THE TEXAS HEART INSTITUTE® JOURNAL

Clinical Investigation

Correlation Between Lipoprotein(a) and Prognosis for Coronary Artery Disease in Patients Undergoing Percutaneous Coronary Intervention

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Abstract

Background: Elevated lipoprotein(a) (Lp[a]) is a risk factor for first atherosclerotic thrombosis events, but the role of elevated Lp(a) in secondary prevention is controversial. This study aimed to retrospectively investigate the influence of elevated Lp(a) levels on the prognosis of patients with coronary artery disease.

Methods: The team collected and compared clinical information of patients hospitalized during percutaneous coronary intervention (PCI). This study used a multivariate logistic regression model to evaluate the relationships between Lp(a) levels, cardiovascular risk factors, and the prognosis of coronary artery disease in patients undergoing PCI.

Results: There were no statistically significant differences between patients grouped according to Lp(a) level in terms of sex; age; body mass index and obesity; hyperuricemia; smoking; cardiac insufficiency; acute myocardial infarction; multivessel lesion; in-stent restenosis; secondary PCI; apolipoprotein AI level; incidence of high total cholesterol or high low-density lipoprotein cholesterol; or family history of hypertension, diabetes, or coronary artery disease. The average Lp(a) concentration did not statistically significantly decrease after 1 year of statin treatment after PCI. One year after patients began statins, there were no significant differences between Lp(a) groups in the incidence of high triglycerides ($P = .13$), high total cholesterol ($P = .52$), or high low-density lipoprotein cholesterol ($P = .051$). Multivariate logistic regression analysis indicated that diabetes ($P = .02$) was associated with in-stent restenosis, whereas diabetes ($P = .02$) and multivessel lesions ($P < .001$) were associated with secondary PCI in patients who underwent coronary angiography 1 year after PCI. Compared with normal Lp(a) levels, high Lp(a) levels did not significantly increase the incidence of in-stent restenosis or secondary PCI in patients who underwent coronary angiography 1 year after PCI.

Conclusion: Sustained high concentrations of Lp(a) did not significantly increase the incidence of in-stent restenosis or secondary PCI in patients who underwent coronary angiography 1 year after PCI.

Keywords: Coronary artery disease; coronary restenosis; lipoprotein(a); percutaneous coronary intervention

Citation: ShaMa A, Ma C, Huang Y, Hu J, Xu C, Li Z, Wang J, Zeng C. Correlation between lipoprotein(a) and prognosis for coronary artery disease in patients undergoing percutaneous coronary intervention. *Tex Heart Inst J.* 2024;51(2):e238372. doi:10.14503/THIJ-23-8372

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Introduction

Coronary artery disease (CAD) is a common cause of death worldwide, with genetics and environmental factors affecting its development.[1] Despite secondary prevention measures, patients who underwent percutaneous coronary intervention (PCI) still face a high residual risk of CAD 1 year after PCI.[2] Lipoprotein(a) (Lp[a]) consists of low-density lipoprotein cholesterol (LDL-C) and apolipoprotein B_{100} (apo B_{100}), which are linked to plasminogen-like apo(a).[3] The physiologic function of Lp(a) has not been fully elucidated, although it is known to promote wound healing by transporting cholesterol to the injured site and plays a hemostatic role by inhibiting fibrinolysis.[4] It is also involved in cardiovascular diseases. Many studies have demonstrated that elevated Lp(a) increases the incidence of CAD.[5] The 2016 Canadian Cardiovascular Society guidelines for the treatment of dyslipidemia suggest that an Lp(a) level above 30 mg/dL is a risk factor for cardiovascular diseases,[6] whereas the European Atherosclerosis Society suggests that Lp(a) should be controlled below 50 mg/dL.[7] No approved, cost-effective, or safe treatment for reducing Lp(a) levels exists, however, and the benefits of existing therapy for patients with CAD remain uncertain.[8] Although elevated Lp(a) is known to be a risk factor for first atherosclerotic thrombosis events, the role of elevated Lp(a) in secondary prevention is still controversial.[9] The purpose of this study was to retrospectively investigate the relationship between Lp(a) levels and prognosis for CAD in patients undergoing PCI.

