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Original Article

Correlation between sLOX-1 and Omentin-1 Levels and the Severity of Coronary Artery Disease in the Elderly

Xi Chen [§], Na Zhang [§], Xiaohui Liang, Guangmei Hu, Xinjuan Xu [*]

Department of Hypertension, The First Affiliated Hospital of Xinjiang Medical University, Urumqi, Xinjiang, China

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SUMMARY

Background: This study aimed to analyze the correlation between the expression levels of soluble lectin-like oxidized low-density lipoprotein receptor-1 (sLOX-1) and omentin-1 and the severity of coronary artery disease in the elderly.
Methods: The clinical data of 117 elderly patients with coronary heart were retrospectively analyzed and divided into stable angina pectoris (SAP) group, unstable angina pectoris (UAP) group, and acute myocardial infarction (AMI) group. Another 50 health volunteers were included as control group.
Results: Serum sLOX-1 level in AMI group > UAP group > SAP group > control group, and omentin-1 level in AMI group < UAP group < SAP group < control group ($p < 0.05$); ROC curve results showed that AUCs of sLOX-1, omentin-1 and the combination of both predicted the occurrence of coronary heart disease were 0.930, 0.887, and 0.926. The serum sLOX-1 level, Gensini score, PWVβ, β, IMT, I CTP, CatK, MMP-2, MMP-9, TNF-α, hs-CRP, IL-6, IL-10 in severe group > moderate group > mild group. The omentin-1 level, CAC in severe group < moderate group < mild group ($p < 0.05$).
Conclusion: Serum sLOX-1 and omentin-1 levels are closely related to the severity of disease, carotid ultrasound parameters, plaque stability and inflammatory response in elderly patients with coronary artery disease, which can be used as biological markers to predict the occurrence of coronary artery disease.

1. Introduction

Coronary heart disease is characterized by multiple atherosclerotic lesions, luminal narrowing of coronary arteries, and reduced myocardial blood supply.[1] Currently, coronary angiography is the gold standard for clinical diagnosis of coronary artery disease, which can accurately and visually display the affected vessels. However, the relatively high cost of invasive tests and the compensatory function of the myocardium make early detection of the disease more difficult and delay the diagnosis and treatment.[2,3]

Inflammatory responses and abnormal lipid metabolism play an important role in the pathogenesis of coronary atherosclerosis.[4] The lectin-like oxidized low-density lipoprotein receptor-1 (LOX-1) can promote apoptosis of smooth muscle cells, enhance adhesion and chemotactic responses of inflammatory factors, enhance the ability of foam cells to phagocytose lipid substances through endocytose, bind, and degrade oxidized low-density lipoprotein (ox-LDL),[5,6] impair endothelial cell function, and aggravate inflammatory responses and oxidative stress in lesion foci.[7] Omentin is an important factor secreted by adipose tissue[8,9] to participates in and regulate the inflammatory response and improve the inflammatory state of the vascular endothelium.[10] However, the clinical relationship between changes in soluble lectin-like oxidized low-density lipoprotein receptor-1 (sLOX-1) and omentin-1 levels and coronary heart disease remains unclear. In this study, we analyzed the relationship between serum sLOX-1 and omentin-1 levels and the severity of coronary artery disease in elderly patients, so as to investigate the role of both indexes in the cardiovascular system.

2. Patients and methods

2.1. Clinical data

The clinical data of 117 elderly patients with coronary heart disease who visited our hospital from August 2019 to June 2021 were retrospectively analyzed and divided into stable angina pectoris (SAP) group, unstable angina pectoris (UAP) group, and acute myocardial infarction (AMI) group, including 66 males and 51 females; aged 60–89 years, with a mean age of (71.95 ± 5.38) years. Inclusion criteria: age ≥ 60 years; meeting the diagnostic criteria of coronary artery disease in the Guidelines for the diagnosis and treatment of stable coronary artery disease,[11] and the condition was clearly defined by coronary angiography and myocardial enzymology; complete clinical data. Exclusion criteria: concomitant immune system diseases, hematologic diseases, infectious diseases; presence of various acute and chronic infections; previous history of major surgery such as coronary artery bypass grafting, coronary stent im-

