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ApoB/ApoA-I is associated with major cardiovascular events and readmission risk of patients after percutaneous coronary intervention in one year

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Percutaneous coronary intervention (PCI) is a practical and effective method for treating coronary heart disease (CHD). This study aims to explore the influencing factors of major cardiovascular events (MACEs) and hospital readmission risk within one year following PCI treatment. Additionally, it seeks to assess the clinical value of Apolipoprotein B/Apolipoprotein A-I (ApoB/ApoA-I) in predicting the risk of one-year MACEs and readmission post-PCI. A retrospective study included 1938 patients who underwent PCI treatment from January 2010 to December 2018 at Shandong Provincial Hospital affiliated with Shandong First Medical University. Patient demographics, medications, and biochemical indicators were recorded upon admission, with one-year follow-up post-operation. Univariate and multivariate Cox proportional hazards regression models were utilized to establish the relationship between ApoB/ApoA-I levels and MACEs/readmission. Predictive nomograms were constructed to forecast MACEs and readmission, with the accuracy of the nomograms assessed using the concordance index. Subgroup analyses were conducted to explore the occurrence of MACEs and readmission. We observed a correlation between ApoB/ApoA-I and other lipid indices, including total cholesterol (TC), triglycerides (TG), low-density lipoprotein cholesterol (LDL-C), and high-density lipoprotein cholesterol (HDL-C) ($P < 0.001$). Univariate and multivariate Cox regression analyses demonstrated that ApoB/ApoA-I is an independent risk factor for MACEs in post-PCI patients ($P = 0.038$). Within one year, the incidence of MACEs significantly increased in the high-level ApoB/ApoA-I group (ApoB/ApoA-I ratio ≥ 0.824) ($P = 0.038$), while the increase in readmission incidence within one year was not statistically significant. Furthermore, a nomogram predicting one-year MACEs was established (Concordance Index: 0.668). Subgroup analysis revealed that ApoB/ApoA-I was associated with the occurrence of both MACEs and readmission in male patients, those using CCB/ARB/ACEI, those without multivessel diseases, or those with LDL-C < 2.6 mmol/L. The ApoB/ApoA-I ratio serves as an independent risk factor for one-year MACEs in post-PCI patients and correlates closely with other blood lipid indicators. ApoB/ApoA-I demonstrates significant predictive value for the occurrence of MACEs within one year.

Trial registration **Chinese clinical trial registry: No.ChiCTR22000597-23.**

Keywords Percutaneous coronary intervention, ApoB/ApoA-I, Major cardiovascular events, Readmission

Abbreviations

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PCI	Percutaneous coronary intervention
CHD	Coronary heart disease
NSTEMI	Non-ST-segment elevation myocardial infarction
STEMI	ST-segment elevation myocardial infarction
MACEs	Major adverse cardiovascular events
ApoB/ApoA-I	Apolipoprotein B/Apolipoprotein A-I
TG	Triglycerides
TC	Cholesterol
LDL-C	Low-density lipoprotein cholesterol
HDL-C	High-density lipoprotein cholesterol
ASCVD	Arteriosclerotic cardiovascular disease
ACS	Acute coronary syndrome
AMI	Acute myocardial infarction
HR	Hazard ratios
CI	Confidence intervals
FPG	Fasting plasma glucose
sd-LDL	Small dense LDL particles

Atherosclerotic cardiovascular disease (ASCVD) stands as the foremost cause of mortality worldwide[1,2]. Acute coronary syndrome (ACS), comprising unstable angina, non-ST-segment elevation myocardial infarction (NSTEMI), and ST-segment elevation myocardial infarction (STEMI), manifests as a cluster of clinical syndromes stemming from acute myocardial ischemia. It represents the most perilous and lethal form of ASCVD[3]. Percutaneous coronary intervention (PCI) emerges as the predominant method of angioplasty for patients with coronary heart disease (CHD)[4]. Its objective lies in ameliorating symptoms of myocardial ischemia and hypoxia by widening and clearing narrowed or obstructed coronary arteries, thereby enhancing patient survival rates. However, while PCI effectively alleviates stenotic lesions, questions persist regarding prognosis[5]. Continuation of CHD risk factors predisposes patients to adverse cardiac events such as angina pectoris, chest pain, and post-surgical relapse, necessitating readmission for treatment. Hence, understanding the incidence rate and determinants of major cardiovascular events (MACEs) assumes paramount importance for CHD patients post-PCI[6,7]. Additionally, the issue of unplanned readmissions subsequent to PCI poses a substantial burden on healthcare systems and has garnered increased attention[8,9].

Dyslipidemia emerges as one of the primary risk factors for ASCVD[2,10]. Lipid accumulation assumes a pivotal role in atherosclerosis among ACS patients[11]. Beyond conventional blood lipid markers like low-density lipoprotein cholesterol (LDL-C), triglycerides (TG), and total cholesterol (TC), considerable evidence suggests that apolipoproteins (apo) may serve as reliable predictors for primary prevention. Current guidelines advocate for Apolipoprotein B (ApoB) as an alternative to LDL-C for risk assessment[12]. ApoB facilitates the transportation of all potentially atherosclerotic particles, encompassing very low-density lipoprotein, medium-density lipoprotein, and low-density lipoprotein particles, thus reflecting the total number of atherosclerotic particles. Conversely, Apolipoprotein A-I (ApoA-I) acts as the primary anti-atherosclerotic protein in high-density lipoprotein (HDL) particles, facilitating the transfer of excess cholesterol from peripheral tissues to the liver[13]. Consequently, the ApoB/ApoA-I ratio emerges as an indicator of the balance between atherogenic and anti-atherosclerotic cholesterol in plasma[14]. Studies suggest a correlation between higher ApoB/ApoA-I levels and more severe atherosclerotic lesions, speculating on its relation to ACS progression. Even when other lipids remain within the normal range, the ApoB/ApoA-I ratio significantly correlates with the risk of fatal myocardial infarction (MI), independent of traditional CHD risk factors[15]. Moreover, research indicates that the ApoB/ApoA-I ratio outperforms the Framingham Risk Score and TC/HDL-C as a predictor of CHD risk[16]. Serum levels of ApoB and the ApoB/ApoA-I ratio can serve as cardiovascular disease risk factors and novel targets for lipid-lowering treatment strategies, as confirmed by related studies[17].