Key Points

- Statins had no effect on Lp(a) concentration in patients undergoing PCI.
- Diabetes was associated with in-stent restenosis, whereas diabetes and multivessel lesions were associated with secondary PCI in patients who underwent coronary angiography 1 year after PCI.
- There were no significant differences in the incidence of stable angina, unstable angina, chest distress, dyspnea, acute myocardial infarction, cardiac insufficiency, chronic kidney disease, or stroke among patients with different Lp(a) concentrations 1 year after PCI.
- Sustained high Lp(a) concentrations did not significantly increase the incidence of in-stent restenosis or secondary PCI in patients who underwent coronary angiography 1 year after PCI.

Abbreviations

apo, apolipoprotein
AUC, area under the curve
CAD, coronary artery disease
Lp(a), lipoprotein(a)
LDL-C, low-density lipoprotein cholesterol
OR, odds ratio
PCI, percutaneous coronary intervention

Supplementary Materials

For supplemental materials please see the online version of this paper.

Patients and Methods

Study Population

This single-center, retrospective study included 929 patients who underwent PCI followed by coronary angiography 1 year after PCI in the Department of Cardiology, Daping Hospital, The Third Military Medical University, from January 2017 to December 2019. Patient data, including cardiovascular risk factors, clinical features, and prognosis, were obtained from the hospital's electronic records. Patients who underwent PCI did not have coronary angiography 1 year after PCI, and these patients did not have an electronic record of blood lipids and were excluded. According to the normal reference range of blood lipids, high triglycerides were defined as a triglyceride concentration exceeding 1.70 mmol/L; high total cholesterol was defined as a total cholesterol concentration exceeding 5.72 mmol/L; and high LDL-C was defined as an LDL-C concentration exceeding 3.12 mmol/L. Coronary artery disease was defined by coronary angiography confirming that at least 1 epicardial main vessel stenosis was more than 50%. Multivessel lesions were defined by coronary angiography confirming that at least 2 epicardial main vessel stenoses were more than 50%. In-stent restenosis was diagnosed by coronary angiography, in which the narrow diameter of the lumen in the implanted segment of the stent and the proximal and distal 5-mm segments of the stent exceeded 50%. Family history of hypertension, diabetes, and CAD refers to immediate family members, including parents, brothers, and sisters. Secondary PCI was defined as PCI undertaken in patients with angina and at least 1 epicardial main vessel stenosis of more than 80% 1 year after PCI. This study was conducted in compliance with human studies guidelines and the Declaration of Helsinki, and this study was approved by the Ethical Committee of Daping Hospital.

Statistical Analysis

Values are presented as mean (SD) or number (%). A nonparametric Kruskal-Wallis test was used to compare continuous variables between groups, and the Bonferroni correction method was used to adjust multiple comparisons. Wilcoxon signed-rank test was used to compare blood lipid levels between the patients undergoing PCI, and then coronary angiography 1 year after PCI. Categorical variables between groups were compared using χ^2 or Fisher exact test. Receiver operating characteristic curve was used to evaluate the capacity of baseline Lp(a) levels to discriminate secondary PCI or in-stent restenosis. For a diagnostic test to be meaningful, the area under the curve (AUC) must be greater than 0.50. Generally, an AUC of 0.50 to less than 0.60 is considered a fail, 0.60 to less than 0.70 is considered poor, and 0.80 or more is considered acceptable. A multivariate logistic regression model was used to analyze factors potentially related to the incidence of in-stent restenosis and secondary PCI in patients undergoing coronary angiography 1 year after PCI, adjusted for baseline clinical characteristic factors, including age, smoking, gender, hypertension, diabetes, high total cholesterol, high triglycerides, high LDL-C, multivessel lesions, and Lp(a) group, with 95% CIs. $P<.05$ was considered statistically significant. All statistical analysis was carried out in SPSS, version 26, software (IBM Corp).

