Lp (a): An Independent Predictive Marker In Diseases

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Research Article

Keywords: Lp (a), blood lipid, atherosclerosis, myocardial infarction, correlation

Posted Date: February 7th, 2022

DOI: <https://doi.org/10.21203/rs.3.rs-1290162/v1>

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Abstract

objective To investigate the serum lipoprotein (a)(Lp (a)) level and analyze the correlation between Lp (a) and sex, age, smoking, drinking, different diseases and other blood lipid indexes.

Methods 7432 inpatients in Second Xiangya Hospital from 2013 to 2020 were selected, 8 items of medical records were collected, and statistic were analyzed by SSPS 24.0 software.

Results The differences of LP (a) in different age groups and smoking or not groups were statistically significant ($P < 0.05$). There were noteworthy differences in Lp (a) between chronic kidney disease (CKD) group, atherosclerosis (AS) group and AS patients with other diseases compared with the control group. Significant differences were seen in myocardial infarction (MI) and stroke groups, but not for the diabetes mellitus (DM) group and the high blood pressure group with the control group. The correlation coefficients of LP (a) with apolipoprotein B-100 (apoB), cholesterol (CHO), high-density lipoprotein (HDL-C) and lowdensity lipoprotein (LDL-C) were 0.179, 0.132, 0.143 and 0.143, respectively. Linear regression analysis showed that LP (a) level was positively correlated with age, smoking or not, AS, MI, CKD, $\beta=0.101, 0.042, 0.086, 0.119, 0.015$, respectively.

Conclusion LP (a) is related to apo-B, CHO, HDL-C and LDL-C, AS, MI and CKD.

Introduction

Lipoprotein (a) (LP (a)) is a special kind of lipoprotein, which is formed by covalent combination of lowdensity lipoprotein (LDL) containing apolipoprotein B-100 (apoB) and apolipoprotein (a) (apo (a)) through disulfide bond. The plasma concentration of LP (a) mainly depends on the synthetic rate of LP (a), but not on the decomposing rate. The LP (a) level in plasma varies greatly among individuals and is rarely affected by diet and environment lead to no significant fluctuation in body[1, 2].

Studies have shown that LP (a) may play a key role in promoting atherosclerosis (AS) through fibrinolysis and lipid metabolism, and it is an independent risk factor for atherosclerosis[3, 4]. However, the evidence of clinical studies are still insufficient. The regulatory target of blood lipid for people at risk of cardiovascular and cerebrovascular diseases published by authoritative institutions is still to reduce LDL-C. At the same time, the study also found that LP (a) levels, apo (a) subtypes and link pack area (LPA) single nucleotide polymorphisms have racial differences, and the degree of cardiovascular and cerebrovascular disease risk caused by increased LP (a) levels also varies with race[5]. At present, the majority of studies are according to white people, while the number of Asian people is relatively small. Therefore, in order to further improving the evidence chain of LP (a) with cardiovascular and cerebrovascular diseases in Chinese population, and to understand its influencing factors, this study analyzed the correlation between Lp (a) and some clinical survey data, so as to provide references for clinical diagnosis, treatment and prevention.

Methods

2.1 Study subjects

Information was collected regarding people who were hospitalized in the Second Xiangya Hospital of Central South University (Changsha, China) between 2013 and 2020. The inclusion criteria of each disease were clearly diagnosed according to the clinical diagnosis and treatment guidelines. The hypertension group was based on the Chinese

