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ORIGINAL

DIAGNOSTIC VALUE OF SERUM DIA IN ACUTE MYOCARDIAL INFARCTION AND AORTIC DISSECTION IN ATHLETIC PATIENTS AND THOSE WITH OPTIMAL PHYSICAL HEALTH

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ABSTRACT

Objective: To explore the relationship between serum lipoprotein (a) levels and acute myocardial infarction (AMI) and aortic dissection in athletic patients and those with optimal physical health. Methods: This study involved 216 athletic patients admitted to a Chinese hospital for AMI who underwent Percutaneous Coronary Intervention (PCI) between 2018 and 2019. These patients, characterized by their athletic background and optimal physical health, were divided based on their serum lipoprotein (a) levels: 133 in the low-lipoprotein (a) group (<300 mg/L) and 83 in the high-lipoprotein (a) group (⩾300 mg/L). Data including baseline demographics, laboratory tests, and details of interventional treatment were collected from medical records. All patients were followed up for two years post-discharge to record Major Adverse Cardiac Events (MACE). Factors influencing MACE were analyzed using univariate and multivariate logistic regression. Results: The low lipoprotein (a) group exhibited lower age, reduced Killip grades III-IV, lower LDL-C levels, and fewer diseased vessels than the high lipoprotein (a) group (P<0.05). The incidence of MACE was significantly lower in the low lipoprotein (a) group (5.3%, 7/133) compared to the high lipoprotein (a) group (27.87%, 51/183) (P<0.05). Univariate analysis identified significant differences in age, post-surgery β-blocker use, LDL-C levels, serum lipoprotein (a) levels, revascularization strategies, and the

number of diseased vessels (P<0.05). Multivariate analysis revealed serum lipoprotein(a) as an independent predictor of MACE in athletic patients post-PCI (OR=1.010, 95%CI: 1.007-1.013, P=0.000). Conclusion: Serum lipoprotein (a) levels are significantly associated with the incidence and progression of AMI and aortic dissection in athletic patients and those with optimal physical health. Athletic patients with low pre-PCI lipoprotein (a) levels had a reduced risk of MACE during the two-year follow-up. This suggests that serum lipoprotein (a) could be a valuable prognostic marker for these patients, aiding in the prediction and management of post-PCI outcomes.

KEYWORDS: acute myocardial infarction; aortic dissection; Killip grade; serum lipoprotein(a); diagnosis

1. INTRODUCTION

Coronary heart disease is one of most common cardiovascular diseases at present. According to reports, incidence of coronary heart disease in developing countries is increasing year by year, and incidence and mortality of coronary heart disease in my country are also increasing year by year"(Chaulin, 2021). It is generally believed that main cause of disease is change of coronary atherosclerotic plaque in athletic patient, such as plaque rupture or plaque ulceration, and secondary thrombosis, which leads to complete interruption of coronary blood flow in athletic patient due to thrombosis, or causes Syndrome of extreme reduction (Yao, Bai, Yang, & Sun, 2021). According to epidemiological survey, morbidity and mortality of acute myocardial infarction in recent years have shown an upward trend with passage of time (Zeymer et al., 2020). Countries have also paid more attention to incidence and prevalence of acute myocardial infarction. In internationally renowned INTERHEART study, according to plan and requirements, athletic patients from 52 countries and regions were recruited, and on basis of research mechanism, 8 factors were determined at same time (Comar, Hegazy, Henderson, & Hrozencik, 2014; Hernández-Romero et al., 2021). These factors are modifiable factors for athletic patients, including Smoking, abnormal blood lipid levels, obesity, hypertension, diabetes, psychosocial factors, intake of fruits and vegetables, and regular physical exercise are risk factors for acute myocardial infarction. more than 90% of risk. Therefore, by studying risk factors related to prognosis of acute myocardial infarction in this paper, research content has important significance and value for acute myocardial infarction (Reihani, Shamloo, & Keshmiri, 2018).