Results

Comparison of Clinical Characteristics of Patients With Different Lp(a) Concentrations

The 929 patients in this study were allocated to 1 of 4 groups according to their Lp(a) concentration: group 1 (normal: Lp(a) concentration 0-300 mg/L), group 2 (moderate: Lp(a) concentration 301-500 mg/L), group 3 (high: Lp(a) concentration 501-1000 mg/L), and group 4 (extreme: Lp(a) concentration >1000 mg/dL). The nonparametric Kruskal-Wallis test was used to compare the continuous variables, and the χ^2 or Fisher exact test was used to compare the categorical variables between Lp(a) groups. Baseline characteristics of the 929 patients are presented in Table I. As shown in Table I, Lp(a) groups did not differ by sex, age, body mass index, hypertension, diabetes, obesity, hyperuricemia, smoking, cardiac insufficiency, acute myocardial infarction, or multivessel lesion. There were no significant differences in the concentrations of total cholesterol ($P=.25$), high-density lipoprotein cholesterol ($P=.24$), LDL-C ($P=.10$), or apo AI ($P=.33$). There were no significant differences in the incidence of high total cholesterol ($P=.51$) or high LDL-C ($P=.15$). There were no statistically significant differences in family history of hypertension ($P=.35$), diabetes ($P=.85$), or CAD ($P=.28$). Furthermore, 66.20% of the patients had Lp(a) concentrations of 0 to 300 mg/L, 15.50% had Lp(a) concentrations of 301 to 500 mg/L, 14.21% had Lp(a) concentrations of 501 to 1000 mg/L, and 4.09% had Lp(a) concentrations greater than 1000 mg/L.

A multigroup comparison of continuous variables and categorical variables was conducted to assess the differences between Lp(a) groups. As shown in Supplemental Table I, the incidence of hypertriglyceridemia was greater in group 1 (normal) than in group 2 (moderate) ($P=.001$).

Changes in Blood Lipids During PCI and After 1 Year After PCI

Among 929 patients who received PCI and underwent coronary angiography 1 year after PCI, Wilcoxon signed-rank test was used to compare blood lipid levels between the patients undergoing PCI and 1 year after PCI. As shown in Supplemental Table II, after patients received statins for 1 year after PCI, the concentrations of triglycerides ($P<.001$), total cholesterol ($P<.001$), LDL-C ($P<.001$), and apo B ($P<.001$) were statistically significantly lower in the patients who underwent coronary angiography 1 year after PCI, although statins had no effect on the Lp(a) concentration in patients undergoing PCI ($P=.35$).

Comparison of Clinical Characteristics of Patients With Different Lp(a) Concentrations 1 Year After PCI

After grouping according to Lp(a) levels, 1 year after PCI, there were statistically significant differences in the average concentrations of triglycerides ($P=.03$), total cholesterol ($P=.007$), LDL-C ($P<.001$), and apo B ($P=.02$), whereas there were no statistically significant differences in the average concentrations of apo AI ($P=.54$) and high-density lipoprotein cholesterol ($P=.35$) (Table II). There were no statistically significant differences in the incidence of high triglycerides ($P=.13$), high total cholesterol ($P=.52$), or high LDL-C ($P=.051$). There were no statistically significant differences in the incidence of in-stent restenosis ($P=.39$) or secondary PCI ($P=.64$) between Lp(a) groups. There were no statistically significant differences in the inci-

TABLE I. Baseline Characteristics of Patients Undergoing Percutaneous Coronary Intervention, by Lp(a) Concentration (n=929)