norms for the health management of hypertension, the atherosclerosis group according to the ESC Arteriosclerosis guidelines, the stroke group was in accordance with the Chinese guidelines for the diagnosis and treatment of acute ischemic stroke. According to the STEMI guidelines, the diabetic group was guided by the ADA diabetes guideline and CDK group gist of the KDIGO guidelines. Smoking group inclusion criteria: smoking ≥ 1 cigarette per day and smoking years ≥ 1 year. The inclusion criteria of drinking group: drinking ≥ 22 alcohol per day and drinking for ≥ 1 year. The control group is the patients hospitalized in the same period without the above diseases. The exclusion criteria were as follows: pregnancy and postpartum, congenital heart disease, impaired liver function, rapid deterioration of renal function, malignant tumor, autoimmune disease, blood system disease, serious infection, etc. Finally, 4838 cases selected in the disease group and 2594 cases bring into the control group; There were totally 7432 subjects, including 4359 males and 3073 females, all subjects were randomly selected (mean age, 50.0 ± 14.0 years, ranging from 18.0 to 89.0 years). According to the standard of China hypertension health management standard, patients are divided into normal blood pressure, grade 1 hypertension (systolic blood pressure 140~159 mmHg and/or diastolic blood pressure 90~99 mmHg), grade 2 hypertension (systolic blood pressure 160~179 mmHg and/or diastolic blood pressure 100~109 mmHg) and grade 3 high blood pressure (systolic blood pressure ≥ 180 mmHg and/or diastolic blood pressure ≥ 110 mmHg). The patients were divided into youth group (18.0~44.0 years old), middle-aged group (45.0~59.0 years old) and elderly group (60.0 years old and above). According to their normal lifestyle, they were divided into smoking group/non-smoking group and drinking group/nondrinking group. The disease group was divided into diabetes group, coronary artery group, carotid artery, cerebral atherosclerosis group, CKD (hereinafter referred to as group) and no as group; According to whether there is old or new myocardial infarction and stroke, it can be divided into myocardial infarction group/non-myocardial infarction group and stroke group/no stroke group.

The study protocol was approved by the Ethics Committee of the Second Xiangya Hospital of Central South University. All experiments were performed in accordance with relevant guidelines and regulations.

The informed consent was obtained from all participants or, if participants are under 18, from a parent and/or legal guardian.

2.2 Specimen collection and detection

2.2.1. Specimen collection

All participants were required to maintain their normal lifestyle and avoid strenuous physical exercise for 3 days before blood collection. The individual was asked to empty the stomach and be rest, then 3-4 ml venous blood was collected for examination. The random specimen was collected in the procoagulant tube for about 3-4 ml. The serum was centrifuged within 2 hours after collection.

2.2.2 Specimen detection

LP (a), cholesterol (CHO), triglyceride (TG), high density lipoprotein cholesterol (HDL-C), low density lipoprotein cholesterol (LDL-C), apolipoprotein A1 (ApoA1) and apoB were detected.

2.2.3 Chemicals

The Hitachi 7600 automatic biochemical analyzers and their matching reagents were used. LP (a) test kit (immunoturbidimetric method) and supporting calibrators and quality control materials were provided by Shanghai Changzheng company; CHO, TG, HDL-C and LDL-C kits were provided by heguangchun company in Japan; The

ApoA1, apoB kits and the related calibrators and quality control materials are provided by Ningbo Meikang company. All tests should be controlled by internal quality control before testing.

2.3 statistical analysis

Statistical analysis of the data was carried out using the SPSS 24.0. All data were outliers using the Dixon test. The distribution was tested by Kolmogorov Smirnov test. When $P > 0.05$, the data are obeyed the Gaussian distribution, otherwise the

data are non-Gaussian distributed. Turkey method is used in multiple Gaussian distributed groups, and the differences among groups are analyzed by ANOVA

analysis. Values are expressed as median $\pm$ SD. If it does not meet the Gaussian distribution, use the non-parametric test of Mann-Whitney U test for comparison, and Kruskal Wallis test is used for multiple groups. The data are represented by the median (P25, p75). In the linear regression analysis of each factor, gender is a categorical variable (male = 1, female = 2); Age is a hierarchical variable (Youth = 1, middle age = 2, old age = 3); Hypertension is a grade variable (normal blood pressure = 0, grade 1 hypertension = 1, grade 2 hypertension = 2, grade 3 hypertension = 3); Whether AS, MI, stroke, diabetes, smoking and drinking were classified variables (no = 0, Yes = 1). The correlation analysis of nonnormal distribution data adopts Sperman correlation coefficient. When binary logistic regression is carried out for each factor, box Tidwell method is used to test whether the conversion value between continuous independent variable and dependent variable logit is linear, and judge whether continuous independent variable needs to be converted into ordered classification variable; Then, multiple linear regression is used for collinearity test to evaluate whether there is multicollinearity between their variables. After eliminating the independent variables with multicollinearity, binary logistic regression is used to evaluate the impact of multiple factors on the disease. Results were considered statistically significant at a p-value < 0.05 .