While numerous studies have explored the intimate connection between the ApoB/ApoA-I ratio and cardiovascular risk, scant attention has been directed towards its association with MACEs. Similarly, the relationship between ApoB/ApoA-I and readmission post-PCI remains unclear. Hence, we conducted a retrospective study to evaluate the correlation of the ApoB/ApoA-I ratio with both MACEs and readmission within one year following PCI treatment.

Methods

Study population

This retrospective study was conducted at Shandong Provincial Hospital affiliated with Shandong First Medical University, Jinan, China. Between December 2009 and December 2018, patients hospitalized for PCI whose records were available in the hospital database were included. Exclusion criteria comprised: (1) thyroid disease, infectious disease, and immune system disorders; (2) moderate to severe rheumatic valvular disease; (3) recent use of anti-inflammatory and antioxidant drugs; (4) liver and kidney dysfunction, or other serious systemic conditions; (5) hematological disorders, tumors, or other acute or chronic illnesses; (6) mental health disorders. The study adhered to the Declaration of Helsinki and received approval from the Ethics Committee of Shandong Provincial Hospital affiliated with Shandong First Medical University. The trial was registered at the Chinese Clinical Trial Registry (ChiCTR22000597-23). Written informed consent was waived due to the retrospective nature of the study by the Ethics Committee of Shandong Provincial Hospital affiliated with Shandong First Medical University. Patients were categorized into two groups based on ApoB/ApoA-I ratio levels: high [ApoB/ApoA-I $\geq$ 0.824, 992 cases] and low [ApoB/ApoA-I $<$ 0.824, 946 cases]. The study schematic diagram is depicted in Fig. 1.

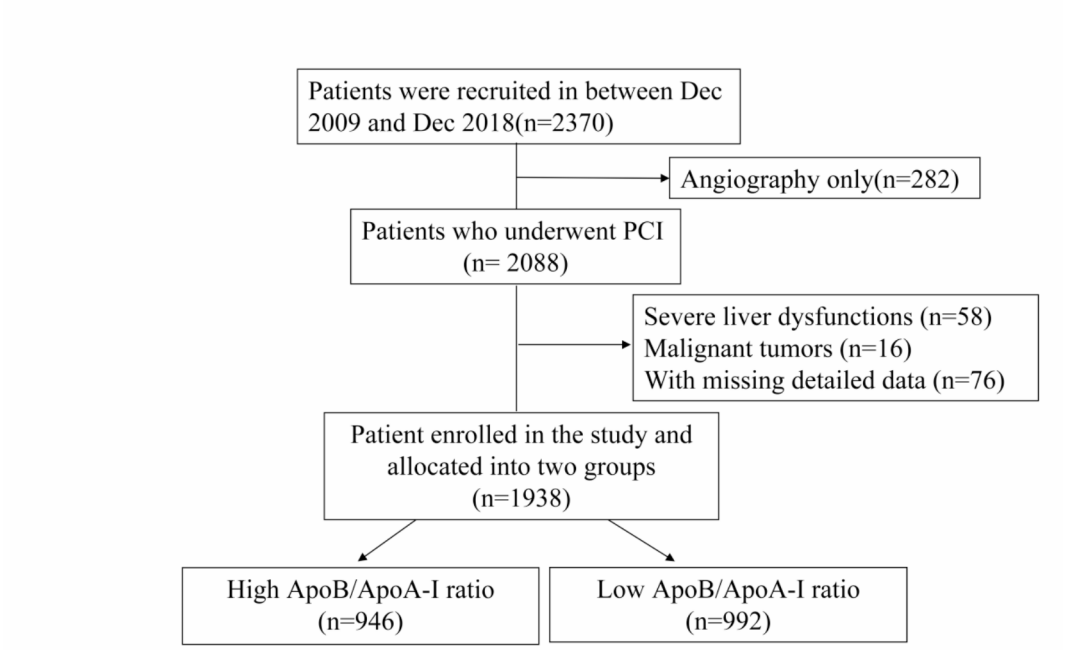

Fig. 1. Flowchart of patient enrollment.

Data collection

Peripheral venous blood samples were collected from patients upon admission to the hospital ward. Lipid profiles, including TC, TG, LDL-C, HDL-C, ApoA-I, ApoB, and other biochemical indicators, were measured at the clinical laboratory diagnostic center of Shandong Provincial Hospital affiliated with Shandong First Medical University. Demographic data such as age, gender, past medical history, smoking and drinking status, clinical characteristics, blood samples, biochemical data, medication, coronary angiography, and surgical information were obtained from electronic medical records. Medical history, including the use of statins, CCB/ARB/ACEI, Trimetazidine, Nitrate, β-blockers, diuretics, and hypoglycemics, was also extracted from medical records and follow-up period. Follow-up was conducted from the discharge date until one year post-procedure. Follow-up data were collected through medical record review, outpatient visits, and/or telephone interviews.

Outcomes

The primary outcome measure of this study was MACEs, encompassing cardiovascular death, nonfatal myocardial infarction, nonfatal stroke, coronary revascularization, and hospitalization for unstable angina. Secondary outcomes included the incidence of hospital readmission within one year, defined as all-cause readmission from discharge to one year post-surgery.

Statistical analysis

Statistical analyses were conducted using R version 3.6.3, Python version 3.7, and GraphPad Prism 8.0. Continuous variables were presented as mean ± standard deviation, while categorical variables were expressed as counts and percentages. Baseline data were compared using the t-test for continuous variables and the χ^2 test for categorical variables. Pearson analyses were employed to assess the correlation between ApoB/ApoA-I and other lipid indices. Cumulative hazard curves were generated to depict the cumulative incidence of MACEs and readmission over time, with comparisons made using the log-rank test. Cox-regression analyses and subgroup analyses were performed to determine whether ApoB/ApoA-I served as an independent risk factor for MACEs or readmission, presenting hazard ratios (HR) and 95% confidence intervals (CI). Predictive nomograms were constructed to forecast prognosis for MACEs and readmission, with the predictive accuracy assessed using the concordance index (C-index). Statistical significance was set at a two-tailed P value less than 0.05.

