Pre-Procedural Lpa level and ApoB/ApoA1 Ratio and the Risk for Contrast-Induced Acute Kidney Injury in Patients Undergoing Emergency Percutaneous Coronary Intervention

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Abstract

Background: High serum Lpa level and ApoB/ApoA1 ratio are risk factors for cardiovascular disease and kidney disease, and have been found to be related to the prevalence and prognosis of a variety of kidney diseases. However, it is not clear whether the preoperative serum Lpa level and ApoB/ApoA1 ratio are related to the incidence of CI-AKI after PCI.

Methods: The 931 patients who underwent emergency PCI surgery from July 2018 to July 2020 were included. According to whether the serum creatinine level was higher than the baseline level ($\geq 25\%$ or ≥ 0.5 mg/dL) 48-72 hours after PCI, these participants were divided into CI-AKI group (n=174) and non-CI-AKI group (n=757). We detected the serum Lpa, ApoA1 and ApoB levels of the patients when they were admitted to the hospital, and calculated ApoB/ApoA1. Logistic regression and restricted cubic spline analyses were used to determine the correlation between Lpa concentration and ApoB/ApoA1 ratio and the risk of CI-AKI.

Results: Among 931 patients undergoing emergency PCI, 174 (18.69%) patients developed CI-AKI. The preoperative Lpa level and ApoB/ApoA1 ratio of the CI-AKI group were higher than those of the non-CI-AKI group, and the differences were statistically significant ($P < 0.05$). In each logistic regression model, we found that preoperative serum Lpa level and ApoB / ApoA1 ratio were inversely associated with the incidence of CI-AKI ($P < 0.05$). After adjusting all the risk factors included in this study, restricted cubic spline analyses found that the preoperative Lpa level and the ApoB/ApoA1 ratio were positively correlated with the incidence of CI-AKI within a certain range.

Conclusion: The preoperative high levels of Lpa and the ratio of ApoB/ApoA1 are independent risk factors for the onset of CI-AKI.

1. Introduction

Contrast-induced acute kidney injury (CI-AKI) refers to the acute renal damage caused by the use of contrast agents during angiography or other medical procedures. As a common and serious complication after PCI in patients with coronary heart disease, CI-AKI is an important reversible and temporary cause of hospital-acquired renal failure. According to the patient's risk factors, the type and dose of contrast media, and the type of radiotherapy, the incidence of CI-AKI is from 1.7 to 37.7%[1–3].

The pathogenesis of CI-AKI is not fully understood. The nephrotoxicity of contrast media, renal medullary ischemia and hypoxia, oxidative stress, and inflammation are considered to be related to the development of CI-AKI[4]. Besides, dyslipidemia is also found to be a risk factor for CI-AKI. Dyslipidemia contributes to CI-AKI by causing endothelial cell damage and inducing inflammation[5, 6]. Studies have found that elevated levels of LDL and sdLDL are independently associated with the risk of CI-AKI[7, 8].

High levels of lipoprotein(a) (Lpa) and Apolipoprotein B (ApoB) and low levels of Apolipoprotein A1 (ApoA1) are considered to be risk factors for cardiovascular disease and kidney disease[9–12]. Studies

have found that Lpa in patients with primary nephropathy are significantly negatively correlated with GFR, and ApoB/ApoA1 is positively correlated with the incidence of CKD[11, 13]. Lpa and ApoB can be deposited in the glomerular mesangial area and stimulate the synthesis of extracellular matrix (ECM), leading to glomerular ischemia and hypoxia, which are related to the pathogenesis of CI-AKI. ApoA1 can promote the reverse transport of cholesterol and inhibit the deposition of cholesterol in the glomerulus, which may have a protective effect on the disease of CI-AKI[4, 14]. However, there is no research report on the correlation between serum Lpa level and ApoB/ApoA1 ratio and CI-AKI. Therefore, this study mainly discussed the correlation between preoperative serum Lpa level and ApoB/ApoA1 ratio and CI-AKI.

2. Materials And Methods

2.1. Study population

This retrospective study included 1446 patients who were hospitalized in the Department of Cardiology of Renmin Hospital of Wuhan University from June 2018 to July 2020 and underwent emergency PCI surgery. 515 patients were excluded, of which 71 patients suffered from severe liver or kidney damage, 118 patients suffered from infectious diseases, cardiogenic shock, stroke, valvular heart disease, tumors, etc., 203 patients have incomplete medical history such as drug use, 17 patients have undergone kidney transplantation, 54 patients are missing postoperative creatinine level data, 13 patients were allergic to artificial contrast agents, 39 patients had been exposed to contrast agents in the 7 days before surgery, and 931 patients were eventually enrolled.