Results

3.1 Comparison of serum LP (a) levels in different gender, age, smoking and drinking groups

All subjects were divided into groups according to gender, age, smoking and drinking, and the differences of LP (a) levels among each group were compared. The distribution of LP (a) was judged non normal distribution, and the data were expressed in median (P25, p75). The differences between groups were statistically analyzed by nonparametric test. There was no significant difference in serum LP (a) levels between men and women, drinking group and nondrinking group ($P > 0.05$, respectively). But significant difference was found in Lp (a) among adolescent, middle-aged and elderly groups ($P < 0.05$). Meanwhile, LP (a) in smoking group was obviously higher than that in non-smoking group. The level of LP (a) in the disease group was significantly higher than that in the control group. The results are shown in Table 1.

3.2 Comparison of LP (a) with smoking and drinking

7171 patients were divided into four groups: no smoking / no drinking, smoking / no drinking, drinking / no smoking and smoking / drinking. The results showed that the LP (a) level of smoking alone was the highest, and that of drinking alone was the lowest. The difference was statistically significant($p < 0.05$). Table 2. Showed that male and middle-aged groups had higher risks among all groups people.

3.3 Comparison of LP (a) between different disease groups and control group

Table 3 summarized the difference of LP (a) between the disease group and the control group was statistically significant ($p < 0.05$). Moreover, no significant difference was observed in groups that the patient with hypertension (among different grades), diabetes, and HBP+DM with the control group ($p > 0.05$). But for AS group and AS patients with other diseases compared with the control group, the differences were statistically significant ($p < 0.05$). The study also showed that there were significant differences between the groups that patients with myocardial infarction and stroke with the control group ($p < 0.05$).

3.4 Linear regression analysis of LP (a) with sex, age, smoking, drinking and disease

Logarithm transformation was processed to meet the approximate normal distribution. After conversion, Lp (a) was analyzed by linear regression analysis with gender, age, smoking status, drinking condition, and whether hypertension, AS, diabetes, myocardial infarction and stroke were found. The results showed that Lp (a) level was correlated with age, smoking, AS, myocardial infarction, stroke, chronic kidney disease and hypertension ($P < 0.05$). By comparing the standardization coefficient β , LP (a) was most significantly correlated with myocardial infarction, AS and age. The results showed that LP (a) level was still correlated with smoking and chronic kidney disease, but not with other factors and diseases ($P > 0.05$). The results are shown in Table 4.

3.5 Correlation between Lp (a) and blood lipid index and EGFR

Spearman correlation analysis was made between Lp (a) and other blood lipid indexes and EGFR. The results are shown in Table 5. LP (a) level was correlated with apo-B, HDL-C, LDL-C and Chol ($P < 0.05$), but not with TG, apo-A1 and EGFR ($P > 0.05$). By comparing the correlation coefficients, it is found that LP (a) has the most significant correlation with apo-B. The results are shown in Table 5.