* Corresponding author. Department of Hypertension, The First Affiliated Hospital of Xinjiang Medical University, Urumqi, Xinjiang 830054, China.
E-mail address: zcxu2020@126.com (X.j. Xu)
§ Both authors contributed equally to this work and should be considered as equal first coauthors.

plantation or surgical treatment within 6 months before enrollment; concomitant valvular heart disease, arrhythmia, cardiomyopathy thyroid disease; combined diffuse intravascular coagulation, shock and other critical illness; cerebral infarction in the last 6 months or new cerebral hemorrhage; liver and kidney insufficiency. Another 50 cases of elderly volunteers were enrolled as the control group, including 29 males and 21 females; aged 61–86 years, with an average of (70.62 ± 4.32) years, no previous history of coronary heart disease, hypertension, hyperglycemia, etc. This study has been approved by the Ethics Committee of The First Affiliated Hospital of Xinjiang Medical University. All study participants provided written informed consent before participating in the study.

2.2. Methods

(1) Baseline data. Age, sex, height, weight, blood pressure, history of hypertension, history of diabetes, history of alcohol consumption, history of smoking and medication (aspirin, statins, nitrates, β-blockers, ACEI/ARB class) were collected from all subjects.
(2) Laboratory-related indicators. 5 mL of fasting venous blood was collected from all subjects (on the day of physical examination for the control group and before treatment for the coronary group), centrifuged at 3000 r/min for 10 min at 4 °C. The control group and before treatment for the coronary plasma was separated and stored in a refrigerator at -80 °C. sLOX-1, omentin-1, carboxy-terminal cross-linked telopeptide of type I collagen (ICTP), cathepsin K (CatK), matrix metalloproteinase-2 (MMP-2), matrix metalloproteinase-9 (MMP-9), interleukin-6 (IL-6) and interleukin-10 (IL-10) were measured by enzyme-linked immunosorbent assay (enzyme-linked immunoassay instruments were purchased from Beckman, USA). The levels of tumor necrosis factor-α (TNF-α) were measured by double antibody sandwich enzyme-linked immunosorbent assay (Kepong Xingye (Beijing) Technology Co.). The high sensitivity C-reactive protein (hs-CRP) level was determined by the immunoscattering turbidimetric assay (kit purchased from Guangzhou Jianlun Biotechnology Co., LTD.).
(3) Gensini score. ① Stenosis degree: 1 point: stenosis ≤ 25%; 2 points: 26%–50%; 4 points: 51%–75%; 8 points: 76%–90%; 16 points: 91%–99%; 32 points: 100%. ② Lesion location: 5 points: left main coronary artery disease; 2.5 points: left anterior descending branch or proximal circumflex branch; 1.5 points: left anterior descending branch middle part; 1 point: right coronary artery, middle and distal part of left circumflex branch, distal segment of left anterior descending branch; 0.5 points: the second diagonal branch and the posterior branch. The score of each lesion is the product of the score of the lesion and the score of the degree of stenosis, and the Gensini score is the sum of the integral of all the lesions. Mild: Gensini score of 1–20; moderate: Gensini score of 21–40; Severe: Gensini score > 40.
(4) Carotid ultrasound parameters. Carotid artery compliance (CAC), pulse wave velocity (PWVβ), total atherosclerosis (β), and arotid intima-media thickness (IMT) of the anterior and posterior walls were measured by a 4-dimensional color Doppler ultrasound diagnostic system (Philips Heartray IE33, Shanghai Jumu Medical Devices Co.).

2.3. Statistical analysis

SPSS 23.0 statistical software was used to test normality using the Shapiro-Wilk test. Measurement data conforming to the normal distribution were expressed as ($\bar{x} \pm S$), and one-way analysis of variance (ANOVA) was used for comparison between multiple groups; Spearman analysis was performed to analyze the correlation. Unconditional multivariate logistic regression was used to analyze the correlation between various factors and coronary heart disease, and receiver operating characteristic (ROC) curves were drawn to discriminate the correlation between serum sLOX-1, omentin-1 and disease-related indicators in patients with coronary heart disease. $p < 0.05$ indicates that the difference is statistically significant.

3. Results

3.1. Baseline data

There were no significant differences in terms of gender, age, and body mass index among four groups ($p > 0.05$). There were no statistically significant differences compared with the history of hypertension, history of alcohol consumption, and drug use among SAP, UAP, and AMI groups ($p > 0.05$). The percentage of SBP, DBP, history of diabetes, and history of smoking in the AMI group was higher than that in SAP and UAP groups ($p < 0.05$) (Table 1).