2.2. Clinical assessments

After all subjects were admitted to the hospital, their clinical characteristics were recorded, including age, gender, and complications such as hypertension, diabetes, as well as the history of smoking, drinking and drug use.

2.3. Blood sampling and analysis

When the patients were admitted to the hospital, 5 mL of venous blood was collected into the procoagulant tube and 2 mL of venous blood into the EDTA-K2 anticoagulant tube. Then, the coagulation tube was accelerated for 30 minutes at room temperature and centrifuged at 3,500 rpm for 10 minutes to separate the serum and stored it in the refrigerator at -80°C for later use. We used Siemens Advia 2400 automatic biochemical analyzer (Erlangen, Germany) and its supporting reagents to determine Lpa, ApoB, ApoA1, TP (total protein), ALB (albumin), urea, Cr (creatinine), eGFR (estimated glomerular filtration rate), UA(uric acid), Glu(glucose), total cholesterol(TC), triglyceride (TG), high-density lipoprotein cholesterol (HDL-C), low density lipoprotein cholesterol (LDL-C) and hs-CRP (high-sensitivity C-reactive protein). SysmexCA-7000 system (Kobe, Japan) was used to detect red blood cell count (RBC) and hemoglobin(Hb) concentration. The estimated glomerular filtration rate (eGFR) was calculated using a CKD-EPI 2009SCr equation.

2.4. Percutaneous coronary intervention and drug therapy

PCI includes balloon dilation and/or stent placement of infarct-related blood vessels. The operation is performed by the surgeon in accordance with the interventional procedures. All patients took aspirin 300 mg, clopidogrel 300 mg or ticagrelor 180 mg before operation, and long-term oral aspirin 100 mg (once a day), clopidogrel 75 mg (once a day), or ticagrelor 90 mg (2 times a day) after the operation. Statins, β -receptor blockers, nitrate drugs and angiotensin-converting enzyme inhibitors were routinely used without contraindications. CI-AKI is defined as a contrast agent for acute kidney injury that occurs within 48–72 hours after administration, with a 25% increase in serum creatinine (sCr) or 0.5 mg/dL (44.2 μ mol/L) as the diagnostic criteria. Normal saline (0.9%) was administered intravenously at a rate of 1 ml/kg/h for 12 hours after exposure to the contrast agent.

2.5 Statistical methods

Statistical analysis was performed using SPSS23.0 and R 3.5.2. Age, TP and ALB were the quantitative data, which obeyed the normal distribution and were expressed as mean $\pm$ standard deviation. The overall mean was compared by two independent sample *t* test; ApoB / ApoA1 ratio, Lpa, RBC, Hb, Glu, urea, Cr, UA, TC, TG, LDL-C, HDL-C, hsCRP, contrast volume and contrast exposure time, which do not obeyed the normal distribution and were expressed as interquartile range. The overall mean was compared by Mann-Whitney U test. Chi-square test was used for the comparison of rates, and univariate logistic regression analysis was used to explore the correlation between Lpa and ApoB/ApoA1 and CI-AKI; Multivariate logistic regression was performed for screening risk factors ($P < 0.1$). After adjusting for all confounding factors, restricted cubic spline analyses were used to explore the non-linear correlation between Lpa concentration and ApoB/ApoA1 ratio and the incidence of CI-AKI.

3. Results

3.1 Characteristics of the study population

Among the 931 patients included in the study, 174 patients developed CI-AKI(18.69%). As listed in Table 1, compared with non-CI-AKI group, age, Lpa, ApoB,, urea, Cr, UA and hsCRP levels as well as ApoB/ApoA1 ratio in CI-AKI group are higher, Hb and eGFR are lower, and Diuretics medication rate is higher($P < 0.05$).