3.6 Logistic regression analysis of LP (a) and other factors on as and myocardial infarction

3.8.1 two variables logistic regression (stepwise method) was used to assess the impact of continuous variables (Lp (a), TG, CHOL, HDL-C, LDL-C, Apo-A1, Apo-B, age) and categorical variables (gender, hypertension history, diabetes history, smoking history, drinking history) on AS (dependent variable) in the study subjects. Firstly, the box Tidwell method is used to test whether the logit conversion values of continuous independent variables and dependent variables are linear. 22 items are included in the linear test model. The significance level after Bonferroni correction is 0.0023. The linear test results showed that there is a linear relationship between all continuous independent variables and the logit conversion values of dependent variables, which does not need to be converted into ordered classification variables. Then, multiple linear regression was used for collinearity test to evaluate whether there was multicollinearity between their variables. The results showed that CHOL, HDL-C, LDL-C and ApoB had obvious multicollinearity. After excluding CHOL and ApoB, the verification continued. The results showed that there was no collinearity between their variables. Finally, logistic regression results showed that AS's influencing factors included Lp (a), gender, age, diabetes, hypertension and whether smoking. $\chi^2 = 384.1$, $P < 0.001$, the model was established. The p value, or and 95% CI of each influencing factor are shown in Table 6

3.8.2 the two variables logistic regression was performed according to the above steps. The results showed that the related factors of MI were Lp (a), LDL-C, Apo-A1, age, gender, history of hypertension and diabetes. $\chi^2 = 404.8$, $P < 0.001$, the model was established. The p value, or and 95% CI of each influencing factor are shown in Table 7

3.8.3 When LP (a) is 430nmol / L (recommended by ESC guidelines), binary logistic regression is performed with coronary heart disease and myocardial infarction factors. It is found that the or values of both factors are greater

than 1, and the or values of myocardial infarction factors in coronary heart disease population are also greater than 1, which indicates that when LP (a) is 430nmol / L, the risk of coronary heart disease is 1.779 times that of patients without coronary heart disease, The risk of myocardial infarction is 1.626 times higher than that without myocardial infarction, and the risk of myocardial infarction in the population with coronary heart disease is 1.626 times higher than that without myocardial infarction. The results are shown in Table 8.

Discussion

LP (a) is composed of LDL like particles, containing all the atherosclerotic components of LDL and apolipoprotein. The mechanisms of apolipoprotein promotes atherosclerotic thrombosis through inflammation caused by oxidized phospholipids (OXPL) and potential anti fibrinolysis by inhibiting plasminogen activation^[6]. However, based on the low LP (a) level of most patients at risk of cardiovascular disease (70% ~ 80%) , and the significantly increase of LDL-C, most of the risk driven by apoB is due to the large number of LDL particles^[7]. Many clinicians believe that LP (a) is no longer a risk factor when LDL-C is controlled. Recent studies have shown that the incidence of adverse cardiovascular events is still more than 30% after the use of drugs to reduce LDL^[8] Some studies believe that part of this "residual risk" is caused by the high rise of LP (a)^[9-11]. LP (a) varies among different races^[12-14], and the pathogenic risk of LP (a) also presents different levels. There are few research reports based on the Chinese population. The risk early warning value (430nmol / L) of LP (a) mentioned in the latest Chinese expert consensus on the prevention and treatment of hypertension complicated with atherosclerosis^[15] is also quoted from the guidelines of ESC^[16]. Therefore, it is imperative to evaluate LP (a) and cardiovascular disease risk in Chinese population as soon as possible.

Linear correlation analysis of blood lipid indexes LP (a) has correlation with apo-B, HDL-C, LDL-C and CHOL, and apo-B is the most significant. The reason may be that LP (a) itself contains part of apo-B and at the same time, this mechanism also affects the correlation of other indexes, such as LDL-C and CHO. At present, all clinical indicators recommend that patients with cardio cerebrovascular sclerosis should routinely use drugs to reduce LDL-C. Studies have shown that statins may increase LP (a) by 10 ~ 20% while reducing LDL-C^[17], which makes the correlation between Lp (a) and other blood lipid indexes complicated.