Variable	Lp(a) concentration, mg/L				
	Normal (0-300) (n=615)	Moderate (301-500) (n=144)	High (501-1000) (n=132)	Extreme (>1000) (n=38)	*P* value[a]
Male sex, No. (%)	476 (77.40)	104 (72.22)	96 (72.73)	28 (73.68)	.45
Age, mean (SD), y	62.94 (10.74)	63.98 (9.73)	63.55 (9.81)	61.50 (10.62)	.62
Body mass index, mean (SD)	24.55 (3.29)	23.95 (3.11)	24.32 (3.33)	24.18 (3.48)	.22
Triglyceride, mean (SD), mmol/L	2.05 (1.76)	1.58 (0.97)	1.61 (0.75)	2.08 (1.93)	.01
Total cholesterol, mean (SD), mmol/L	4.32 (1.18)	4.44 (1.14)	4.53 (1.19)	4.83 (2.08)	.25
High-density lipoprotein cholesterol, mean (SD), mmol/L	1.05 (0.25)	1.08 (0.24)	1.07 (0.29)	1.13 (0.27)	.24
LDL-C, mean (SD), mmol/L	2.77 (0.91)	2.92 (0.93)	2.94 (0.89)	3.10 (1.51)	.10
apo AI, mean (SD), mmol/L	1.15 (0.23)	1.14 (0.22)	1.12 (0.23)	1.13 (0.19)	.33
apo B, mean (SD), mmol/L	0.92 (0.28)	0.96 (0.28)	0.99 (0.28)	1.00 (0.47)	.02
Hypertension, No. (%)	380 (61.79)	90 (62.50)	78 (59.09)	24 (63.16)	.93
Diabetes, No. (%)	164 (26.67)	32 (22.22)	32 (24.24)	12 (31.58)	.57
Obesity, No. (%)	34 (5.53)	5 (3.47)	6 (4.55)	2 (5.26)	.77
High triglycerides, No. (%)	273 (43.39)	42 (29.17)	46 (34.85)	16 (42.11)	.004
High total cholesterol, No. (%)	72 (11.71)	15 (10.42)	13 (9.85)	7 (18.42)	.51
High LDL-C, No. (%)	192 (31.22)	57 (39.59)	50 (37.88)	11 (28.95)	.15
Hyperuricemia, No. (%)	79 (12.85)	14 (9.72)	22 (16.67)	5 (13.16)	.40
Smoking, No. (%)	338 (54.96)	67 (46.53)	70 (53.03)	22 (57.89)	.07
Cardiac insufficiency, No. (%)	33 (5.37)	7 (4.86)	10 (7.58)	2 (5.26)	.75
Multivessel lesion, No. (%)	423 (68.78)	94 (65.28)	88 (66.67)	30 (78.94)	.33
Acute myocardial infarction, No. (%)	152 (24.72)	38 (26.39)	32 (24.24)	8 (21.05)	.92
Family history of hypertension, No. (%)	58 (9.43)	19 (13.19)	12 (9.09)	6 (15.79)	.35
Family history of diabetes, No. (%)	21 (3.41)	6 (4.17)	3 (2.27)	1 (2.63)	.85
Family history of coronary artery disease, No. (%)	24 (3.90)	7 (4.86)	6 (4.55)	4 (10.53)	.28

apo, apolipoprotein; LDL-C, low-density lipoprotein cholesterol; Lp(a), lipoprotein(a).

[a] $P < .05$ was considered statistically significant.

dence of stable angina ($P = .39$), unstable angina ($P = .08$), chest distress ($P = .07$), dyspnea ($P = .56$), acute myocardial infarction ($P = .40$), cardiac insufficiency ($P = .76$), chronic kidney disease ($P = .16$), or stroke ($P = .999$) between Lp(a) groups.

A multigroup comparison of continuous variables was then conducted that reached statistical differences between Lp(a) groups. As shown in Supplemental Table III, 1 year after PCI, the average apo B concentration was higher in group 3 (high) than in group 1 (normal) ($P = .001$). The average LDL-C concentration was higher in group 3 (high) than in group 1 (normal) ($P = .002$).

