two groups were determined by two-sided t-test. Data were expressed as medians (interquartile ranges, P25 and P75) in the case of skewed distribution and compared between two groups using the Mann–Whitney test. Categorical variables were presented as counts (percentages) and compared by Chi square test.

Univariate Cox's proportional hazards regression modelling was performed to identify determinants of ISR in diabetic patients. Baseline variables were selected if they had either a clinically plausible relation with the ISR or appeared to be imbalanced between ISR and non-ISR patients with a P-value less than 0.2. The potential variables were entered into multivariate Cox's proportional hazards regression modelling using the stepwise method (entry, 0.05; removal, 0.05) to determine their independent risk associated with ISR in diabetes. The hazard ratio (HR) and 95% confidence intervals (95% CIs) were calculated to estimate the adjusted risk of ISR in diabetic patients. The predictive value of the Cox's regression model was evaluated using the area under the receiver operating characteristics curve (AUC).

According to the ROC, the optimal cutoff point of the RLP-C was identified, and patients were divided into 2 groups. Propensity score matching analysis was performed in the two groups with a proportion of 1:1. Log-rank tests were used to compare Kaplan–Meier curves for overall follow-up to assess ISR between the two groups.

Statistical analyses were performed using SPSS software for Windows (version 24.0, SPSS Inc., Chicago, Illinois, USA). A two-sided probability value of < 0.05 was considered statistically significant in all analyses.

**Results**

**Baseline clinical and angiographic characteristics (Total population)**

The baseline clinical and angiographic characteristics of the total population are shown in Table 1. Significant differences were observed between the ISR and non-ISR group in terms of smoking, medical history, TG, HDL-C and RLP-C. After adjusting for other confounding factors in the multivariate Cox's proportional hazards regression, the RLP-C level was identified as one of the independent predictors associated with ISR in diabetic patients (Table 2). ROC curve analysis indicated that the AUC was 0.722 (95% CI = 0.693–0.751, P < 0.001), which showed a good predictive accuracy of RLP-C for the risk of ISR in diabetic patients after baseline PCI (Fig. 1). The baseline RLP-C level at 0.505 mmol/L (19.4 mg/dL) was identified as the optimal cutoff point to predict the risk of ISR with a sensitivity of 82.8% and a specificity of 52.0%.

Continuous variables were expressed as the mean (X̄) ± standard deviation (SD) in the case of
Table 1  Baseline clinical and angiographic characteristics of study population