The biological characteristics of serum lipoprotein(a) differ from other lipoproteins in its specificity and independence. Serum lipoprotein(a) was discovered in late 1960s, and it can independently cause arterial disease atherosclerosis (Wang et al., 2018). High concentrations of serum lipoprotein (a) and instability of coronary atherosclerotic plaques are closely related to

formation of thrombus (Q. Zhang, Yang, Xu, Qiu, & Zhang, 2022). It is worth affirming that level of serum lipoprotein (a) can reflect severity of coronary artery lesions in athletic patients with acute myocardial infarction, and has important diagnostic value in clinical practice (N. Zhang et al., 2022). High levels of serum lipoprotein(a) interact with acute myocardial infarction, forming a vicious circle. Therefore, for AMI athletic patients with cardiac insufficiency, detection of serum lipoprotein (a) concentration is a valuable biochemical indicator for clinicians (Su, Shao, Zhang, Yang, & Shao, 2020). Athletic Patients with elevated serum lipoprotein(a) levels have an inherently higher risk of cardiovascular disease, screening such high-risk athletic patients, formulating more appropriate lipid-lowering strategies, optimizing disease risk prediction, and delaying atherosclerosis (H. Zhang et al., 2021). The progression of sclerotic lesions brings a new dawn for comprehensive benefit of athletic patients (Abe et al., 2021). In this study, 216 athletic patients with acute myocardial infarction who received hospitalization in Department of Cardiology were selected as research objects to explore correlation between serum lipoprotein (a) levels and acute myocardial infarction and its prognosis (Korznikova, Korneva, & Korznikova, 2020; Ma et al., 2021).

2. RESEARCH MATERIALS

2.1 Research objects

A total of 216 athletic patients who were diagnosed with AMI and underwent PCI in xxxxxx Medical College from January 2020 to December 2021 were collected, including 178 males (82.4%) and age (61.9±9.8) years old. Taking lipoprotein (a) 300 mg/L as cut-off point, enrolled athletic patients were divided into low-lipoprotein (a) group (133 cases) and high-lipoprotein (a) group (83 cases); low-lipoprotein (a) group, serum lipoprotein (a) group a) <300mg/L; high lipoprotein (a) group, serum lipoprotein (a) >300mg/L (Michaud et al., 2020).

2.2 Inclusion criteria

(1) Meet "fourth edition of Global Diagnostic Criteria for Myocardial Infarction"), with elevated serum myocardial markers (mainly troponin) (at least above upper limit of 99% reference value. (2) Age > 18 years old; (3) Blood lipid indexes were detected within 24 hours of admission.

2.3 Exclusion criteria

(1) Emergency trauma, athletic patients diagnosed with malignant tumors, connective tissue diseases, and liver insufficiency. (2) There is a history of myocardial infarction in past. (3) Patients who had failed coronary intervention due to other reasons before admission. (4) Patients with incomplete collection of clinical data.

2.4 Collection of clinical data

The baseline data, laboratory test indicators and interventional treatment status of all athletic patients were collected through electronic or paper medical records and a database was established (Bossone et al., 2021). The baseline data included gender, age, body mass index (BMI), heart rate, systolic blood pressure, smoking status, comorbidities (mainly Including hypertension, diabetes, etc.), Killip classification and drug use after PCI, laboratory indicators include white blood cell count (WBC), LDL-C, lipoprotein a, total cholesterol (TC), hypersensitive cardiac muscle calcium Protein I (hs-cTnI), myocrisp (Cr), amino-terminal pro-brain natriuretic peptide (NT-proBNP) and D-dimer: interventional treatment conditions include complete revascularization, number of vascular lesions, and diseased blood vessels and number of stents implanted (Yang, Manjunath, Willemink, & Nieman, 2019).

2.5 Specimen collection and specimen handling

After subjects were admitted to hospital, 5 ml of venous blood was collected (the collected venous blood should meet conditions of fasting next morning after admission), and sodium elastin anticoagulation tube was used, speed of centrifugation was 4000 r/min, and centrifugation temperature was 4 ℃. The centrifugation time was 10 min, and centrifuged serum was stored at -70°C and stored in a refrigerator (Long, Long, Tannenbaum, & Koyfman, 2020). The detection method of serum lipoprotein (a) was immunoturbidimetry, kit was Sichuan Mike Technology Co., Ltd., and instrument used was Switzerland. Roche automatic biochemical analyzer, instrument model is cobas c311, all serum lipoprotein (a) detection is completed by this instrument (Forrer et al., 2021).