3.2. Serum sLOX-1 and omentin-1 levels

Serum sLOX-1 level in AMI group > UAP group > SAP group > control group, and omentin-1 level in AMI group < UAP group < SAP group < control group ($p < 0.05$) (Table 2).

3.3. Predictive value of serum sLOX-1 and omentin-1 levels for coronary artery disease

Systolic blood pressure (SBP), diastolic blood pressure (DBP), history of diabetes, history of smoking, and serum sLOX-1 and

Table 2
Comparison of serum sLOX-1 and omentin-1 levels in each group ($\bar{x} \pm s$).

Group	sLOX-1 (ng/L)	Omentin-1 (ng/mL)
Control group (n = 50)	669.66 ± 192.43	8.75 ± 2.22
SAP group (n = 42)	968.08 ± 201.90$^{###}$	6.39 ± 1.10$^{###}$***
UAP group (n = 40)	1205.72 ± 274.34$^{###}$***	5.60 ± 1.43$^{###}$***
AMI group (n = 35)	1540.33 ± 310.92$^{###}$***$^{+++}$	4.75 ± 1.28$^{###}$***$^{+}$

Note: AMI, acute myocardial infarction; SAP, stable angina pectoris; sLOX-1, soluble lectin-like oxidized low-density lipoprotein receptor-1; UAP, unstable angina pectoris; $^{###}$ $p < 0.001$ compared to control group; *** $p < 0.001$ compared to SAP group; $^{+}$ $p < 0.05$, $^{+++}$ $p < 0.001$ compared to UAP group.

Table 1
Comparison of baseline data ($\bar{x} \pm s$, n).

Group	Sex (M/F)	Age (years)	Body mass index (kg/m^2)	SBP (mmHg)	DBP (mmHg)	History of hypertension	History of diabetes mellitus	History of alcohol consumption	Smoking history	Drug use A/B/C/D/E
Control group (n = 50)	29/21	69.5 ± 5.2	22.62 ± 2.59	120.32 ± 4.16***	75.26 ± 3.26***	-	-	-	-	-
SAP group (n = 42)	24/18	70.6 ± 4.9	23.06 ± 3.02	129.62 ± 4.26***	76.02 ± 2.76***	29	5**	12	4***	41/42/34/21/26
UAP group (n = 40)	23/17	71.0 ± 5.1	23.12 ± 2.95	133.65 ± 3.65***	86.95 ± 3.46***	23	4**	10	10**	39/38/38/19/24
AMI group (n = 35)	19/16	69.9 ± 5.0	23.51 ± 2.86	142.26 ± 5.26	92.02 ± 5.16	20	15	11	15	35/35/32/19/25

Note: A, aspirin; AMI, acute myocardial infarction; B, statin; C, nitrate; D, β-blocker; DBP, diastolic blood pressure; E, ACEI/ARB; SAP, stable angina pectoris; SBP, systolic blood pressure; UAP, unstable angina pectoris; compared with AMI group, ** $p < 0.01$, *** $p < 0.001$.

omentin-1 levels with statistical difference among clinical groups were taken as independent variables [where SBP, DBP, serum sLOX-1 and omentin-1 were continuous variables, and history of diabetes and history of smoking were dichotomous variables (0 = none, 1 = yes)], and the presence or absence of coronary artery disease was used as the dependent variable (0 = not occurring, 1 = occurring) for unconditional multivariate logistic regression analysis. The results showed that SBP (β = 1.913, *S.E.* = 1.187, *Beta* = 0.146, *t* = 2.271, *p* = 0.021), history of diabetes (β = 3.528, *S.E.* = 1.621, *Beta* = 0.723, *t* = 2.381, *p* < 0.001), and serum sLOX-1 (β = 5.216, *S.E.* = 2.357, *Beta* = 1.144, *t* = 3.367, *p* < 0.001) and omentin-1 (β = -7.342, *S.E.* = 2.464, *Beta* = -0.681, *t* = -5.063, *p* < 0.001) levels were independent influencing factors of coronary heart disease, of which SBP, history of diabetes and serum sLOX-1 were risk factors and omentin-1 was protective factor. ROC curves were made for serum sLOX-1 and omentin-1 levels in relation to the presence or absence of coronary artery disease, respectively, and the results showed that the AUCs of sLOX-1, omentin-1 and the combination of both for predicting the occurrence of coronary artery disease were 0.930 (95% CI: 0.891–0.969), 0.887 (95% CI: 0.825–0.949), 0.926 (95% CI: 0.886–0.966), respectively (Table 3 and Figures 1-2).