Capacity of Lp(a) Levels for Discriminating Secondary PCI or In-Stent Restenosis

The receiver operating characteristic curve was used to investigate the capacity of Lp(a) levels to discriminate secondary PCI or in-stent restenosis. As shown in Figure 1, Lp(a) level has no capacity to discriminate secondary PCI (AUC, 0.539 [95% CI, 0.50-0.58]; $P = .055$). Also, Lp(a) level has no capacity to discriminate in-stent restenosis (AUC, 0.536 [95% CI, 0.48-0.60]; $P = .24$).

Multivariate Logistic Regression Model to Analyze Risk Factors Related to Secondary PCI and In-Stent Restenosis

A multivariate logistic regression model was used to analyze risk factors associated with the incidence of secondary PCI, with 95% CIs. As shown in Table III, after adjustment for the clinical characteristic factors,

TABLE II. Comparison of Patient Clinical Characteristics, by Lp(a) Concentration 1 Year After PCI (N=929)

Variable	Lp(a) concentration, mg/L				
	Normal (0-300) (n=615)	Moderate (301-500) (n=144)	High (501-1000) (n=132)	Extreme (>1000) (n=38)	*P* value[a]
Triglycerides, mean (SD), mmol/L	1.67 (1.39)	1.30 (0.61)	1.59 (1.15)	1.65 (1.15)	.03
Total cholesterol, mean (SD), mmol/L	3.29 (0.95)	3.43 (0.86)	3.53 (0.97)	3.73 (1.64)	.007
High-density lipoprotein cholesterol, mean (SD), mmol/L	1.04 (0.25)	1.07 (0.22)	1.04 (0.23)	1.07 (0.26)	.35
LDL-C, mean (SD), mmol/L	1.97 (0.73)	2.11 (0.67)	2.20 (0.73)	2.33 (1.16)	<.001
apo AI, mean (SD), mmol/L	1.13 (0.21)	1.15 (0.22)	1.11 (0.21)	1.12 (0.18)	.54
apo B, mean (SD) mmol/L	0.65 (0.21)	0.69 (0.21)	0.72 (0.22)	0.74 (0.35)	.02
High triglycerides, No. (%)	185 (30.08)	29 (20.14)	37 (28.03)	11 (28.95)	.13
High total cholesterol, No. (%)	12 (1.95)	2 (1.39)	3 (2.27)	2 (5.26)	.52
High LDL-C, No. (%)	45 (7.32)	13 (9.03)	19 (14.39)	5 (13.16)	.051
In-stent restenosis, No. (%)	60 (9.76)	20 (13.89)	17 (13.33)	3 (7.89)	.39
Secondary PCI, No. (%)	193 (31.38)	49 (32.79)	48 (34.03)	14 (36.84)	.64
Stable angina, No. (%)	113 (18.37)	28 (19.44)	28 (21.21)	9 (23.68)	.39
Unstable angina, No. (%)	109 (17.72)	21 (14.58)	16 (12.12)	4 (10.53)	.08
Chest distress, No. (%)	368 (59.84)	81 (56.25)	69 (52.27)	18 (47.37)	.07
Dyspnea, No. (%)	45 (7.32)	7 (4.86)	10 (7.58)	2 (5.26)	.56
Acute myocardial infarction, No. (%)	26 (4.23)	4 (2.78)	4 (3.03)	1 (2.63)	.40
Cardiac insufficiency, No. (%)	50 (8.13)	7 (4.86)	13 (9.85)	3 (7.89)	.76
Chronic kidney disease, No. (%)	20 (3.25)	8 (5.56)	7 (5.30)	2 (5.26)	.16
Stroke, No. (%)	10 (1.63)	2 (1.39)	2 (1.52)	1 (2.63)	.999
Death, No. (%)	1 (0.16)	0	0	0	

apo, apolipoprotein; LDL-C, low-density lipoprotein cholesterol; Lp(a), lipoprotein(a); PCI, percutaneous coronary intervention.