Table 1
Basic clinical and procedural characteristics of the CI-AKI and non-CI-AKI groups

characteristics	CI-AKI group (n = 174)	non-CI-AKI group (n = 757)	t/ χ^2	P value
Clinical variables				
Age(years)	65.48 ± 11.29	62.21 ± 11.29	-3.442	0.001
Male(n, %)	129(74.14)	601(79.39)	2.307	0.129
Smoking (n, %)	48(27.59)	222(29.33)	0.208	0.648
Drinking (n, %)	22(12.64)	105(13.87)	0.181	0.671
Hypertension (n, %)	119(68.39)	474(62.62)	2.041	0.153
Diabetes mellitus(n, %)	48(27.59)	188(24.83)	0.566	0.452
LVEF < 40% (n, %)	29(16.67)	100(13.21)	1.416	0.234
Laboratory variables				
Lpa(mg/L)	223.50(110.50, 457.75)	132.00(71.00, 300.50)	-4.367	< 0.001
ApoA1(g/L)	1.18(1.02,1.37)	1.20(1.08,1.36)	-0.853	0.394
ApoB(g/L)	0.84(0.66,1.03)	0.78(0.63,0.93)	-3.083	0.002
ApoB/ApoA1 ratio	0.71(0.55, 0.90)	0.64(0.51,0.78)	-3.365	0.001
RBC(10^{12} /L)	4.45(3.83, 4.85)	4.47(4.06, 4.85)	-1.091	0.275
Hb(g/L)	135.00(119.25, 147.00)	139.00(125.00, 150.00)	-2.589	0.010
TP(U/L)	65.18 ± 5.95	64.70 ± 5.94	-0.945	0.345
ALB(U/L)	39.71 ± 4.30	40.16 ± 3.94	1.346	0.179
urea (mmol/L)	6.11(4.93, 7.50)	5.60(4.56, 6.74)	-3.427	0.001
Cr(μ mol/L)	77.50(65.00, 94.25)	70.00(60.00, 84.00)	-4.132	< 0.001
eGFR(mL/min)	82.68(64.48, 97.65)	93.60(81.43, 103.25)	-5.697	< 0.001

Continuous data were presented as mean ± SD or median (interquartile range, IQR) and compared using Student's independent t-test or Mann-Whitney U test, while categorical data were presented as percentage (%) and compared using Chi-Square test. LVEF, left ventricular ejection fraction; Lpa, lipoprotein a; ApoA1, apolipoproteins A1; ApoB, apolipoproteins B; RBC, red blood cell; Hb, hemoglobin; TP, Total protein; ALB, albumin; Cr, creatinine; eGFR, estimated glomerular filtration rate (mL/min/1.73 m²); UA, uric acid; Glu, glucose; TC, total cholesterol; TG, triglyceride; HDL-C, high-density lipoprotein cholesterol; LDL-C, low-density lipoprotein cholesterol; hs-CRP, high-sensitivity C-reactive protein; ACEI/ARB, angiotensin-converting enzyme inhibitor/angiotensin receptor blocker.

characteristics	CI-AKI group (n = 174)	non-CI-AKI group (n = 757)	<i>t</i> / χ^2	<i>P</i> value
UA ($\mu\text{mol/L}$)	387.50(323.50, 487.25)	375.00(311.50, 447.00)	-2.002	0.045
Glu(mmol/L)	5.92(4.94, 7.77)	5.71(4.82, 7.18)	-1.204	0.228
TC (mmol/L)	4.20(3.21, 4.99)	4.02(3.30, 4.75)	-1.674	0.094
TG (mmol/L)	1.43(0.96, 1.99)	1.32(0.91, 1.93)	-1.227	0.220
HDL-C(mmol/L)	0.93(0.81, 1.20)	0.98(0.82, 1.14)	-0.369	0.712
LDL-C(mmol/L)	2.41(1.67, 3.18)	2.29(1.74, 2.96)	-1.183	0.237
LDL-C/HDL-C ratio	2.41(1.77,3.32)	2.38(1.74,3.04)	-1.391	0.164
hsCRP(mg/L)	5.09(1.59,18.66)	2.28(0.45, 8.83)	-4.804	< 0.001
Medication(n,%)				
Statin	160(91.95)	667(88.11)	2.106	0.147
ACEI/ARB	117(67.24)	523(69.09)	0.225	0.636
β -bloker	140(80.46)	583(77.01)	0.968	0.325
Calcium channel blocker	16(9.20)	56(7.40)	0.641	0.423
Diuretics	37(21.26)	101(13.34)	7.033	0.008
Procedural characteristic				
Contrast volume (mL)	129.00(120.00,146.25)	130.00(120.00,142.00)	-0.370	0.712
Contrast exposure time (min)	56.00(49.50,65.00)	56.00(48.00,64.00)	-0.890	0.374
Multivessel disease(n,%)	77(44.25)	328(43.33)	0.049	0.825
Continuous data were presented as mean $\pm$ SD or median (interquartile range, IQR) and compared using Student's independent t-test or Mann-Whitney U test, while categorical data were presented as percentage (%) and compared using Chi-Square test. LVEF, left ventricular ejection fraction; Lpa, lipoprotein a; ApoA1, apolipoproteins A1; ApoB, apolipoproteins B; RBC, red blood cell; Hb, hemoglobin; TP, Total protein; ALB, albumin; Cr, creatinine; eGFR, estimated glomerular filtration rate (mL/min/1.73 m ²); UA, uric acid; Glu, glucose; TC, total cholesterol; TG, triglyceride; HDL-C, high-density lipoprotein cholesterol; LDL-C, low-density lipoprotein cholesterol; hs-CRP, high-sensitivity C-reactive protein; ACEI/ARB, angiotensin-converting enzyme inhibitor/angiotensin receptor blocker.				