Results

Baseline clinical characteristics

Out of 2088 CHD patients who underwent PCI treatment following coronary angiography, 1938 patients were included in the study. Among them, 264 (13.6%) experienced MACE events within one year post-PCI. The median ApoB/ApoA-I ratio was 0.824 (interquartile range: 0.664–1.009). A total of 992 (51.2%) patients had

low ApoB/ApoA-I ratio, while 946 (48.8%) had high ratio. Baseline characteristics were stratified based on the median ApoB/ApoA-I ratio. Patients with a high ApoB/ApoA-I ratio exhibited more cardiovascular risk factors, including diabetes, dyslipidemia, myocardial infarction, multivessel disease, and occlusion, and were less likely to be receiving statins. Additionally, patients in the high ratio group tended to have higher levels of TC, TG, LDL-C, LDL-C/HDL-C ratio, ApoB, uric acid, fasting plasma glucose (FPG), and HbA1c, as well as lower HDL-C and ApoA-I. Baseline demographic and procedural characteristics are presented in Table 1.

Correlations between ApoB/ApoA-I and other lipid indexes

ApoB/ApoA-I exhibited moderate to strong correlations with other lipid indexes, including LDL-C, TC, TG, and HDL-C (all $P < 0.001$). The strongest correlation was observed with LDL-C (Pearson $R = 0.645$). Detailed results of the Pearson correlations are illustrated in Fig. 2.

	Total	Low APOB/ApoA-I	High APOB/ApoA-I	*P* value
N(%)	1938	992(51.2)	946(48.8)	-
Age, y	60.39 ± 9.88	61.58 ± 9.60	59.67 ± 10.04	0.002
Male, n(%)	1361(70.2)	697(70.3)	664(70.2)	0.972
Heart rate, bpm	69.47 ± 11.11	68.50 ± 10.76	70.48 ± 11.38	< 0.001
SBP, mmHg	134.43 ± 18.97	134.21 ± 18.36	134.66 ± 19.59	0.605
DBP, mmHg	79.04 ± 12.15	78.65 ± 11.83	79.43 ± 12.47	0.158
Hospital day, d	9.74 ± 4.84	9.48 ± 4.44	10.01 ± 5.20	0.016
Medical history, n (%)				
Hypertension	1173(60.5)	602(60.7)	571(60.4)	0.883
Diabetes	615(31.7)	284(28.6)	331(35.0)	0.003
Dislipidemia	62(3.2)	17(1.7)	45(4.8)	< 0.001
Previous PCI	144(7.4)	81(8.2)	63(6.7)	0.206
Myocardial infarction	744(38.4)	335(33.8)	409(43.2)	< 0.001
Procedural characteristics, n (%)				
Multivessel disease	1773(91.5)	891(89.8)	882(93.2)	0.007
Vascular occlusion	540(27.9)	245(24.7)	295(31.2)	0.001
Risk factor, n (%)				
Smoking	954(49.2)	475(47.9)	479(50.6)	0.226
Drinking	913(47.1)	460(46.4)	453(47.9)	0.504
Laboratory tests				
TC, mmol/L	4.51 ± 1.14	3.98 ± 0.86	5.06 ± 1.14	< 0.001
TG, mmol/L	1.73 ± 1.08	1.53 ± 0.97	1.95 ± 1.13	< 0.001
LDL-C, mmol/L	2.74 ± 0.89	2.29 ± 0.65	3.21 ± 0.87	< 0.001
HDL-C, mmol/L	1.11 ± 0.26	1.16 ± 0.26	1.06 ± 0.24	< 0.001
LDL-C/HDL-C ratio	2.58 ± 0.99	2.04 ± 0.65	3.15 ± 0.97	< 0.001
Lipoprotein a, g/L	0.17(0.08–0.35)	0.15(0.07–0.29)	0.20(0.09–0.38)	< 0.001
ApoA-I, g/L	1.11 ± 0.20	1.12 ± 0.21	1.11 ± 0.20	< 0.001
ApoB, g/L	0.94 ± 0.31	0.75 ± 0.18	1.14 ± 0.30	< 0.001
ApoB/ApoA-I ratio	0.87 ± 0.31	0.65 ± 0.13	1.10 ± 0.28	< 0.001
Creatinine, μmol/L	71.70 ± 16.48	71.88 ± 15.83	71.51 ± 17.13	0.625
Uric acid, μmol/L	333.94 ± 84.54	325.19 ± 82.66	343.12 ± 85.51	< 0.001
Hcy, μmol/L	15.43 ± 7.48	15.70 ± 7.63	15.16 ± 7.30	0.110
FPG, mmol/L	6.38 ± 1.92	6.15 ± 1.69	6.62 ± 2.11	< 0.001
Discharge medication, n (%)				
Statins	1811(93.4)	938(94.6)	873(92.3)	0.043
CCB/ARB/ACEI	1181(60.9)	599(60.4)	582(61.5)	0.607
Trimetazidine	1078(55.6)	578(58.3)	500(52.9)	0.017
Nitrate	1180(60.9)	606(61.1)	574(60.7)	0.853
β-blockers	1589(82.0)	824(83.1)	765(80.9)	0.208
Diuretic	310(16.0)	146(14.7)	164(17.3)	0.116
Hypoglycemics	494(25.5)	223(22.5)	271(28.6)	0.002

Table 1. Baseline characteristics of the patients.