[a] $P < .05$ was considered statistically significant.

diabetes (odds ratio [OR], 1.48 [95% CI, 1.07-2.05]; $P = .02$) and multivessel lesion (OR, 3.12 [95% CI, 2.23-4.52]; $P < .001$) increased the risk of secondary PCI in patients undergoing coronary angiography 1 year after PCI. Patients in group 2 (moderate) (OR, 1.12 [95% CI, 0.80-1.79]; $P = .40$), group 3 (high) (OR, 1.27 [95% CI, 0.84-1.92]; $P = .26$), and group 4 (extreme) (OR, 1.05 [95% CI, 0.51-2.13]; $P = .90$) have no obvious differences from patients in group 1 (normal) (reference) in risk of secondary PCI.

A multivariate logistic regression model was used to analyze risk factors associated with the incidence of in-stent restenosis, with 95% CIs. As shown in Table III, after adjustment for the clinical characteristic factors, diabetes (OR, 1.76 [95% CI, 1.10-2.79]; $P = .02$) increased the risk of in-stent restenosis in patients undergoing coronary angiography 1 year after PCI. Patients in group 2 (moderate) (OR, 1.50 [95% CI, 0.86-2.61]; $P = .16$), group 3 (high) (OR, 1.33 [95% CI, 0.73-2.40]; $P = .35$), and group 4 (extreme) (OR, 0.57 [95% CI, 0.16-2.03]; $P = .38$) have no obvious differences from patients in group 1 (normal) (reference) in risk of in-stent restenosis.

Discussion

Elevated Lp(a) levels increase the risk of CAD through thrombosis, atherogenesis, and inflammation.[10] The Lp(a) concentration is determined mainly by genes that vary from person to person,[11] and the Lp(a) concentration is unlikely to decrease with exercise and diet therapy.[12] Although there are acknowledged contributions of Lp(a) to atherosclerotic cardiovascular disease, there is a lack of well-established treatments for lowering Lp(a), standardization of assays, and common guidelines for