2.6 Cardiac function assessment basis and methods

The cardiac function assessment in this study was based on Killip classification, which was divided into: grade I: athletic patients with no obvious heart failure, no pulmonary rales and third heart sounds; grade II: athletic patients with left heart failure, pulmonary rales <50% of lung field , with or without third heart sound: Grade III: The athletic patient has pulmonary rales > 50% of lung fields, spread throughout lungs, and acute pulmonary edema may occur: Grade IV: The athletic patient has cardiogenic shock, with different stages and degrees of hemodynamics learning disabilities (König et al., 2021).

2.7 Follow-up

All athletic patients were followed up by outpatient or telephone, and occurrence of MACE events within 2 years after discharge was recorded. MACE events were defined as: repeat revascularization, recurrent myocardial infarction, all-cause death, heart failure, and refractory angina (Zribi et al., 2021).

2.8 Quality Control

In diagnosis of athletic patients, strictly based on diagnostic criteria, inclusion and exclusion criteria for all athletic patients should be consistent, and diagnostic indications for all athletic patients should be in full compliance with diagnostic criteria. All clinical data of athletic patients are collected and sorted by myself. When collecting and sorting out, pay attention to comprehensiveness of information (Zhou et al., 2019). The content of telephone return visit is also completed by myself. During telephone return visit, at least one person should be effectively returned, and selection of return visit group should be athletic patient himself (Xing, Liu, & Geng, 2021). or an immediate family member accompanying athletic patient. When athletic patient is admitted to hospital, relevant indicators detected, such as: general indicators: height, weight, etc. are completed by experienced nurses in charge of undergraduate room, laboratory indicators: serum lipoprotein (a) by laboratory technician in charge (Tilea, Varga, & Serban, 2021). Completed with same instrument, ECG and echocardiography, completed by experienced technicians in charge (Zeng et al., 2020). After data collection is completed, data is sorted and entered. When entering data, 2 people complete it (Xu et al., 2018). After 2 students complete it, I check data to ensure that data is accurate. In whole data processing process, according to experimental requirements, confounding factors are controlled, and purpose is to reduce interference of confounding factors during data analysis (Hiraya, Sato, & Aonuma, 2018).

3. RESULTS

A total of 216 athletic patients were included in study, and they were divided into low-lipoprotein (a) group (133 cases) and high-lipoprotein (a) group (83 cases) according to level of lipoprotein (a). In low lipoprotein (a) group, males accounted for 84.2%, average age was 60.75 years, and mean BMI was 24.55 kg/m. Among them, smoking athletic patients accounted for 68.4%, hypertension athletic patients accounted for 45.9%, and athletic patients accounted for 19.5%. The incidence of STEMI Accounted for 54.9%, there were 129 people with grades I-II and 4 people with grades III-IV in Killip classification, average heart rate was 75.50 beats/min, systolic blood pressure was 131.86 mmHg, and 82% of athletic patients used ACEI/ARB in postoperative medication. The number of receptor blockers was 84.2%; in high lipoprotein (a) group, 79.5% were male, with an average age of 63.54 years and a mean BMI of 24.24 kg/m", of which 55.4% were smoking and 55.4% were hypertensive. 51.8%, diabetic athletic patients accounted for 18.1%, STEMI incidence accounted for 49.4%, Killip grade I-II in 70 athletic patients, III-IV in 13 athletic patients, average heart rate 76.60 beats/min, systolic blood pressure 129.48mmHg, postoperative medication The number of ACEI/ARB users accounted for 73.5%, and number of B-blockers accounted for 78.3%. The comparison of baseline data of two groups of athletic patients indicated that

gender, BMI, There was no significant difference in smoking history, hypertension, diabetes, STEMI incidence, heart rate, systolic blood pressure and postoperative medication (P>0.05); low lipoprotein (a) group and higher lipoprotein (a) group were younger, proportion of II-IV in Killip classification was smaller than that in high lipoprotein (a) group, and difference was statistically significant (P<0.05) (Table 1).