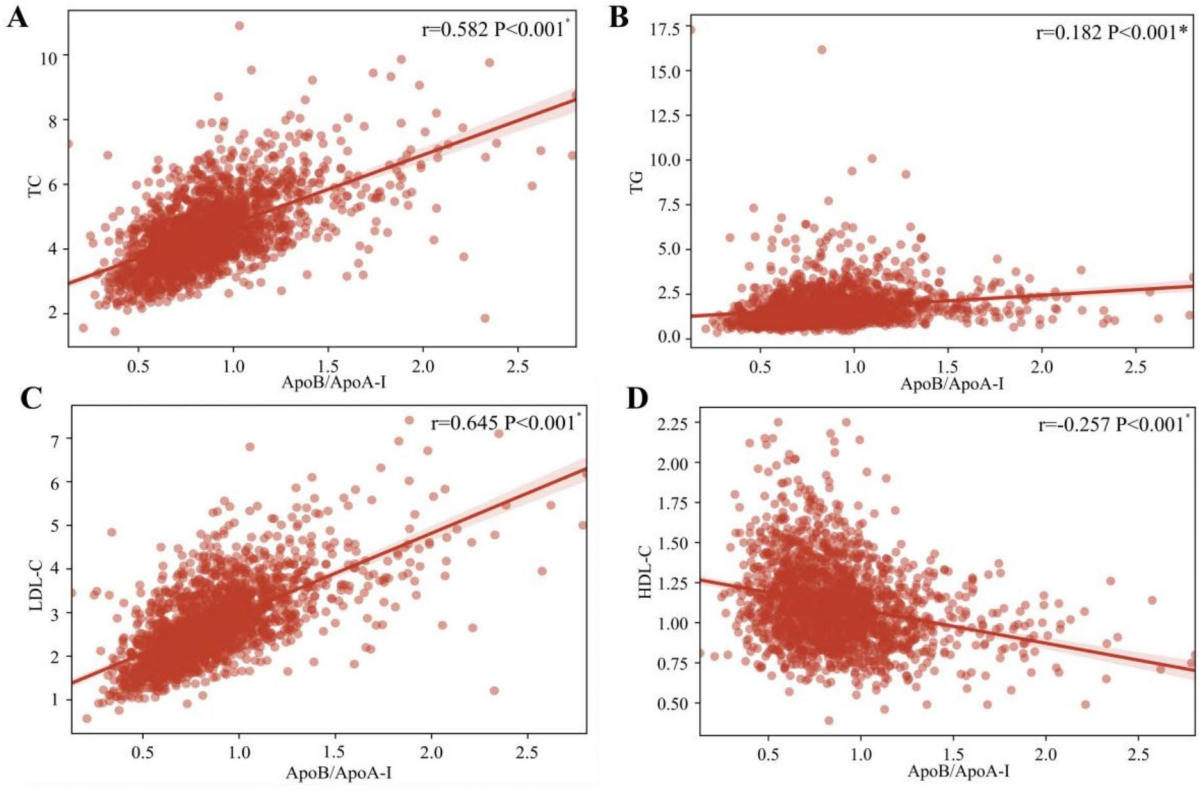

Fig. 2. Pearson Correlations Between ApoB/ApoA-I Ratio and Other Lipid Indexes. All $P < 0.001$. (**A**) Pearson Correlation Between ApoB/ApoA-I Ratio and TC (**B**) Pearson Correlation Between ApoB/ApoA-I Ratio and TG (**C**) Pearson Correlation Between ApoB/ApoA-I Ratio and LDL-C (**D**) Pearson Correlation Between ApoB/ApoA-I Ratio and HDL-C.

Results of survival analysis

Angina pectoris and revascularization accounted for a higher proportion in MACEs, which were 252 (13.0%) and 150 (7.7%) cases respectively. Cumulative hazard curves revealed that patients in the high ApoB/ApoA-I ratio group had a significantly higher occurrence of one-year MACEs (log-rank $P = 0.037$; Fig. 3A) and angina pectoris (log-rank $P = 0.024$; Fig. 3C) compared to those in the low ratio group. Although patients in the high ratio group appeared to have a higher occurrence of one-year readmission (log-rank $P = 0.065$; Fig. 3B) and revascularization (log-rank $P = 0.058$; Fig. 3D) compared to those in the low ratio group, the difference was not statistically significant.

Association between the ApoB/ApoA-I ratio and MACE and readmission occurrence during the follow-up period

Multivariate analyses were employed to investigate the impact of ApoB/ApoA-I ratio levels on MACEs and readmission, adjusting for other blood lipid indexes. Adjusted HRs and 95% CIs of MACEs and readmission according to ApoB/ApoA-I ratio levels were presented in Table 2. In the unadjusted model, there was an association between ApoB/ApoA-I ratio and MACEs (HR, 1.60; 95% CI, 1.13–2.27; $P = 0.007$). This association strengthened after adjusting for age, sex, and other lipid covariates, including TC, TG, LDL-C, and HDL-C (HR, 3.13; 95% CI, 1.92–5.12; $P < 0.001$). Similarly, there was an association between ApoB/ApoA-I ratio and readmission (HR, 1.43; 95% CI, 1.03–1.98; $P = 0.034$) in the unadjusted model. Additional adjustment for other covariates, including age, sex, and other lipid indexes, did not alter this association (HR, 2.40; 95% CI, 1.49–3.87; $P < 0.001$).