| Characteristics          | Total (n = 2312)          | ISR (n = 372)           | Non-ISR (n = 1940)       | P values |
|--------------------------|---------------------------|-------------------------|--------------------------|----------|
| Age, years               | 56.21 ± 9.65              | 55.31 ± 9.67            | 56.38 ± 9.63             | 0.050    |
| Male, n (%)              | 1652 (71.5)               | 272 (73.1)              | 1380 (71.1)              | 0.438    |
| BMI, kg/m²               | 26.45 ± 3.04              | 26.52 ± 2.99            | 26.43 ± 3.05             | 0.618    |
| SBP, mmHg                | 131.45 ± 17.65            | 130.40 ± 15.92          | 131.65 ± 17.96           | 0.209    |
| DBP, mmHg                | 77.97 ± 10.23             | 77.63 ± 10.97           | 78.04 ± 10.09            | 0.509    |
| Smoking, n (%)           | 928 (40.1)                | 116 (31.2)              | 812 (41.9)               | < 0.001  |
| Drinking, n (%)          | 360 (15.6)                | 64 (17.2)               | 296 (15.3)               | 0.343    |
| Medical history, n (%)   |                           |                         |                          |          |
| Hypertension             | 1568 (67.8)               | 240 (64.5)              | 1328 (68.5)              | 0.136    |
| Hyperlipidaemia          | 1140 (49.3)               | 228 (61.3)              | 912 (47.0)               | < 0.001  |
| History of MI            | 204 (8.8)                 | 24 (6.5)                | 180 (9.3)                | 0.078    |
| History of stroke        | 180 (7.8)                 | 16 (4.3)                | 164 (8.5)                | 0.006    |
| Family history of CAD    | 208 (7.6)                 | 68 (18.3)               | 340 (17.5)               | 0.727    |
| Symptom for CAG          | 1354 (58.6)               | 234 (62.9)              | 1120 (57.7)              | 0.064    |
| Laboratory results       |                           |                         |                          |          |
| TG, mmol/L               | 2.22 ± 1.54               | 2.62 ± 2.16             | 2.14 ± 1.37              | < 0.001  |
| TC, mmol/L               | 4.43 ± 1.07               | 4.81 ± 1.12             | 4.35 ± 1.04              | < 0.001  |
| LDL-C, mmol/L            | 2.77 ± 0.90               | 2.81 ± 0.87             | 2.77 ± 0.94              | 0.348    |
| HDL-C, mmol/L            | 1.00 ± 0.26               | 1.04 ± 0.97             | 0.57 ± 0.44              | < 0.001  |
| RLP-C, mmol/L            | 0.65 ± 0.59               | 0.94 ± 1.00             | 0.59 ± 0.44              | < 0.001  |
| FBG, mmol/L              | 7.69 ± 2.50               | 7.64 ± 2.20             | 7.71 ± 2.55              | 0.591    |
| HbA1c, %                 | 7.35 ± 1.30               | 7.41 ± 1.21             | 7.34 ± 1.31              | 0.283    |
| hs-CRP, mg/L             | 4.72 ± 6.98               | 4.60 ± 5.93             | 4.75 ± 7.16              | 0.662    |
| Creatinine, μmol/L       | 77.35 ± 19.15             | 77.10 ± 18.23           | 77.40 ± 19.33            | 0.782    |
| GFR, ml/min              | 113.70 ± 316.21           | 184.46 ± 782.91         | 100.13 ± 27.43           | 0.038    |
| UA, μmol/L               | 335.49 ± 107.15           | 332.01 ± 129.22         | 336.16 ± 102.40          | 0.560    |
| LVEF, %                  | 61.83 ± 8.14              | 62.15 ± 7.37            | 61.76 ± 8.28             | 0.363    |
| Medical treatment, n (%) |                           |                         |                          |          |
| Statins                  | 2164 (93.6)               | 340 (91.4)              | 1824 (94.0)              | 0.058    |
| Aspirin                  | 2272 (98.3)               | 364 (97.8)              | 1908 (98.4)              | 0.497    |
| β-Blocker                | 1776 (76.8)               | 284 (76.3)              | 1492 (76.9)              | 0.814    |
| Clopidogrel              | 2292 (99.1)               | 364 (97.8)              | 1928 (99.4)              | 0.003    |
| Insulin                  | 555 (24.0)                | 76 (20.4)               | 480 (24.7)               | 0.075    |
| ACEI                     | 700 (30.3)                | 92 (24.7)               | 608 (31.3)               | 0.011    |
| ARB                      | 548 (23.7)                | 80 (21.5)               | 468 (24.2)               | 0.268    |
| Number of target vessels |                           |                         |                          | < 0.001  |
| One, n (%)               | 792 (34.3)                | 124 (33.3)              | 668 (34.4)               |          |
| Two, n (%)               | 948 (41.0)                | 112 (30.1)              | 836 (43.1)               |          |
| Three, n (%)             | 564 (24.4)                | 128 (34.4)              | 436 (22.5)               |          |
| Target vessels           |                           |                         |                          |          |
| LM, n (%)                | 56 (2.4)                  | 8 (2.2)                 | 48 (2.5)                 | 0.710    |
| LAD, n (%)               | 1316 (56.9)               | 244 (65.6)              | 1072 (55.3)              | < 0.001  |
| LCX, n (%)               | 724 (31.1)                | 140 (37.6)              | 584 (30.1)               | 0.004    |
| RCA, n (%)               | 884 (38.2)                | 160 (43.0)              | 724 (37.3)               | 0.039    |
| SYNTAX score             | 12.07 ± 6.98              | 14.08 ± 7.55            | 11.68 ± 6.79             | < 0.001  |
| Minimal stent diameter, mm | 2.95 ± 0.46             | 2.93 ± 0.41             | 2.95 ± 0.47              | 0.460    |
| Stent length, mm         | 22.02 ± 6.60              | 22.63 ± 5.82            | 21.90 ± 6.75             | 0.035    |

ISR in-stent restenosis, BMI body mass index, SBP systolic blood pressure, DBP diastolic blood pressure, MI myocardial infarction, CAD coronary artery disease, TG triglyceride, TC total cholesterol, LDL-C low-density lipoprotein cholesterol, HDL-C high-density lipoprotein cholesterol, RLP-C remnant-like particle cholesterol, FBG fasting blood glucose, hs-CRP high-sensitivity C-reactive protein, GFR glomerular filtration rate, UA uric acid, LVEF left ventricular ejection fraction, ACEI angiotensin converting enzyme inhibitor, ARB angiotensin receptor blocker, ISR in-stent restenosis, LM left main, LAD left anterior descending, LCX left circumflex artery, RCA right coronary artery, SYNTAX synergy between PCI with taxus and cardiac surgery
normal distribution and compared between two groups by the independent samples t-test. Data were expressed as medians (interquartile ranges) in the case of skewed distribution and compared using the Mann–Whitney U-test. Categorical variables are presented as counts (percentages) and compared by the Chi square test.