3.4. Comparison of serum sLOX-1, omentin-1, and Gensini scores as well as carotid ultrasound parameters in groups with different degrees of coronary artery disease

Serum sLOX-1 level and Gensini score as well as PWVβ, β, and IMT in severe group > moderate group > mild group, whereas omentin-1 level and CAC in severe group < moderate group < mild group (*p* < 0.05) (Table 4).

3.5. Comparison of plaque stability indexes and inflammatory response indexes

Serum I CTP, CatK, MMP-2, MMP-9 levels as well as serum TNF-α, hs-CRP, IL-6 and IL-10 levels in severe group > moderate group > mild group (*p* < 0.05) (Table 5).

3.6. Correlation between serum sLOX-1 and omentin-1 and condition-related indicators

Gensini score, PWVβ, β, IMT, I CTP, CatK, MMP-2, MMP-9, TNF-α, hs-CRP, IL-6, IL-10 were positively correlated with sLOX-1 (*r* > 0, *p* < 0.05) and negatively correlated with omentin-1 in patients with coronary artery disease (*r* < 0, *p* < 0.05); CAC was negatively correlated with sLOX-1 (*r* < 0, *p* < 0.05) and positively correlated with omentin-1 (*r* > 0, *p* < *0.05*) (Table 6).

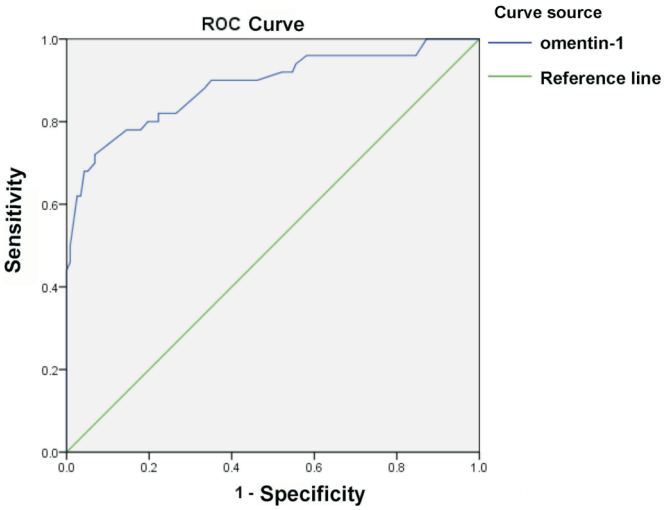

Figure 1. ROC analysis of omentin-1 for predicting coronary heart disease. ROC: receiver operator characteristic curve.

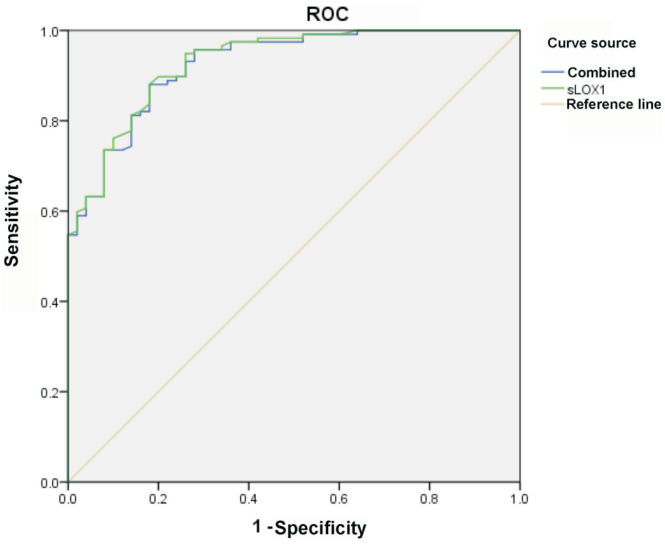

Figure 2. ROC analysis of sLOX-1, combined indexes in predicting coronary heart disease. ROC: receiver operator characteristic curve, sLOX-1: soluble lectin-like oxidized low-density lipoprotein receptor-1.