3.2 Associations between the prevalence of CI-AKI and serum Lpa level and ApoB/ApoA1 ratio

In order to analyze the correlation between the prevalence of CI-AKI and serum Lpa level and ApoB/ApoA1 ratio, we divided patients into Lpa quartiles and calculated the odds ratios (ORs) of CI-AKI risk, taking

patients in the first Lpa quartiles as reference (Table 2); similarly, divide the patients into ApoB/ApoA1 ratio quartiles and calculate the ORs of the risk of CI-AKI, taking patients the first ApoB/ApoA1 ratio quartile as reference (Table 3). The multivariate adjusted odds ratio of the risk of CI-AKI indicates that there were positive correlations between the risk of CI-AKI and the preoperative serum Lpa level and the ApoB/ApoA1 ratio.

Table 2
Association of the incidence of CI-AKI with preoperative serum Lpa level in patients who underwent emergency PCI.

Lpa quartile	n	Conc range, mg/L	OR(95%CI)		
			Model 1	Model 2	Model 3
Quartile 1 (low)	233	< 75	Reference	Reference	Reference
Quartile 2	233	75–143	1.156(0.682–1.961)	1.120(0.658–1.908)	1.025(0.571–1.841)
Quartile 3	233	143–328	1.849(1.127–3.032)	1.822(1.108–2.996)	1.838(1.066–3.169)
Quartile 4 (high)	232	≥328	2.360(1.456–3.826)	2.241(1.377–3.645)	1.929(1.117–3.332)
P for trend			< 0.001	< 0.001	0.003
Model 1 no adjustment.					
Model 2 adjusted for age, gender.					
Model 3 adjusted for age, gender, smoking, drinking, hypertension, diabetes, LVEF < 40%, RBC, Hb, TP, ALB, urea, Cr, UA, eGFR, Glu, TC, TG, HDL-C, LDL-C, hsCRP, Statins, ACEI/ARB, β-blockers, calcium channel blockers, diuretics, contrast volume, contrast exposure time and multivessel disease.					

Table 3

Association of the incidence of CI-AKI with preoperative ApoB/ApoA1 ratio in patients who underwent emergency PCI.

ApoB/A1 quartile	n	Ratio range	OR(95%CI)		
			Model 1	Model 2	Model 3
Quartile 1 (low)	234	< 0.52	Reference	Reference	Reference
Quartile 2	233	0.53–0.65	1.325(0.810–2.166)	1.342(0.817–2.203)	1.469(0.859–2.512)
Quartile 3	234	0.66–0.79	1.000(0.598–1.673)	1.077(0.641–1.811)	1.199(0.675–2.129)
Quartile 4 (high)	231	≥0.79	2.182(1.371–3.473)	2.537(1.577–4.082)	2.687(1.527–4.729)
P for trend			0.003	< 0.001	0.002
Model 1 no adjustment.					
Model 2 adjusted for age, gender.					
Model 3 adjusted for age, gender, smoking, drinking, hypertension, diabetes, LVEF < 40%, RBC, Hb, TP, ALB, urea, Cr, UA, eGFR, Glu, TC, TG, HDL-C, LDL-C, hsCRP, Statins, ACEI/ARB, β-blockers, calcium channel blockers, diuretics, contrast volume, contrast exposure time and multivessel disease.					