Among the 1938 patients, there were 1435 patients underwent elective PCI for stable angina and unstable angina, 278 patients underwent PCI due to STEMI, and 225 patients underwent PCI due to NSTEMI. We conducted a hierarchical regression analysis to observe the relationship between the occurrence of APOB/ApoA-I and MACEs in different types of diseases in Table 3. In the unadjusted model there was an association between ApoB/ApoA-I ratio and MACEs in patients who underwent elective PCI (HR, 1.706; 95% CI, 1.130–

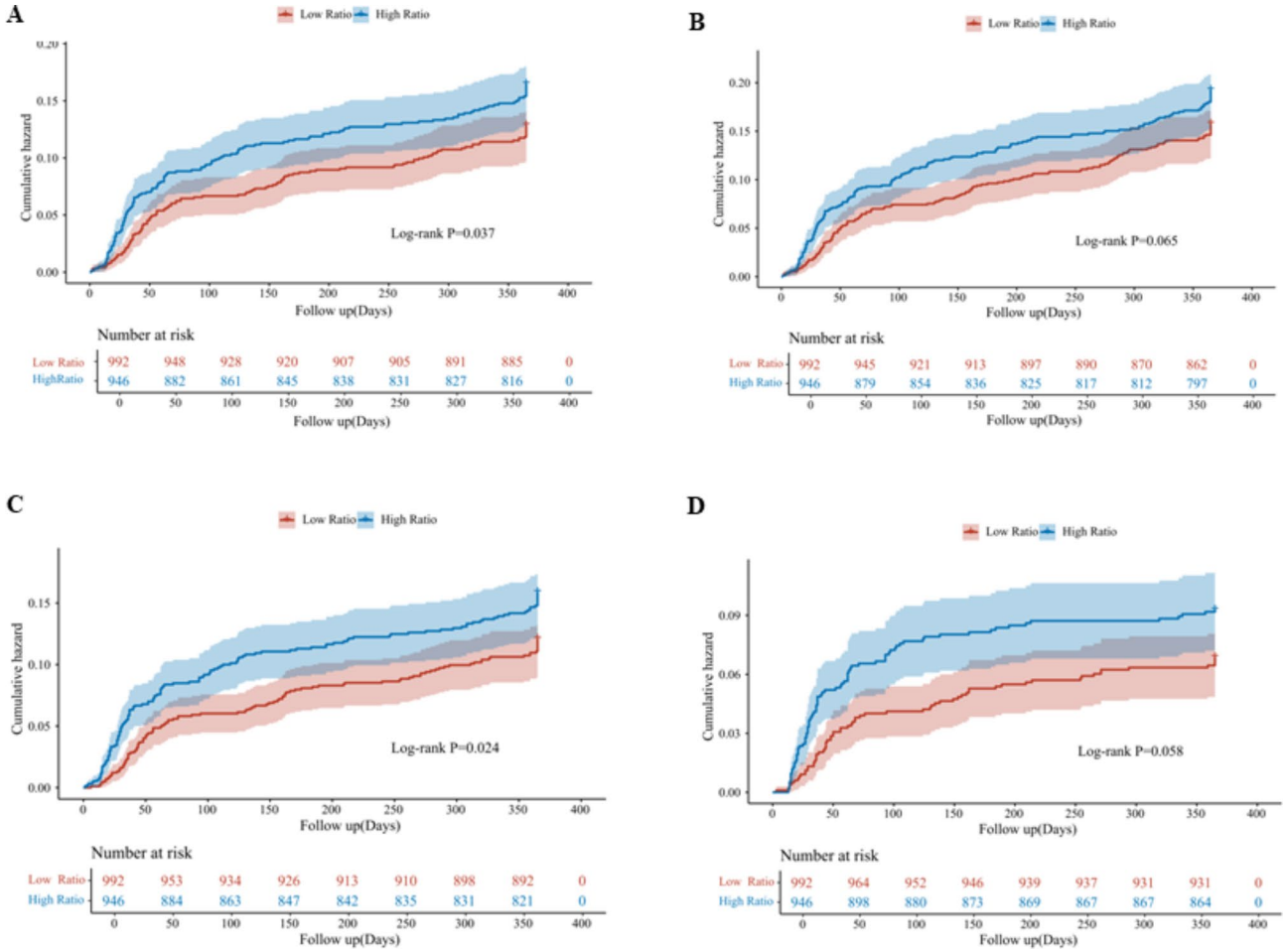

Fig. 3. Cumulative Hazard Curves of Events According to ApoB/ApoA-I Ratio. (**A**) Cumulative Hazard of MACEs by Group of ApoB/ApoA-I Ratio at One Year Follow-up (**B**) Cumulative Hazard of Readmission by Group of ApoB/ApoA-I Ratio at One Year Follow-up (**C**) Cumulative Hazard of Angina pectoris by Group of ApoB/ApoA-I Ratio at One Year Follow-up (**D**) Cumulative Hazard of Revascularization by Group of ApoB/ApoA-I Ratio at One Year Follow-up.

End point	Category	Crude model		Model 1		Model 2	
		HR(95%CI)	*P* value	HR(95%CI)	*P* value	HR(95%CI)	*P* value
MACEs	ApoB/ApoA-I	1.60 (1.13–2.27)	0.007	1.65 (1.17–2.34)	0.005	3.13 (1.92–5.12)	< 0.001
	Low ratio	1.0 (Reference)		1.0 (Reference)		1.0 (Reference)	
	High ratio	1.29 (1.01–1.65)	0.038	1.31 (1.03–1.67)	0.030	1.54 (1.14–2.09)	0.005
Readmission	ApoB/ApoA-I	1.43 (1.03–1.98)	0.034	1.49 (1.07–2.08)	0.018	2.40 (1.49–3.87)	< 0.001
	Low ratio	1.0 (Reference)		1.0 (Reference)		1.0 (Reference)	
	High ratio	1.23 (0.99–1.54)	0.065	1.26 (1.01–1.57)	0.045	1.43 (1.08–1.88)	0.012

Table 2. Association between the ApoB/ApoA-I ratio and MACE and readmission occurrence during the follow-up period. Abbreviations: HR hazard ratio; CI confidence interval; Crude model: unadjusted. Model 1: adjusted for age, sex. Model 2: adjusted for age, sex, TC, TG, LDL-C and HDL-C.

Subgroup	N	HR	95%CI	*P* value	HR (adjusted)	95%CI (adjusted)	*P* Value (adjusted)
Total	1938	1.603	(1.134, 2.266)	0.007	3.133	(1.917, 5.121)	< 0.001
0	1435	1.706	(1.130, 2.575)	0.011	3.401	(1.952, 5.928)	< 0.001
1	278	0.750	(0.289, 1.943)	0.553	1.075	(0.224, 5.167)	0.928
2	225	2.845	(1.146, 7.064)	0.024	5.634	(1.414, 22.449)	0.014

Table 3. Hierarchical regression analysis. 0: patients who underwent elective PCI, 1: patients who underwent PCI due to STEMI, 2: patients who underwent PCI due to NSTEMI. Crude model: unadjusted model. Model 1: adjusted for age, sex, TC, TG, LDL-C and HDL-C.