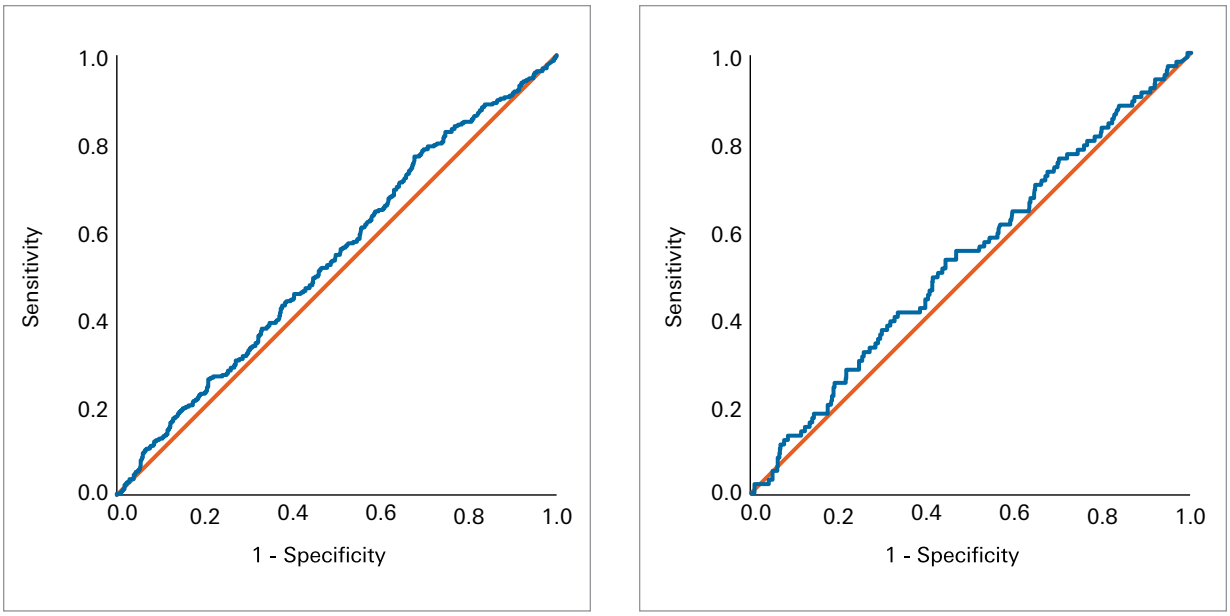

Fig. 1 Receiver operating characteristic curves show (**A**) the capacity of Lp(a) level for discriminating secondary percutaneous coronary intervention (AUC, 0.539 [95% CI, 0.50-0.58]; $P = .055$) and (**B**) the capacity of Lp(a) level for discriminating in-stent restenosis (AUC, 0.536 [95% CI, 0.48-0.60]; $P = .24$). $P < .05$ was considered statistically significant.

AUC, area under the curve; Lp(a), lipoprotein(a).

TABLE III. Multivariate Logistic Regression Model on the Association of Secondary PCI and In-Stent Restenosis With Cardiovascular Risk Factors

Variable	Adjusted OR (95% CI), *P* value[a]	
	Secondary PCI	**In-stent restenosis**
Lp(a), mg/L		
Normal, 0-300	1 [Reference]	1 [Reference]
Moderate, 301-500	1.19 (0.795-1.788), .40	1.50 (0.86-2.61), .16
High, 501-1000	1.27 (0.84-1.93), .26	1.33 (0.73-2.40), .35
Extreme, >1000	1.05 (0.51-2.13), .90	0.57 (0.16-2.03), .38
Smoking	1.05 (0.71-1.57), .80	0.84 (0.47-1.50), .55
Male sex	0.96(0.63-1.46), .85	1.02 (0.56-1.85), .94
Age	1.001 (0.99-1.02), .87	0.999 (0.98-1.02), .95
Hypertension	0.87 (0.64-1.18), .37	1.02 (0.65-1.61), .94
Diabetes	1.48 (1.07-2.05), .02	1.76 (1.10-2.79), .02
High total cholesterol	1.17 (0.66-2.06), .59	1.67 (0.74-3.77), .21
High triglycerides	0.81 (0.59-1.12), .20	0.75 (0.47-1.22), .25
High low-density lipoprotein cholesterol	0.74 (0.48-1.13), .16	0.53 (0.27-1.02), .06
Multivessel lesion	3.18 (2.23-4.52), <.001	1.45 (0.88-2.39), .15

Lp(a), lipoprotein(a); PCI, percutaneous coronary intervention.

[a] $P < .05$ was considered statistically significant.

diagnosing or assessing cardiovascular risk.[13] The effects of currently available lipid-modifying agents on Lp(a) are variable and modest, except for PCSK9 inhibitors, which reduced Lp(a) levels.[14] Research results on the effect of statins on Lp(a) level were different. In a meta-analysis of 5,256 patients from 6 randomized controlled trials, statins significantly increased plasma Lp(a) levels by 8.5% to 19.6%.[15] In another meta-analysis of 29,069 patients from 7 randomized controlled trials, the initiation of statin therapy had no significant effects on Lp(a) concentrations.[16] The results of this study indicated that statins can reduce the concentrations of total cholesterol and LDL-C and also can reduce the incidence of high total cholesterol and high LDL-C in various Lp(a) groups, but the average Lp(a) concentration was not significantly reduced in patients who underwent PCI (Supplemental Table II).