**Baseline clinical characteristics, angiographic characteristics and Kaplan–Meier curves (propensity score matching population)**

After propensity score matching analysis, a total of 762 pairs of matched patients were created. The C statistics for the propensity score model was 0.01. The baseline clinical characteristics before and after propensity score-matched analysis are shown in Fig. 2. No significant differences were observed between the low and high RLP groups after propensity score–matched analysis in terms of age, gender, BMI, medical history, duration of diabetes mellitus, HbA1c, fasting glycaemia, drug use, other biomarkers, angiographic characteristics and procedural characteristics.

Log-rank tests were used to compare Kaplan–Meier curves for overall follow-up to assess ISR between the two groups (Fig. 3).

**Discussion**

**Main findings**

The present observational cohort study from a high volume cardiovascular centre in China revealed potential atherosclerosis resulting from remnant lipoproteins in the occurrence and development of in-stent restenosis in diabetic patients. The major findings were as follows: (1) the presence of remnant-like particle cholesterol is an independent risk factor for in-stent restenosis in diabetic patients; and (2) diabetic patients with high RLP-C levels (≥ 0.505 mmol/L) have greater risk for in-stent restenosis compared to patients with low RLP-C levels.

**Abnormal lipid metabolism and atherosclerosis in DM**

It is well known that LDL-C is the major risk factor for atherosclerosis and CVD [11, 15]. However, several recent meta-analyses have indicated that a high residual risk of CVD remains even in patients whose LDL-C levels reach the treatment target after statin treatment [16, 17]. Additionally, current dyslipidaemia guidelines recommend non-HDL-C as the primary target of lipid-lowering therapy [10], including VLDL-C, which is the major component of RLP-C during fasting.

However, diabetic patients have dyslipidaemia characterized by high levels of RLP-C [7] but normal levels of LDL-C. Existing research has shown that increased remnant lipoprotein level is a risk factor for ischaemic heart disease. With an empty stomach, an increase of 1 mmol/L residual lipoprotein increases ischaemic heart disease risk by 2.8 times [18]. Recently, prospective studies tracking coronary events in diabetic patients have shown that remnant-like particle cholesterol are the most important independent risk factors of coronary artery disease and can predict coronary events [19, 20].

Both in vitro and animal experiments have confirmed that the formation of atherosclerosis induced by increases in remnant lipoproteins is similar to the formation of atherosclerosis caused by the accumulation of lipid in the arterial wall induced by increases in low-density lipoprotein. Many studies have confirmed that the mechanism of atherosclerosis induced by remnant lipoproteins mainly manifests in the following aspects: (1) induction of proliferation of smooth muscle cells but no involvement in oxidative stress [21]; (2) induction of apoptosis in endothelial cells [22]; (3) induction of mononuclear/macrophage migration in endothelial cells [23]; (4) induction of McP-1 expression in umbilical venous

**Table 2 Independent predictors of ISR in patients with DM after baseline PCI**

| Variables                                      | HR    | 95% CI       | P values |
|-----------------------------------------------|-------|--------------|----------|
| Model 1                                        | 1.609 | 1.478–1.735  | < 0.001  |
| Model 2                                        | 2.857 | 2.324–3.511  | < 0.001  |
| Model 3                                        | 2.763 | 2.216–3.446  | < 0.001  |

Model 1: age, male, smoking, hyperlipidaemia, history of stroke
Model 2: model 1 + TG, TC, HDL-C, GFR, clopidogrel, ACEI
Model 3: Model 2 + number of target vessels, LAD, LCX, RCA, SYNTAX score, stent length

**Fig. 1** The predictive values of RLP-C level for predicting the risk of ISR. ROC receiver operating characteristic, CI confidence interval
blood endothelial cells as well as early growth response factor-1 (egr-1) mRNA and protein expression in vascular smooth muscle cells, which induces the occurrence of inflammation [24]; and (5) induction of elevated levels of other atherogenic lipoproteins.