Table 3
Predictive value of serum sLOX-1 and omentin-1 levels for coronary artery disease.

Indicator	Cut-off value	Sensitivity	Specificity	Jorden Index	AUC	Standard error	*p*-value	95% CI
sLOX-1	1023.840 mmol/L	0.943	0.852	0.795	0.930	0.020	0.000	0.891–0.969
omentin-1	5.815 mmol/L	0.960	0.778	0.738	0.887	0.032	0.000	0.825–0.949
Combined	-	0.913	0.921	0.834	0.926	0.020	0.000	0.886–0.966

Note: AUC, area under the curve; sLOX-1, soluble lectin-like oxidized low-density lipoprotein receptor-1.

Table 4
Comparison of serum sLOX-1, omentin-1, and Gensini scores as well as carotid ultrasound parameters in groups with different degrees of coronary artery disease ($\bar{x} \pm s$).

Group	sLOX-1 (ng/L)	omentin-1 (ng/mL)	Gensini score (points)	CAC (mm^2/kPa)	PWVβ (m/s)	β	IMT (mm)
Mild group (n = 47)	1032.26 ± 195.26	7.26 ± 0.95	26.38 ± 4.15	0.84 ± 0.12	5.21 ± 0.54	4.09 ± 0.58	0.62 ± 0.12
Moderate group (n = 41)	1352.16 ± 201.21$^{###}$	6.02 ± 0.84$^{###}$	53.62 ± 6.94$^{###}$	0.61 ± 0.11$^{###}$	7.12 ± 0.64$^{###}$	6.85 ± 1.03$^{###}$	0.75 ± 0.14$^{###}$
Severe group (n = 29)	1509.62 ± 253.32$^{###}$***	4.39 ± 0.43$^{###}$***	86.62 ± 10.56$^{###}$***	0.43 ± 0.09$^{###}$***	9.95 ± 1.23$^{###}$***	8.95 ± 1.34$^{###}$***	0.92 ± 0.37$^{###}$***

Note: β, total atherosclerosis; CAC, carotid artery compliance; IMT, intima-media thickness; PWVβ, pulse wave velocity; sLOX-1, soluble lectin-like oxidized low-density lipoprotein receptor-1. Compared to the mild group, $^{###}$ *p* < 0.001; Compared to moderate group, *** *p* < 0.001.

Table 5
Comparison of plaque stability indexes and inflammatory response indexes in groups with different degrees of coronary artery disease ($\bar{x} \pm s$)

Group	I CTP (ng/mL)	CatK (pg/mL)	MMP-2 (μg/L)	MMP-9 (μg/L)	TNF-α (pg/mL)	hs-CRP (mg/L)	IL-6 (pg/mL)	IL-10 (pg/mL)
Mild group (n = 47)	10.32 ± 2.25	9.68 ± 1.67	84.62 ± 15.32	90.63 ± 17.16	0.62 ± 0.12	2.21 ± 0.34	1.21 ± 0.38	2.43 ± 0.35
Moderate group (n = 41)	$16.68 \pm 3.37^{\#\#\#}$	$18.62 \pm 3.32^{\#\#\#}$	$123.65 \pm 20.65^{\#\#\#}$	$120.35 \pm 21.62^{\#\#\#}$	$1.12 \pm 0.32^{\#\#\#}$	$4.16 \pm 0.48^{\#\#\#}$	$2.03 \pm 0.41^{\#\#\#}$	$4.02 \pm 0.43^{\#\#\#}$
Severe group (n = 29)	$25.65 \pm 5.28^{\#\#\#}$***	$30.35 \pm 5.02^{\#\#\#}$***	$163.45 \pm 25.67^{\#\#\#}$***	$172.63 \pm 25.67^{\#\#\#}$***	$2.31 \pm 0.58^{\#\#\#}$***	$6.63 \pm 0.37^{\#\#\#}$***	$2.65 \pm 0.35^{\#\#\#}$***	$8.13 \pm 0.59^{\#\#\#}$***

Note: CatK, cathepsin K; hs-CRP, high sensitivity-C-reactive protein; I CTP, carboxy-terminal cross-linked telopeptide of type I collagen; IL-6, interleukin-6; IL-10, interleukin-10; MMP-2, matrix metalloproteinase-2; MMP-9, matrix metalloproteinase-9; TNF-α, tumor necrosis factor-α. Compared to the mild group, $^{\#\#\#}$ $p < 0.001$; Compared to moderate group, *** $p < 0.001$.