Most lipid-modifying therapies do not have a substantial effect on reducing Lp(a)-related major adverse cardiovascular events.[14] Compared with placebo, statin therapy did not lead to significant clinical differences in Lp(a) levels in patients at risk of cardiovascular disease.[17] PCSK9 inhibitors may reduce the Lp(a) concentration by increasing the scavenging effect, but the degree of this effect may not be sufficient to reduce the risk of Lp(a)-related cardiovascular disease in these patients.[18,19] It is still possible that the Lp(a)-lowering effect of PCSK9 inhibitors represents some added value on the basis of the extensive LDL-C–lowering effect.[11] Currently, there is no approved cost-effective and safe treatment for lowering Lp(a) levels. Although elevated Lp(a) is a risk factor for cardiovascular disease in general population studies, its contribution to the risk of cardiovascular events in patients with established cardiovascular disease is uncertain, and the role of elevated Lp(a) in secondary prevention remains controversial.[9,16] The results of this study indicated that 1 year after PCI, sustained high Lp(a) concentrations did not significantly increase the incidence of secondary PCI or in-stent restenosis in patients undergoing PCI, and there were no significant differences in clinical symptoms between Lp(a) groups 1 year after PCI (Table II). The results of the receiver operating characteristic curve showed that elevated Lp(a) levels cannot be used to predict secondary PCI or in-stent restenosis in patients undergoing PCI (Fig. 1). The results of multivariate logistic regression analysis further indicated that the main risk factors for secondary PCI were diabetes and multivessel lesions, and the main risk factor for in-stent restenosis was diabetes, whereas there were no significant differences in the incidence of secondary PCI or in-stent restenosis between Lp(a) groups 1 year after PCI (Table III).

Currently, the main challenges in the treatment of elevated Lp(a) are the side effects and uncertain cardiovascular benefits of drug therapies and the practicability of conventional lipoprotein separation.[20] It is insufficient to simply wait for Lp(a)-lowering drugs to work without doing anything,[21] and intervening in modifiable risk factors is even more important in the case of high Lp(a) concentrations.[22] The 2022 European Atherosclerosis Society Lp(a) consensus statement suggests that high Lp(a) levels should be interpreted in the context of other risk factors and absolute global cardiovascular risk, and the main targets for the treatment of patients with elevated Lp(a) levels are other cardiovascular risk factors.[23] Therefore, taking active measures to control other reversible cardiovascular risk factors, such as smoking, high LDL-C, hypertension, diabetes, and obesity, is important to reduce the impact of elevated Lp(a) on cardiovascular disease. Regardless of Lp(a) level, a healthy lifestyle is associated with a lower risk of CAD.[24]

Study Limitations

This study has several limitations. First, this was a retrospective study, and it is difficult to rule out the effects of activity and diet, which may introduce bias. Second, this was a single-center study, and various ethnic populations may have different results. Third, the sample size of this study was small, which may have led to some bias. Fourth, an imbalanced sample size is a main limitation of this study, which would probably limit statistical inference of the group effect of Lp(a).

Conclusions

Sustained high Lp(a) concentrations did not significantly increase the incidence of in-stent restenosis or secondary PCI in patients who underwent coronary angiography 1 year after PCI.

Article Information

Published: 17 December 2024

Author Contributions: Azhi ShaMa designed the study and prepared the manuscript. Ma, Huang, Hu, Xu, and Li performed the data collection. Chunyu Zeng and Jing Wang designed the study, reviewed the manuscript, and provided substantial improvements. All authors have read the final version of the manuscript and agreed with its content.

Conflict of Interest Disclosure: None.

Funding/Support: This study was supported by the National Natural Science Foundation of China, fund No. 81930008.

Ethics Approval: This study was conducted in accordance with the Declaration of Helsinki and approved by the Ethical Committee of Daping Hospital. This was a retrospective study, and the patient information was anonymized prior to analysis. Therefore, the Ethical Committee of Daping Hospital waived the requirement for informed consent.

Data Availability: The datasets used in this study were retrieved from the corresponding author according to the reasonable requirements.

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