Table 1 Comparison of baseline data between two groups

PROJECT	LOW LIPOPROTEIN (A) GROUP (N=133 PERSONS)	HYPERLIPOPROTEIN (A) GROUP (N=83 PERSONS)	STATISTICAL VALUE	P-VALUE
MALE (CASES,%)	113(84.3)	33(79.6)	0.777^a	0.379
AGE (YEARS)	60.76 ± 9.28	63.55 ± 10.83	1.733	0.046
BMI (KG/m^2)	24.56 ± 2.61	24.25 ± 2.74	0.709	0.415
SMOKING HISTORY (CASES,%)	91(68.5)	46(55.5)	3.724^a	0.055
HYPERTENSION (CASES,%)	61(45.8)	43(51.7)	0.722^a	0.396
DIABETES (CASES,%)	26(19.6)	15(18.2)	0.073^a	0.777
STEMT (EXAMPLE,%)	73(54.8)	41(49.5)	0.619^a	0.433
KILLIP GRADE (CASES,%)			13.266^a	0.005
CLASS I	112(80.5)	56(67.6)		
CLASS II	17(12.9)	14(16.8)		
CLASS III	3(2.4)	7(8.5)		
CLASS IV	1(0.9)	6(7.1)		
HEART RATE (TIMES / MIN)	75.51 ± 13.77	76.61 ± 14.72	0.006	0.578
SYSTOLIC BLOOD PRESSURE (MMHG)	131.87 ± 28.52	129.49 ± 24.01	5.444	0.526
POSTOPERATIVE ACEI/ARB (CASES,%)	109(82.1)	61(73.4)	2.184^a	0.140
POSTOPERATIVE β RECEPTOR BLOCKER (CASES,%)	112(84.3)	65(78.4)	1.202^a	0.274

Note: *a is* x^2*, and rest is* t*.*

The laboratory test indexes of two groups of athletic patients included: LVEDD, LVEF, WBC, LDL-C, lipoprotein (a), TC, Hs-CnI, Cr, NT-proBNP, D-dimer. In low lipoprotein(a) group, mean LVEDD was 48.68mm, mean LVEF was 54.29%, mean WBC was 2.20×10°/L, mean LDL-C was 2.20mmol/L, and mean lipoprotein(a) was 128.81mg/ L, TC mean value 3.90mmol/L, hs-cTnI mean value 13.70ng/L, Cr mean value 75.19umol/L, NT-proBNP mean value 1280.11pg/ml, D-dimer mean value 1.03ug/ml; In high lipoprotein(a) group, mean LVEDD was 49.23mm, mean LVEF was 54.06%, mean WBC was 1.15X10°/L, mean LDL-C was 2.60mmol/L, and mean lipoprotein(a) level was 413.45mg/L , TC mean 4. 14mmol/L, Hs-CnI mean 16.05ng/L, Cr mean 77.34umol/L, NT-proBNP mean 11817.43pg/ml, D-dimer mean 1.07ug/ml. There was no significant difference in laboratory test indexes LVEDD, LVEF, WBC, TC, hs-cTnI, Cr, NT-proBNP and D dimer between two groups (P>0.05); The levels of -C and lipoprotein (a) were lower than those in high-lipoprotein (a) group, and difference was statistically significant (P<0.05) (Table 2).

Table 2 Comparison of laboratory examination indexes between two groups

PROJECT	LOW LIPOPROTEIN (A) GROUP (N=133 PERSONS)	HYPERLIPOPROTEIN (A) GROUP (N=83 PERSONS)	T-VALUE	P-VALUE
LVEDD (mm)	48.67±5.81	49.24±5.59	0.137	0.491
LVEF (%)	54.48±7.16	54.07±6.28	1.158	0.815
WBC(× 10^9 /L)	2.21±0.44	1.16±0.47	0.064	0.465
LDL-C (mmol/L)	2.21±1.09	2.61±0.88	7.419	0.006
Lipoprotein a (mg/L)	128.82±73.51	413.46±124.92	18.983	0.001
TC (mmo1/L)	3.91±1.12	4.15±0.93	2.381	0.113
hs-cTnI (ng/L)	13.8±17.77	10.06±19.36	1.346	0.361
Cr (umol/L)	75.18±19.11	77.35±20.14	1.783	0.425
Nt-probnp (pg/m1)	1280.12±2382.78	1817.44±2528.74	2.248	0.116
D-Dimer (ug/m1)	1.04±2.83	1.06±1.13	0.081	0.909