Subgroups	N	HR	95%CI	*P* value
Total	1938	1.603	(1.134,2.266)	0.007
Male				
0	577	1.015	(0.523,1.968)	0.966
1	1361	1.983	(1.318,2.985)	0.001
Vascular occlusion				
0	1398	1.711	(1.098,2.667)	0.018
1	540	1.335	(0.751,2.372)	0.325
multivessel disease				
0	165	23.979	(4.473,128.560)	< 0.001
1	1773	1.402	(0.974,2.017)	0.069
CCB/ARB/ACEI				
0	757	1.722	(0.894,3.315)	0.104
1	1181	1.593	(1.054,2.408)	0.027
Statins				
0	127	1.619	(0.753,3.480)	0.217
1	1811	1.513	(1.029,2.226)	0.035
FPG				
< 6.1	1172	1.728	(1.064,2.808)	0.027
6.1–7.0	291	1.862	(0.864,4.013)	0.112
≥ 7.0	475	1.142	(0.588,2.216)	0.695
LDL-C				
< 1.8	242	2.416	(0.583,10.010)	0.224
1.8–2.6	682	1.868	(0.719,4.856)	0.200
≥ 2.6	1014	2.043	(1.306,3.197)	0.002

Table 4. Subgroup analysis of occurrence of MACEs.

2.575; $P = 0.011$) and patients who underwent PCI due to NSTEMI (HR, 2.845; 95% CI, 1.146–7.064; $P = 0.024$). In the adjusted model after adjusting for age, sex and other lipid covariates including TC, TG, LDL-C, HDL-C there was also an association between ApoB/ApoA-I ratio and MACEs in patients who underwent elective PCI (HR, 3.401; 95% CI, 1.952–5.928; $P < 0.001$) and patients patients who underwent PCI due to NSTEMI (HR, 5.634; 95% CI, 1.414–22.449; $P = 0.014$).

Subgroup analysis results indicated that ApoB/ApoA-I was associated with the occurrence of MACEs in male patients, patients without vascular occlusion, without multivessel disease, using statins or CCB/ARB/ACEI, FPG < 6.1 mmol/L, and LDL-C ≥ 2.6 mmol/L ($P < 0.05$). ApoB/ApoA-I was associated with readmission in male patients, patients without multivessel disease, using CCB/ARB/ACEI, or LDL-C ≥ 2.6 mmol/L ($P < 0.05$). Detailed results of the subgroup analysis are presented in Tables 4 and 5.

Results of unadjusted and adjusted cox regression to predict one year MACEs and readmission at follow-up

Cox regression analyses were utilized to examine the impact of baseline characteristics associated with one-year MACEs and readmission. Variables with a P value < 0.1 on univariate analysis were included in the multivariable model. The results revealed that vascular occlusion (HR: 1.954, 95% CI: 1.527-2.500, $P < 0.001$), ApoB/ApoA-I ratio (HR: 1.464, 95% CI: 1.021-2.100, $P = 0.038$), use of statins (HR: 0.406, 95% CI: 0.288–0.571, $P < 0.001$), and use of CCB/ARB/ACEI (HR: 2.047, 95% CI: 1.539–2.723, $P < 0.001$) were independent predictors of one-year MACEs. Vascular occlusion (HR: 1.824, 95% CI: 1.451–2.293, $P < 0.001$), albumin (HR: 0.963, 95% CI: 0.938–0.990, $P = 0.007$), use of statins (HR: 0.461, 95% CI: 0.330–0.644, $P < 0.001$), and use of CCB/ARB/ACEI (HR:

Subgroups	N	HR	95%CI	*P* value
Total	1938	1.427	(1.027,1.984)	0.034
Male				
0	577	0.815	(0.429,1.549)	0.532
1	1361	1.836	(1.248,2.700)	0.002
Vascular occlusion				
0	1398	1.358	(0.883,2.089)	0.163
1	540	1.426	(0.835,2.435)	0.194
Multivessel disease				
0	165	15.011	(3.484,64.675)	< 0.001
1	1773	1.250	(0.884,1.769)	0.207
CCB/ARB/ACEI				
0	757	1.250	(0.667,2.340)	0.486
1	1181	1.533	(1.036,2.270)	0.033
Statins				
0	127	1.486	(0.681,3.246)	0.320
1	1811	1.355	(0.943,1.946)	0.100
FPG				
< 6.1	1172	1.505	(0.951,2.381)	0.081
6.1–7.0	291	1.585	(0.745,3.371)	0.231
≥ 7.0	475	1.122	(0.601,2.096)	0.718
LDL-C				
< 1.8	242	1.846	(0.456,7.466)	0.390
1.8–2.6	682	1.400	(0.558,3.513)	0.473
≥ 2.6	1014	1.878	(1.232,2.861)	0.003

Table 5. Subgroup analysis of occurrence of readmission.

	Univariate analysis		Multivariate analysis	
	Hazard ratio(95%CI)	*P* value	Hazard ratio(95%CI)	*P* value
Multivessel disease	1.729 (1.009–2.961)	0.046	1.378 (0.801–2.373)	0.247
Vascular occlusion	2.127 (1.668–2.713)	< 0.001	1.954 (1.527–2.500)	< 0.001
Hospital day	1.018 (0.997–1.039)	0.093	1.001 (0.978–1.025)	0.914
ApoB/ApoA-I	1.603 (1.134–2.266)	0.007	1.464 (1.021–2.100)	0.038
Albumin	0.961 (0.933–0.990)	0.009	0.972 (0.944–1.000)	0.052
FPG	1.065 (1.008–1.124)	0.024	1.027 (0.959–1.100)	0.441
Statins	0.363 (0.259–0.511)	< 0.001	0.406 (0.288–0.571)	< 0.001
CCB/ARB/ACEI	2.134 (1.608–2.832)	< 0.001	2.047 (1.539–2.723)	< 0.001
Hypoglycemics	1.326 (1.020–1.722)	0.035	1.110 (0.810–1.520)	0.516

Table 6. Univariate and Multivariate analysis results of Cox proportional hazards model to predict one year MACEs.