3.3 Preoperative Lpa level and ApoB / ApoA1 ratio and CI-AKI

Restricted cubic spline analyses found that after adjusting for all confounding factors included in this study, when the Lpa level was less than 500 mg/L, the prevalence of CI-AKI was positively correlated with Lpa; When the Lpa level was greater than 500 mg/L, there was no correlation between the prevalence of CI-AKI and the Lpa level. When ApoB/ApoA1 ratio was less than 0.625, the prevalence of CI-AKI was not correlated with ApoB/ApoA1 ratio; when ApoB/ApoA1 ratio was greater than 0.625, the prevalence of CI-AKI was positively correlated with ApoB/ApoA1 ratio. As shown in Fig. 1.

Restricted cubic spline model of the odds ratios of prevalence of CI-AKI with preoperative serum Lpa level (Fig. 1a) and ApoB/ApoA1 ratio (Fig. 1b) in patients with PCI. The gray area represents the 95% confidence interval. Adjust age, gender, smoking, drinking, hypertension, diabetes, LVEF < 40%, RBC, Hb, TP, ALB, urea, Cr, UA, eGFR, Glu, TC, TG, HDL-C, LDL-C, hsCRP, Statins, ACEI/ARB, β-blockers, calcium channel blockers, diuretics, contrast volume, contrast exposure time and multivessel disease.

4. Discussion

To our knowledge, this is the first report that preoperatively higher Lpa level and higher ApoB/ApoA1 ratio in patients undergoing emergency PCI surgery are independent predictors of CI-AKI. In this study, we found that patients with CI-AKI had higher preoperative serum Lpa level and ApoB/ApoA1 ratio than

patients without CI-AKI. After adjusting for various confounding factors we found that the preoperative serum Lpa level and ApoB/ApoA1 ratio were independently related to the incidence of CI-AKI, indicating that the preoperative serum Lpa level and ApoB/ApoA1 ratio are the potential risk factors of CI-AKI.

With the increasing prevalence of coronary heart disease and the advancement of medical technology, PCI surgery is becoming more and more popular. CI-AKI, as a common and serious postoperative complication, has received high attention. CI-AKI can increase the risk of renal failure and death, as well as increase the length of hospitalization and increase the cost of hospitalization[15]. Since there is no standard effective treatment for contrast-induced acute kidney injury, identifying high-risk patients with CI-AKI and preventing the occurrence of contrast-induced nephropathy are still the focus of treatment. Sensitive and easily available clinical indicators are of great value for identifying CI-AKI patients and effectively preventing the occurrence of the disease.

Lp(a), ApoA-1 and ApoB are common clinical serum lipid indicators. They are not only related to the prevalence of coronary heart disease, but also related to kidney damage[9–12]. With the introduction of the concept of "lipid nephrotoxicity", the study of blood lipids and kidney disease has become more and more in-depth[16]. Regarding the correlation between blood lipids and the pathogenesis of CI-AKI, there have been reports in the literature that LDL and sdLDL are related to the pathogenesis of CI-AKI[7, 8]. At the same time, the effective prevention of lipid-lowering drugs for CI-AKI also indirectly clarifies the correlation between blood lipids and the disease of CI-AKI[17, 18]. In this study, we found that Lpa level and ApoB/ApoA1 ratio are independent risk factors for CI-AKI. Restricted cubic spline analyses found that after adjusting for all confounding factors included in this study, the preoperative Lpa level and the ApoB/ApoA1 ratio were positively correlated with the incidence of CI-AKI within a certain range.

Lp(a) is a polymer complex in the circulatory system, composed of low-density lipoprotein (LDL)-like particles and apolipoprotein (apo) (a). It is a low-density lipoprotein-like protein with an apo(a) group[19, 20]. The plasma Lp(a) concentration is mostly determined by the LPA gene[21]. Clarke et al. found that the mutation of the LPA gene was the strongest cardiovascular genetic risk factor among 2,100 candidate genes for cardiovascular disease[22]. In addition to cardiovascular disease, Lpa also involved in the pathogenesis of kidney disease[23]. Studies have found that Lpa levels increased in patients with nephrotic syndrome, and low concentrations of Lpa stimulate the growth of mesangial cells, while high concentrations of Lpa have anti-proliferative or toxic effects[24–27]. Besides, Lp(a) can induce the activation of ROM in glomeruli by a pathway that is sensitive to inhibition of protein kinase C and elevation of intracellular CAMP levels, leading to glomerular damage[28]. These effects may cause kidney damage and lead to the incidence of CI-AKI.