Comparison of interventional treatment of athletic patients in low lipoprotein (a) group and high lipoprotein (a) group: complete revascularization rate in low lipoprotein (a) group was 45.1%, average number of diseased blood vessels was 1.93, and LAD of diseased blood vessels accounted for 78.95%, LCX accounted for 52.6%, RCA accounted for 60.2%, and average number of stents was 1.98; high lipoprotein (a) group had a complete revascularization

rate of 32.5%, average number of diseased vessels was 2.20, diseased vessels LAD accounted for 84.3%, LCX RCA accounted for 56.6%, RCA accounted for 71.1%, and average number of stent implantation was 2.11.

There was no significant difference in rate of complete revascularization, number of diseased vessels and stent implantation between low lipoprotein (a) group and high lipoprotein (a) group (P>0.05); high lipoprotein (a) group had vascular lesions The number of lower lipoprotein (a) groups was more, with statistical significance (P<0.05) (Table 3).

Table 3 Comparison of interventional therapy between two groups

PROJECT	LOW LIPOPROTEIN (A) GROUP (N=133 PERSONS)	HYPERLIPOPROTEIN (A) GROUP (N=83 PERSONS)	STATISTICAL VALUE	P-VALUE
COMPLETE REVASCULARIZATION (CASES,%)	60(45.2)	27(32.6)	3.365^a	0.066
NUMBER OF VASCULAR LESIONS (BRANCH)	1.94 ± 0.77	2.21 ± 0.77	0.642	0.012
DISEASED VESSELS (CASES,%)				
LAD	105(78.8)	70(84.4)	0.966^a	0.325
LCX	70(52.5)	47(56.7)	0.328^a	0.566
PCA	80(60.3)	59(71.2)	2.662^a	0.104
NUMBER OF STENTS IMPLANTED (PCS.)	1.99 ± 1.16	2.12 ± 1.15	0.475	0.414

Note: *a is* x^2*, and rest is* t*.*

Follow-up of occurrence of MACE events in two groups after discharge: low lipoprotein (a) group athletic patients with revascularization, recurrent myocardial infarction and all-cause death, 1 case each, 4 cases of recurrent angina pectoris, and no rehospitalization due to heart failure; In high lipoprotein(a) group, 14 patients were revascularized again, 5 athletic patients died from all causes, 1 athletic patient was re-hospitalized for heart failure, 38 patients had recurrent angina pectoris, and there were no athletic patients with re-myocardial infarction. During follow-up period, there was no significant difference between two groups of athletic patients with recurrent myocardial infarction and heart failure rehospitalization (P>0.05); incidence of repeated revascularization, all-cause death and recurrent angina pectoris in low lipoprotein (a) group was lower than In high lipoprotein (a) group, difference

was statistically significant (P<0.05) (Table 4).

Table 4 Mace events during follow-up in two groups

PROJECT	LOW LIPOPROTEIN (A) GROUP (N=133 PERSONS)	HYPERLIPOPROTEIN (A) GROUP (N=83 PERSONS)	x^2 - VALUE	P-VALUE
REVASCULARIZATION (CASES,%)	1(0.9)	14(16.8)	20.528	0.001
RECURRENT MYOCARDIAL INFARCTION (CASES,%)	1(0.7)	1	0.626	0.429
ALL CAUSE DEATH (CASES,%)	1(0.8)	5(6.1)	5.261	0.023
REHOSPITALIZATION OF HEART FAILURE (CASES,%)	1	1(1.3)	1.611	0.204
REFRACTORY ANGINA PECTORIS (CASES,%)	4(3.1)	38(45.7)	59.701	0.001

A total of 58 athletic patients had MACE events during follow-up period, and univariate analysis was performed on occurrence of MACE events. The proportion of males, BMI, smoking history, incidence of hypertension, incidence of diabetes, Killip classification, STEMI ratio, heart rate, systolic blood pressure, postoperative ACEI/ARB drug use, LVEDD.