It is reported that the concentration of LP (a) in blood is mainly affected by genetic factors, and the changes of diet, exercise and other lifestyle have little effect^[18]. In this study, the group comparison results showed that there was no significant difference in Lp (a) between men and women, drinking group and non-drinking group, whether in the control group or all subjects. The corrected linear regression analysis also showed that LP (a) had no correlation with gender and drinking status, which was basically consistent with the literature reports. However, after subdividing the living habits of patients, it is found that the LP (a) level of patients who smoke alone and drink alone is statistically significant, and the LP (a) level of patients who drink alone decreases. A foreign report^[19] once described that low to moderate drinking can decrease blood lipid levels, which is consistent with the results of this study. In addition, based on our analysis, we believed that we cannot infer that LP (a) increases with age, because as a horizontal study, there is no track or investigate the changes of LP (a) of each subject with age, which needs to be confirmed by further research.

Studies have shown that LP (a), as a risk factor of cardiovascular disease, can also predict the degree of coronary artery stenosis and the need for revascularization^[20]. At present, most reports at home and abroad point out that LP (a) level is correlated with AS, myocardial infarction, hypertension and other diseases. The comparison results of

disease groups in this study show that the LP (a) of AS group, AS combined with other diseases, myocardial infarction group and stroke group are significantly higher than that of the control group, the difference is statistically significant. But these diseases are interrelated, and some subjects suffer from a variety of diseases, so the interaction between diseases should be excluded. Therefore, the linear regression analysis of LP (a) after log₁₀ conversion and other factors was carried out in this study. The results showed that LP (a) level was correlated with AS, myocardial infarction, stroke and hypertension, which was consistent with our expectations and most of the reported results. After adjusting for age and gender factors, multivariate regression analysis showed that LP (a) level was still significantly correlated with AS and myocardial infarction, but not with stroke. This suggests that the higher the level of LP (a) in patients, the greater the risk of AS and myocardial infarction, which is of certain significance for the prediction and prevention of these diseases^[21]. At the same time, it may infer that when the disease develops to stroke, the level of LP (a) has a greater impact of age and gender. Logistic regression analysis was conducted for the prevalence of AS and the incidence of old and new myocardial infarction. The results showed that the influencing factors of AS included Lp (a), Apo-B, LDL-C, TG, age, diabetes, hypertension and whether smoking, and the related factors were Lp (a), which further explained the significance of Lp (a) detection in patients with myocardial infarction. Before that, many retrospective studies analyzed the clinical value of LP (a) as a prevention index of myocardial infarction, but did not make an in-depth study on the situation when the patient's LP (a) level was at the risk early warning value. However, this study analyzed the risk early warning value (430nmol / L) of LP (a) level and the influencing factors of coronary heart disease and myocardial infarction for the first time. It also shows that people with coronary heart disease whose LP (a) level is the risk early warning value are more likely to suffer from myocardial infarction at the same time.

Hypertension is a systemic disease characterized by vascular remodeling, which is often accompanied by abnormal changes in lipid metabolism. It is reported in the literature that 70% of patients with hypertension ^[22, 23]. Other studies have shown that LP (a) is also related to acute cerebral infarction ^[24] and ischemic stroke ^[25]. The results of this study showed that LP (a) level is correlated with hypertension, but not correlated with hypertension after adjusting age and gender factors, suggesting that this difference may be caused by patients suffering from a variety of diseases and the influence of gender and age factors. It is also reported that the main risk factors of stroke are 10 factors such as hypertension and smoking ^[26, 27], which is consistent with our conclusions obtained in this paper. The further studies are asked to explore the relationship between Lp (a) and stroke and hypertension.

Chronic kidney disease (CKD) is a common outcome disease caused by the development of kidney related diseases. The time of renal injury in patients is generally more than 3 months, and often leads to the occurrence of chronic cardiac insufficiency ^[28], with a very high mortality. CKD patients usually accelerate atherosclerosis due to increased inflammation, disturbance of lipid metabolism and other mechanisms, and eventually lead to myocardial infarction or ischemic stroke. Literature ^[29] shows that the decrease of arteriosclerosis is related to the increase of patient survival rate. As clinical researchers, we explored whether LP (a) can be used as a judgment index to reduce vascular hardness and improve vascular function in patients with CKD, so as to improve the survival rate of patients. At present, research reports of Japanese scholars ^[30] point out that LP (a) is significantly related to CKD and independent of other factors, which is basically consistent with the research results of this paper.