1.714, 95% CI: 1.333–2.205, $P < 0.001$) were independent predictors of one-year readmission. Detailed results of the Cox regression are presented in Tables 6 and 7.

The nomograms for the prediction of MACEs and readmission

To predict patient outcomes after successful PCI, prognostic nomograms were established using a multivariate Cox regression model based on significantly independent factors for one-year MACEs and readmission (Fig. 4A, B). Each prognostic factor corresponds to multiple risk points, obtained by drawing a vertical line from the corresponding value of the prognostic factor up to the axis labeled "dots." By summing the total risk points, one can determine the one-year MACEs and readmission probabilities for a specific patient. The C-index of the model was 0.668 (95% CI: 0.635–0.701) for MACEs and 0.648 (95% CI: 0.617–0.679) for readmission.

Discussion

Lipid metabolism is highly intricate, with LDL-C unable to fully depict the body's lipid metabolism state due to its unique characteristics. Recent research focus has honed in on apolipoproteins, notably ApoB and ApoA-I[18].

	Univariate analysis		Multivariate analysis	
	Hazard ratio(95%CI)	*P* value	Hazard ratio(95%CI)	*P* value
Age	1.013 (1.002–1.025)	0.023	1.011 (0.999–1.023)	0.080
Hospital day	1.024 (1.006–1.041)	0.007	1.007 (0.987–1.028)	0.497
Multivessel disease	1.483 (0.932–2.359)	0.096	1.181 (0.737–1.891)	0.489
Vascular occlusion	1.967 (1.570–2.465)	< 0.001	1.824 (1.451–2.293)	< 0.001
ApoB/ApoA-I	1.427 (1.027–1.984)	0.034	1.360 (0.963–1.921)	0.081
Albumin	0.949 (0.923–0.976)	< 0.001	0.963 (0.938–0.990)	0.007
FPG	1.050 (0.996–1.106)	0.069	1.016 (0.952–1.084)	0.639
Statins	0.424 (0.304–0.591)	< 0.001	0.461 (0.330–0.644)	< 0.001
CCB/ARB/ACEI	1.780 (1.387–2.284)	< 0.001	1.714 (1.333–2.205)	< 0.001
Hypoglycemics	1.257 (0.985–1.603)	0.066	1.084 (0.807–1.455)	0.593

Table 7. Univariate and Multivariate analysis results of Cox proportional hazards model to predict one year readmission.

ApoB is present in various atherogenic particles, including VLDL, IDL, large and small dense LDL particles (sd-LDL), making it a pivotal player in lipid transport to the blood vessel wall[19]. Compared to conventionally measured LDL-C, ApoB can more accurately reflect sd-LDL levels, particularly when LDL concentration diminishes. Its primary role is to facilitate the transport of lipoproteins to the blood vessel wall, representing atherogenic particles. On the other hand, ApoA-I predominantly resides in HDL particles, tasked with extracting excess cholesterol from peripheral cells and ferrying it to the liver through HDL particles. This reflects the number of anti-atherosclerotic lipoprotein particles and LDL concentration[20]. Consequently, the ApoB/ApoA-I ratio serves as a straightforward gauge of cholesterol transport balance in the body. A higher ratio indicates increased cholesterol in circulation, rendering it more prone to deposition in the vessel wall, thereby promoting atherosclerosis and cardiovascular events[21–23].

Our study analyzed patients undergoing PCI and found that among the patients grouped according to ApoB/ApoA-I ratio, biochemical indicators including TC, TG, LDL-C, HDL-C, LDL-C/HDL-C ratio, ApoA-I, ApoB, uric acid, fasting blood glucose, and glycated hemoglobin in the high ApoB/ApoA-I ratio group were significantly higher than those in the low ApoB/ApoA-I ratio group. Additionally, the proportion of diabetes and myocardial infarction in the high ratio group was significantly higher, as was the prevalence of multivessel disease and vascular occlusion compared to the low ratio group, with statistically significant differences observed. These findings underscore the ApoB/ApoA-I ratio's consistency with the severity of CHD, rendering it a valuable reference index for coronary atherosclerosis.

The INTERHEART and AMORIS studies, encompassing 52 countries, demonstrated that the ApoB/ApoA-I ratio exhibited the strongest predictive value for AMI among nine traditional cardiovascular risk factors (smoking, drinking, abdominal obesity, high blood pressure, diabetes, psychology, low physical activity, low fruit and vegetable intake, blood lipid). This predictive value surpassed that of any other blood lipid and blood lipid ratio index and remained independent of age, gender, and race[24,25]. Prior research also indicated that rosuvastatin treatment reduced serum lipid levels and improved cardiac function. Various factors such as different dosages of rosuvastatin, age, smoking, alcohol consumption, and diabetes independently contributed to the risk of AF and ischemic events[26]. Moreover, studies revealed a positive correlation between the ApoB/ApoA-I ratio and the lipid volume percentage of left main coronary artery plaques in patients with stable angina pectoris treated with statins. This suggests that individuals with a high ApoB/ApoA-I ratio face a greater risk of left main plaque progression and rupture compared to those with lower levels. Thus, maintaining a low ApoB/ApoA-I ratio is crucial for preventing left main plaque rupture.