Table 6
Correlation between serum sLOX-1 and omentin-1 and condition-related indicators in patients with coronary artery disease.

Indicator	Coefficients	Gensini score	CAC	PWVβ	β	IMT	I CTP	CatK	MMP-2	MMP-9	TNF-α	hs-CRP	IL-6	IL-10
sLOX-1	*r*	0.612	-0.513	0.613	0.597	0.632	0.538	0.411	0.796	0.513	0.671	0.621	0.499	0.532
	p	0.000	0.000	0.000	0.000	0.000	0.000	0.005	0.000	0.000	0.000	0.000	0.000	0.000
Omentin-1	*r*	-0.712	0.413	-0.632	-0.382	-0.483	-0.513	-0.486	-0.675	-0.742	-0.652	-0.513	-0.532	-0.602
	p	0.000	0.003	0.000	0.012	0.000	0.000	0.000	0.000	0.000	0.000	0.000	0.000	0.000

Note: β, total atherosclerosis; CAC, carotid artery compliance; CatK, cathepsin K; hs-CRP, high sensitivity-C-reactive protein; I CTP: carboxy-terminal cross-linked telopeptide of type I collagen; IMT, intima-media thickness; IL-6, interleukin-6; IL-10, interleukin-10; MMP-2, matrix metalloproteinase-2; MMP-9, matrix metalloproteinase-9; PWVβ, pulse wave velocity; sLOX-1, soluble lectinlike oxidized low-density lipoprotein receptor-1; TNF-α, tumor necrosis factor-α.

4. Discussion

With the aggravation of the condition of patients with coronary artery disease, the lumen of the blood vessel continues to narrow or obstruction. The necrosis of cardiomyocytes may develop due to hypoxia and ischemia, even inducing adverse cardiac events such as hospitalization for angina pectoris and cardiogenic death, which affect the prognosis.[12–14] It has been confirmed that sLOX-1 and omentin-1 are related to plaque formation, vascular endothelial inflammatory response, and endothelial dysfunction. In this study, it was found that serum sLOX-1 and omentin-1 levels may be related to the severity of coronary heart disease, carotid ultrasound parameters, plaque stability and inflammatory response in elderly patients, which can be used as biomarkers to predict the occurrence of coronary heart disease. It has been found that LOX-1 can promote rapid development of vulnerable plaques through many pathways including foam cell formation, vascular endothelial cell injury, collagen fiber degradation, and inflammatory response.[15] Activation of the ox-LDL/LOX-1 system leads to increased expression of vascular cell adhesion molecules, intercellular adhesion molecules, and monocyte chemotactic protein-1, which promotes adhesion and aggregation of lymphocytes and monocytes and aggravates the local inflammatory response of blood; inhibits NO release, reduces eNOS synthesis, and increases peroxide production in endothelial cells, which exacerbates endothelial cell damage;[16] downregulates c-IAP-1, Bcl-2 and other anti-apoptotic proteins expression, causing endothelial cell dysfunction and endothelial cell apoptosis, and promoting coronary heart disease.

In this study, serum sLOX-1 level in AMI group > UAP group > SAP group > control group, the AUC of sLOX-1 to predict the occurrence of coronary heart disease was 0.930, and sLOX-1 levels were positively correlated with Gensini score, carotid ultrasound parameters, plaque stability and inflammatory response. Çoner et al.[17] reported that serum sLOX-1 levels were higher in patients with UAP and AMI than in healthy subjects, and sLOX-1 levels were positively correlated with modified Gensini score and hs-CRP levels, similar to the findings of the present study. This indicated that high expression of sLOX-1 may be involved in the pathogenesis of coronary heart disease, which is related to the type of coronary heart disease, and may be a biological marker to predict the occurrence and severity of coronary heart disease. The reason may be that the increased level of oxidative stress in cerebrovascular tissue and the formation of reactive oxygen species can up-regulate the level of LOX-1 in vascular tissue, while the combination of LOX-1 with ligands such as CRP and ox-LDL can aggravate the local oxidative stress response and induce the release of cytokines such as NF-κB, which encourages neuronal apoptosis, plaque instability and the atherosclerotic plaque formation.[18] After phagocytosis of ox-LDL by sLOX-1, inflammatory responses such progressive thinning of fibrous cap of carotid plaque can occur, decreasing plaque stability, inducing plaque rupture and dislodgement and ultimately coronary heart disease.[19]