Not considering the influence of drugs is a deficiency of this study. Traditional lipid-lowering drugs such as statins have little effect on LP (a) level, but affect the level of other blood lipid indexes. In logistic regression analysis, LDL-C, TG and other indexes have weak correlation with AS and myocardial infarction, which may be related to patients taking lipid-lowering drugs. At present, a variety of drugs have been proved to reduce LP (a) level ^[31, 32]. Some

research results showed that reducing LP (a) can reduce the occurrence of cardiovascular events [33], which is a good news for people at risk of increasing LP (a).

In conclude, this paper takes living habits as the starting point to draw out the correlation between various diseases and LP (a) level, which is a progressive research method. This study points out that LP (a) is correlated with apo-B, CHO, HDL-C and LDL-C. LP (a) level is significantly correlated with AS and myocardial infarction, and has a certain correlation with CKD. When LP (a) level is 430nmol / L, the risk of simultaneous AS and myocardial infarction increases. This suggests that LP (a) is closely related to AS and may be a risk predictor of cardiovascular events and chronic kidney disease. In addition, this paper found that the living habits of simple drinking will reduce LP (a) level, which may suggest that the influence of simple factors is of greater significance when studying LP (a) level. The next research work of this study should collect more medical history data, broaden the research scope, and continue to follow up cases, so as to provide more basis for the study of LP (a) level and influencing factors.

Declarations

Author Contributions

M.W. and Z.X.: designed the research; Z.X.: conducted the research; W.L., Z.X.: collected the data; M.W.: analyzed the data and performed the statistical analysis; M.W.: wrote the manuscript; Z.X. edited the manuscript and had primary responsibility for final content; and all authors: read and approved the final version of the manuscript.

Conflict of Interest

The authors declare that the research was conducted in the absence of any commercial or financial relationships that could be construed as a potential conflict of interest.

Notes

The views expressed in this article are the personal views of the author(s) and may not be understood or quoted as being made on behalf of or reflecting the position of the regulatory agency/agencies or organisations with which the author(s) is/are employed/affiliated.

Availability of Data and Materials

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

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Tables

Table 1 Comparison of serum LP (a) levels in different gender, age, smoking and drinking groups

		All		Control group		Disease group	
		n	Lp(a) (mg/L)	n	LPa (mg/L)	n	Lp(a)(mg/L)
All		7432	151(73,327))	2594	128(65,275)	4838	167(78,357)
Sex	Male	4359	154(73,332)	1263	126(65,262)	3096	168(77,362)
	Female	3073	148(73,325)	1331	129(66,287)	1742	166(80,347)
Age	Teen	1155	120(58,267)	833	111(56,245)	322	148(68,387)
	Middle age	2673	143(70,319)*	1135	128(66,274)*	1538	157(74,351)
	Elder	3604	173(81,347)*	626	158(79,314)*	2978	176(82,358)*
Smoke	No smoking	3353	145(72,317)	1214	124(62,279)	2139	158(78,332)
	Smoke	3818	165(78,346)*	1189	142(70,280)*	2629	179(80,382)*
Drink	No drink	3699	151(74,331)	1263	125(63,280)	2436	169(79,358)
	Drink	3472	159(76,333)	1140	139(70,278)	2331	171(79,364)

Table 2 Comparison of LP (a) with gender, age, smoking and drinking

	n	non-smoking/ non-drinking	n	Smoking / non-drinking	n	Drinking / non-smoking	n	Smoking / drinking	P
ALL	3129	246.0±263.8	570	287.2±288.4	224	224.0±274.7	3248	256.3±268.6	0.000
Male	1007	240.9±264.5	534	288±292.4	190	226.1±278.2	2478	256.2±270.0	0.005
Female	2122	248.4±263.5	36	275.3±223.9	34	212.3±257.9	770	256.5±264.3	0.666
Teen	506	231.6±284.6	64	224.9±252.0	37	196.8±231.3	484	222.1±267.1	0.866
Middle	1060	225.4±250.1	192	288.6±292.9	89	214.6±272.6	1246	249.1±250.4	0.005
Elder	1563	264.6±264.7	314	299.1±297.8	98	242.8±292.3	1518	273.1±282.0	0.158