Contrast and enlightenment
China has over 92.4 million diabetic patients (9.7% of the adult population), which ranks at the top with DM patient numbers and higher diabetes-related burden than other countries [25]. A single-centre study from Fuwai Hospital of China [26] reported that second-generation drug-eluting stents have reliable efficacy and safety in diabetic and non-diabetic patients. In the subgroup analysis of diabetic patients, the risk factors associated with target lesion revascularization included current smoker, a history of coronary heart disease, and old myocardial infarction. In the baseline data of this study, only the history of hyperlipidaemia was associated with blood lipids, and the specific content of each component of the relevant blood lipids was not included, especially remnant-like particle cholesterol (possibly due to the large difference in blood lipid spectrum between diabetic and non-diabetic people). All patients included in the present study were treated with second generation drug-eluting stents. The baseline contained the contents of various blood lipid components. Propensity score matching found that remnant lipoproteins played an important role in in-stent restenosis, which supplemented findings from previous studies. In the present study, the TG level was higher in the in-stent restenosis group, which was attributed to the TG-rich remnant lipoproteins [8]. Therefore, TG increased with the increase of remnant lipoproteins. Patients were divided into two groups according to the remnant cholesterol level, and the baseline data of the two groups were matched with propensity scores. After matching, there was no statistically significant difference in TG between the two groups, excluding the influence of
TG on the results, which constituted a two-way verification process with the previous multi-factor analysis.

Previous studies [27] have proven that the association between disorders of TG metabolism and remnant-like particle cholesterol may account for the risk of CAD in diabetic patients. According to the relationship between the level of remnant-like particle cholesterol and the degree of in-stent restenosis confirmed by coronary angiography, the present study further confirmed that the remnant-like particle cholesterol rich in triglycerides play an important role in atherosclerosis in coronary heart disease patients with diabetes mellitus. Therefore, it is important to strengthen the management of remnant-like particle cholesterol in diabetic population in addition to the control of LDL-C levels required by the guidelines.

Statins play an irreplaceable role in secondary prevention of coronary heart disease. However, T2DM patients on statin therapy presenting increased levels of cholesterol remnants and triglycerides are prone to slight decreases in left ventricular systolic function [28], which severely affects the prognosis of diabetic patients with coronary heart disease. Therefore, in the secondary prevention of coronary heart disease in diabetic patients, it is important to use statins to reduce LDL-C while monitoring remnant-like particle cholesterol. Previous studies have confirmed that empagliflozin [29] and pemafibrate [30] lower remnant-like particle cholesterol, indicating that they have a good curative effect and are recommended to delay the progression of coronary atherosclerosis.

Limitations
Some limitations and strengths of the present study need to be acknowledged. First, the study was only a single-centre study, which may weaken the statistical power of the conclusions. Second, propensity score matching provides yields weaker evidence than randomized controlled trials.

Conclusions
In conclusion, the present study further emphasized the importance of atherogenic lipids and remnant particles in cardiovascular pathology (such as in-stent restenosis), especially in diabetic patients. Physicians should take measures to lower the level of remnant-like particle cholesterol to <0.505 mmol/L to better prevent in-stent restenosis in diabetic patients.

Abbreviations
ACEI: angiotensin converting enzyme inhibitor; ARB: angiotensin receptor blocker; BMI: body mass index; BP–DESs: biodegradable polymer drug-eluting stent; CAD: coronary artery disease; CRF: case report form; DBP: diastolic blood pressure; DM: diabetes mellitus; ECG: electrocardiograph; FBG: fasting blood glucose; G2–DESs: second–generation drug-eluting stent; GFR: glomerular filtration rate; HDL-C: high density lipoprotein cholesterol; HPLC: high performance liquid chromatograph; hs-CRP: high-sensitivity C-reactive protein; ISR: in-stent restenosis; LAD: left anterior descending; LCX: left circumflex artery; LDL-C: low density lipoprotein cholesterol; LM: left main; LVEF: left ventricular ejection fraction; MI: myocardial infarction; MVD: multivessel disease; Non-HDL-C: non-high density lipoprotein cholesterol; PCI: percutaneous coronary intervention; RCA: right coronary artery; ROC: receiver operating characteristics; RLP-C: remnant-like particle cholesterol; SBP: systolic blood pressure; SYN-TAX: synergy between PCI with taxus and cardiac surgery; TC: total cholesterol; TG: triglyceride; UA: uric acid; VLDL-C: very low density lipoprotein cholesterol.

Authors’ contributions
YJZ (first author) made substantial contributions to study conception, study design, data acquisition, data analysis, data interpretation and manuscript draft. YJZ (corresponding author) made substantial contributions to study concept, study design and intellectual content. They contributed equally to this work. KZ, YPL, JLW, WJC, CS, CPH and HH made substantial contributions to data acquisition. All authors read and approved the final manuscript.

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Not applicable.

**Competing interests**

The authors declare that they have no competing interests.

**Availability of data and materials**

The datasets generated and analysed for this study are available from the corresponding author upon reasonable request.

**Consent for publication**

Not applicable.

**Ethics approval and consent to participate**

The present study was approved by the Clinical Research Ethics Committee of Beijing Anzhen Hospital, Capital Medical University, and all patients provided written informed consent for participation in the present study.

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