ApoB is one of the main components of LDL-C, which can promote the phagocytosis of oxidized LDL by macrophages and monocytes, thereby transforming into foam cells and inducing atherosclerosis[29]. Lipoproteins containing larger ApoB, such as (VLDL, IDL), can also increase the risk of atherosclerotic thrombosis by inhibiting the fibrinolytic system and stimulating the production of inflammatory factors and inflammatory response[30, 31]. ApoA1 exists in HDL-C and maintains the structure of HDL. It is a

cofactor for lecithin cholesterol acyltransferase (LCAT). It can reversely transport cholesterol to the liver and inhibit the oxidation of low-density lipoproteins, and has the effect of preventing atherosclerosis[32].

ApoB/A1 was found to be related to the onset of kidney disease. Cross-sectional analysis of two independent population-based independent studies showed that higher serum apoA1 levels were associated with lower prevalence of chronic kidney disease (CKD), while lower serum apoA1 levels and higher apoB/A1 ratio was associated with a higher incidence of CKD[11]. Moreover, apoB/A1 was related to the mortality of dialysis patients[33]. The influence of ApoB and ApoA1 on the pathogenesis of CI-AKI is not fully understood, and may be related to the deposition of lipids in the glomeruli and the occurrence of inflammation. ApoB can be swallowed by glomerular macrophages, and then deposited in the mesangial area of the glomerulus, leading to glomerular hypertrophy and increasing transforming growth as well as the level of TGF- β , which in turn causes kidney damage[34–37]. ApoA1 can inhibit the oxidation of LDL-C, promote the reverse transport and metabolism of TC, prevent the deposition of cholesterol in the glomerulus and the recruitment of inflammatory cells in the kidney, and inhibit the occurrence of inflammation, which may have a protective effect on CI-AKI[38, 39]. The apoB/A1 ratio can reflect the balance between the risk factors and protective factors of kidney injury. Compared with apoA1 and ApoB, it is considered as a better predictor of the occurrence and progression of kidney injury.

This study has certain limitations. First, there are racial and regional differences in the level of Lp(a), but the population included in this study is from central China. The applicability of the conclusions of this study in other regions and populations needs to be further verified; Second, although our analysis shows that Lpa and apoB/apoA1 ratio are associated with the prevalence of CI-AKI, we cannot predict the causality. Third, the lack of data on the long-term prognosis and survival rate of patients has led to the failure of this study to evaluate the relationship between Lpa level and ApoB/ApoA1 ratio and the long-term prognosis of patients.

5. Conclusion

In short, in patients receiving emergency PCI treatment, high Lpa levels and high ApoB/ApoA1 ratios before surgery are independent risk factors for CI-AKI. Our findings indicate that the preoperative Lpa level and ApoB/ApoA1 ratio can help identify high-risk patients with CI-AKI, which moderately provided clinician guidance for planning preventive measures and therapeutic strategies.

Declarations

Ethics approval and consent to participate:

The authors are accountable for all aspects of the work to ensure that issues related to the accuracy or completeness of any part of the work are properly investigated and resolved. This study was approved by the Medical Ethics Review Committee of Renmin Hospital of Wuhan University, China. All participants

signed an informed consent form in accordance with the policies of the Renmin Hospital of Wuhan University Ethics Committee.

Consent for publication:

Not applicable

Availability of data and materials:

The datasets generated during and analysed during the current study are not publicly available due to privacy or ethical restrictions but are available from the corresponding author on reasonable request.

Competing interests:

The authors have declared no conflicts of interest.

Funding:

The National Natural Science Foundation of China (81772265) have contributed to analysis and interpretation of data.

Authors' contributions:

(I) Conception and design: J Tao, W Dai, Y Li;

(II) Administrative support: Y Li;

(III) Provision of study materials or patients: CL Ye, Q Yao, M Zhou;

(IV) Collection and assembly of data: Q Yao, M Zhou, CL Ye;

(V) Data analysis and interpretation: J Tao;

(VI) Manuscript writing: All authors;

(VII) Final approval of manuscript: All authors.

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Figures

Fig 1a

Fig 1b

Figure 1

Restricted cubic spline analyses found that after adjusting for all confounding factors included in this study, when the Lpa level was less than 500 mg/L, the prevalence of CI-AKI was positively correlated with Lpa; When the Lpa level was greater than 500 mg/L, there was no correlation between the prevalence of CI-AKI and the Lpa level. When ApoB/ApoA1 ratio was less than 0.625, the prevalence of CI-AKI was not correlated with ApoB/ApoA1 ratio; when ApoB/ApoA1 ratio was greater than 0.625, the prevalence of CI-AKI was positively correlated with ApoB/ApoA1 ratio.