In this study, unconditional multivariate logistic regression analysis showed that SBP, history of diabetes and serum sLOX-1 and omentin-1 levels were independent influencing factors for the occurrence of coronary heart disease, of which SBP, history of diabetes and serum sLOX-1 were risk factors, and omentin-1 was protective factor. Serum omentin-1 levels: AMI group < UAP group < SAP group < control group, and the AUC for omentin-1 to predict the occurrence of coronary artery disease was 0.887. Askin et al.[20] showed that circulating omentin-1 levels were lower in patients with acute coronary syndrome and coronary artery disease compared to healthy individuals, which is basically consistent with the findings of the present study. Xu et al.[21] found that the serum levels of omentin-1 in stroke patients with unstable carotid plaques were lower than in patients with stable plaques, and omentin-1 was a risk factor for unstable plaques (OR = 0.31). Yoo et al.[22] also reported that serum omentin-1 was lowly expressed in patients with type 2 diabetes mellitus with carotid plaque, and both multiple regression analysis and multivariate Logit model analysis showed that omentin-1 was independently correlated with carotid plaque and arterial stiffness. In this study, omentin-1 was negatively correlated with Gensini score, PWVβ, β, IMT, I CTP, CatK, MMP-2, MMP-9, and positively correlated with CAC, further proving that serum omentin-1 was correlated with the severity of the disease and the property of carotid

plaque in patients with coronary heart disease.

Low omentin-1 levels may be a risk factor for the development of coronary artery disease and its mechanism of action may be related to (1) endothelial inflammatory response. Tan et al.[23] found in cultured umbilical vessels that omentin inhibited JNK activation and reduced TNF-α-induced cyclooxygenase (COX)-2 production. It can be hypothesized that omentin may reduce the endothelial inflammatory response. (2) Induced insulin resistance. Insulin resistance is a risk factor of coronary heart disease. The omentin-1 enhances insulin-mediated glucose uptake by adipocytes and promotes Akt phosphorylation, while its ability in promoting insulin-regulated glucose uptake is subsequently reduced when omentin-1 levels are lowered, thus causing insulin resistance and atherosclerosis. (3) Impaired vasodilatory function. The omentin-1 can regulate NO synthase in endothelial cells by activating the Akt signaling pathway, leading to endothelial dysfunction, which in turn aggravated atherosclerosis. Lapointe et al.[24] found that omentin-1 promotes endothelium-derived NO production and induces endothelium-dependent vasodilation in mesenteric arteries and aorta. Watanabe et al.[23] found that long-term infusion of omentin using an osmotic micropump prevented the appearance of lesions in apolipoprotein E knockout mice and facilitated the inhibition of macrophage infiltration and reduced the level of collagen fibers within atheromatous plaques.

In conclusion, serum sLOX-1 and omentin-1 levels are closely related to the severity of disease, carotid ultrasound parameters, plaque stability and inflammatory response in elderly patients with coronary artery disease, and they may be biological markers to predict the occurrence of coronary artery disease. However, there are still some limitations in this study. The changes in serum sLOX-1 and omentin-1 levels were not monitored dynamically, and the mechanism of action was not analyzed in depth. A large sample size, multicenter and prospective clinical study should be conducted in the future to verify the results. Meanwhile, all biomarkers were tested from the extracted blood samples at admission before treatment, and only the levels of the biomarkers were observed in patients who did not receive systematic treatment after diagnosis, which is a shortcoming of the study. Since it is a preliminary study, the changes of the above biomarkers before and after treatment were not observed. In the next step, we will further observe the changes of the above biomarkers before and after treatment to clarify the value of these biomarkers on the prognosis of the disease.

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Conflicts of interest

The authors confirm that there are no conflicts of interest.

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