In this study, the analysis of the correlation between the ApoB/ApoA-I ratio and other blood lipid indicators revealed that TC, TG, and LDL-C increased progressively with rising ApoB/ApoA-I ratio, while HDL-C exhibited a decreasing trend. TC, TG, and LDL-C were positively correlated with the ApoB/ApoA-I ratio, whereas HDL-C showed a negative correlation. Multivariate Cox regression identified vascular occlusion, the ApoB/ApoA-I ratio, statins, and CCB/ARB/ACEI as independent risk factors for one-year MACEs post-PCI. Vascular occlusion, Albumin, statins, and CCB/ARB/ACEI were independent risk factors for one-year readmission post-PCI. Cumulative hazard curves illustrated a significantly higher incidence of MACEs in the high ApoB/ApoA-I ratio group compared to the low ratio group, with a statistically significant difference. However, there was no statistically significant difference in the incidence of readmission between the high ApoB/ApoA-I ratio group and the low ratio group.

Our study highlights the ApoB/ApoA-I ratio as an independent risk factor for MACEs in post-PCI patients. Therefore, managing blood lipids in ASCVD patients and enhancing understanding of the ApoB/ApoA-I ratio may hold significant value for future ASCVD prevention and treatment. During clinical lipid-lowering treatment, the ApoB/ApoA-I ratio can serve as a reference indicator for assessing the efficacy of lipid-lowering medical interventions and guiding cardiovascular disease risk stratification.

However, it's important to acknowledge the limitations of this study. Firstly, it is a single-center study, which may lead to variations in the distribution of clinical indicators across different geographical and ethnic groups. It is anticipated that larger-scale multi-center studies will further enhance the clinical applicability of blood lipid

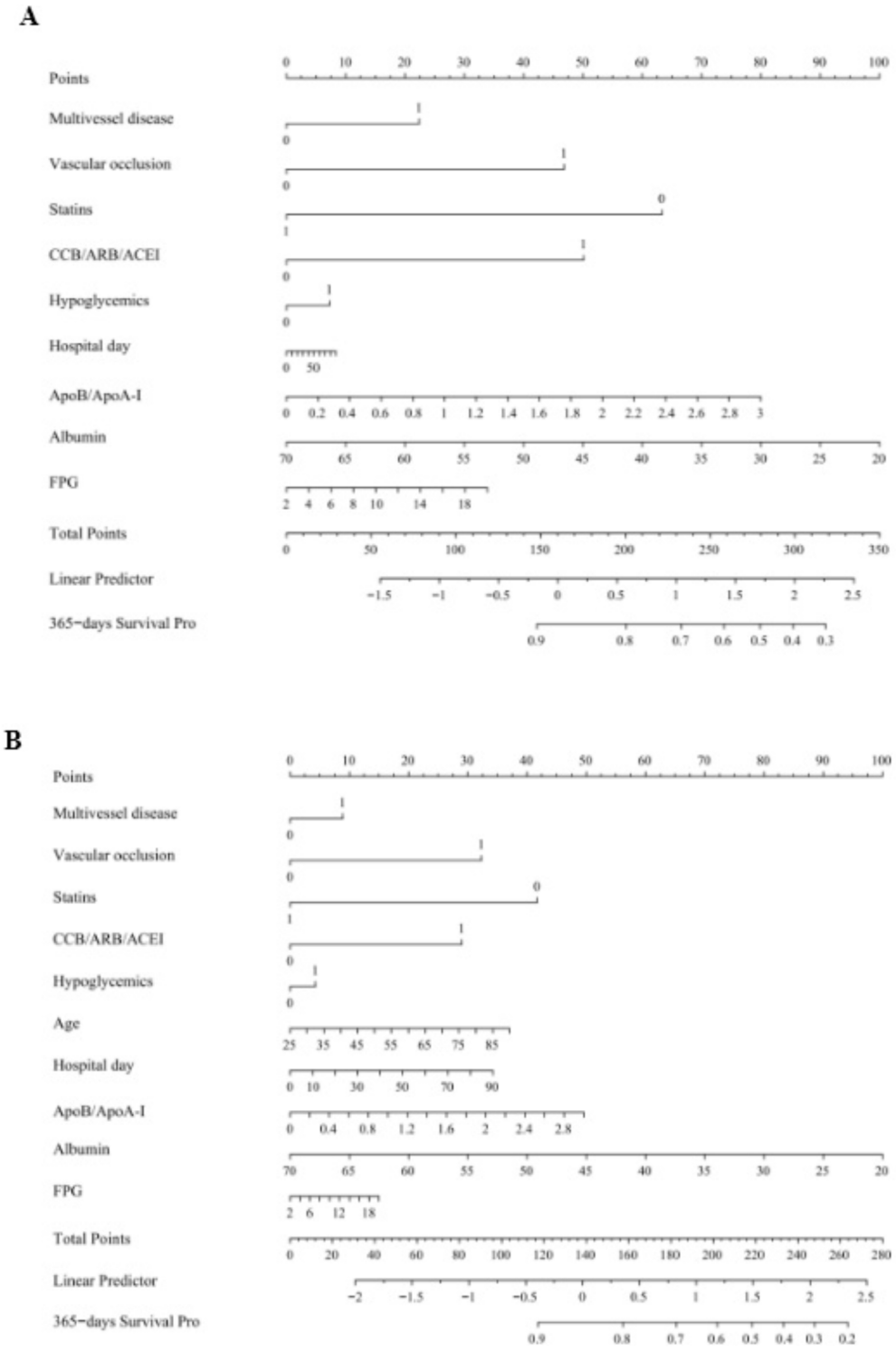

Fig. 4. Nomogram Model based on Multivariate Cox Regression Analysis in the Prediction of One Year MACEs and Readmission. (**A**) Nomogram for One Year MACEs (**B**) Nomogram for One Year Readmission.

indicators. Secondly, the follow-up period of this study was one year, and the data on the occurrence of MACEs after one year could not be fully obtained. The effect of ApoB/ApoA-I on long-term MACEs could not be deeply explored. Follow-up studies are still being carried out. Thirdly, the study was based on data available only at the time of admission and follow-up data, and lifestyle information such as diet and exercise was not collected, which may limit the comprehensiveness of the model to some extent.

Data availability

The datasets used and/or analysed during the current study are available from the corresponding author on reasonable request.

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Author contributions

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Declarations

Competing interests
The authors declare no competing interests.

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