Table 3 Comparison of LP (a) between disease groups and control group

Groups	n	Lp(a) (mg/L)
Control group	2516	128(65,275)
Disease group	4879	167(78,357)*
Hypertension	960	140(70,304)
<u>Grade-1</u>	59	103(78,219)
Grade-2	248	137(72,318)
Grade-3	382	160(62,276)
AS	1038	171(81,394)*
Diabetes	187	134(72,266)
HBP+AS	1503	188(88,400)*
HBP+DM	225	140(64,288)
AS+DM	224	156(80,357)*
HBP+AS+DM	649	168(77,359)*
AMI	1040	236(106,473)*
CVA	673	171(80,353)*
CKD	37	489±109-914*

Notes: Values are expressed as median ± SD. A p-value less than 0.05 was taken to indicate significance. * $p < 0.05$.

Table 4 linear regression between Lp (a) and various factors and diseases after log10 conversion

Factor	Uncorrected		Corrected for gender and age	
	β	P	β	P
SEX♂1♀2	0.004	0.726	0.004	0.723
AGE(YEAR)	0.101	0.000	0.101	0.000
SMOKENO0YES1	0.033	0.006	0.042	0.001
DRINKNO0YES1	0.003	0.768	0.12	0.324
AS(NO:0YES1)	0.109	0.000	0.086	0.000
DMNO0YES1	-0.001	0.931	-0.015	0.193
AMINO0YES1	0.125	0.000	0.119	0.000
CVAINO0YES1	0.035	0.002	0.016	0.180
HBPNO0YES1	0.050	0.000	0.015	0.216
CKDNO0YES1	0.123	0.000	0.015	0.001

Table 5 Correlation between Lp (a) and blood lipid indexes and EGFR

	TG	Apo-B	Apo-A1	CHOL	HDL-C	LDL-C	EGFR
CC	-0.033	0.179	0.042	0.132	0.143	0.143	0.142
95%CI	-0.083 ~-0.017	0.130 0.227	-0.008 0.092	0.082 0.181	0.094 0.192	0.094 0.192	-0.195~0.491
P	0.197	0.0001	0.102	0.0001	0.0001	0.001	0.402

Table 6 logistic regression analysis of influencing factors of As

factor	P	OR	95% CI
Lp(a)(mg/L)	0.0014	1.0007	1.0003 ~ 1.0012
HBPno0yes1	<0.0001	2.0196	1.5771 ~ 2.5864
Ageyear	<0.0001	1.0613	1.0502 ~ 1.0725
Smokeno0yes1	0.0447	1.3411	1.0070 ~ 1.7861
DMno0yes1	<0.0001	3.0375	2.1476 ~ 4.2960
Sex♂1♀2	<0.0001	0.4644	0.3454 ~ 0.6245

Table 7 logistic regression analysis of influencing factors of myocardial infarction

Factor	P	OR	95% CI
Apo-A1(mg/L)	<0.0001	0.3281	0.2251 – 0.4784
LDL-C(mmol/L)	0.0079	1.1507	1.0375 – 1.2762
Lp(a)(mg/L)	<0.0001	1.0012	1.0009 – 1.0015
HBPnoyes1	<0.0001	1.6895	1.3940 – 2.0476
DMnoyes1	<0.0001	1.6985	1.3795 – 2.0914
SexM1F2	<0.0001	0.3027	0.2420 – 0.3787
Ageyear	<0.0001	1.0210	1.133 – 1.0287

Table 8 logistic regression analysis of influencing factors of coronary heart disease and myocardial infarction

	Risk factors	OR	95%CI
ALL	PDA	1.7790	1.5572–2.0066
	AMI	2.1438	1.878–2.4470
PDA population	AMI	1.626	1.3682–1.9325