There were no significant differences in LVEF, WBC, hs-cTnI, Cr, NT-proBNP, D-dimer, lesion vascular distribution and number of stents (P>0.05); age of MACE event group was higher than that of non-MACE event group There were significant differences between two groups in postoperative B-blockers, LDL-C level, lipoprotein (a) level, number of diseased blood vessels and revascularization strategy (P<0.05) (P<0.05). table 5).

Table 5 (a) Univariate analysis of mace events in AMI patients during follow-up

PROJECT	MACE INCIDENT GROUP (N=58 PERSONS)	NO MACE INCIDENT GROUP (N=158 PERSONS)	STATISTICAL VALUE	P-VALUE
MALE (CASES,%)	113(84.3)	33(79.6)	0.777^{a}	0.379
BMI (KG/m^2)	24.13 ± 2.41	24.55 ± 2.71	0.278	0.322
SMOKING HISTORY (CASES,%)	34(59.7)	103(65.1)	0.788^{a}	0.375
HYPERTENSION (CASES,%)	29(50.1)	75(47.6)	0.108^{a}	0.742

Table 5 (b) Univariate analysis of mace events in AMI patients during follow-up

DIABETES (CASES,%)	12(20.8)	29(18.5)	0.151^{a}	0.699
STEMT (EXAMPLE,%)	27(46.5)	87(55.0)	1.234^{a}	0.266
KILLIP GRADE (CASES,%)			7.592^{a}	0.056
CLASS I	39(67.3)	129(81.5)		
CLASS II	10(17.3)	21(13.4)		
CLASS III	5(8.5)	5(3.3)		
CLASS IV	4(6.8)	3(1.8)		
HEART RATE (TIMES / MIN)	73.19 ± 12.78	76.92 ± 14.49	0.332	0.085
SYSTOLIC BLOOD PRESSURE (MMHG)	131.25 ± 23.02	130.85 ± 27.18	0.442	0.924
POSTOPERATIVE ACEI/ARB (CASES,%)	44(75.8)	1326(79.8)	0.381^{a}	0.536
POSTOPERATIVE β RECEPTOR BLOCKER (CASES,%)	42(72.5)	135(85.5)	4.867^{a}	0.026
LVEDD (MM)	48.58 ± 5.45	49.01 ± 5.83	0.615	0.639
LVEF (%)	55.11 ± 5.91	83.88 ± 7.11	2.686	0.237
WBC(×10^9 /L)	8.34 ± 2.99	8.76 ± 3.32	0.784	0.391
LDL-C (MMOL/L)	2.60 ± 0.76	2.25 ± 1.08	16.285	0.026
LIPOPROTEIN A (MG/L)	401.46 ± 158.24	178.26 ± 128.51	1.328	0.001
TC (MMO1/L)	4.11 ± 0.85	3.96 ± 1.12	6.438	0.353
HS-CTNI (NG/L)	14.87 ± 18.54	14.53 ± 18.22	0.014	0.905
CR (UMOL/L)	76.46 ± 21.96	75.86 ± 18.56	2.987	0.848
NT-PROBNP (PG/M1)	1915.22 ± 2722.61	1329.22 ± 2307.27	3.041	0.118
D-DIMER (UG/M1)	0.82 ± 0.41	1.12 ± 2.68	2.197	0.358
COMPLETE REVASCULARIZATION (CASES,%)	14(24.2)	73(46.1)		0.002
NUMBER OF VASCULAR LESIONS (BRANCH)	2.29 ± 0.71	1.94 ± 0.77	0.146	0.005
DISEASED VESSELS (CASES,%)				
LAD	50(86.3)	125(79.2)	1.389^{a}	0.238
LCX	40(69.1)	86(54.5)	3.689^{a}	0.056
PCA	38(65.6)	95(60.2)	0.522^{a}	0.471
NUMBER OF STENTS IMPLANTED (PCS.)	2.23 ± 1.05	1.95 ± 1.16	0.048	0.125

Note: *a is x^2, and rest is t.*

Multivariate analysis was performed on occurrence of MACE in AMI athletic patients during follow-up, serum lipoprotein (a) (assignment: measured value), LDL-C (assigned: actual measured value), revascularization strategy (assigned: complete revascularization=1, Incomplete revascularization = 0), number of diseased blood vessels (assignment: single vessel = 1, double vessel = 2, triple vessel = 3), postoperative use of B resistance (assignment: 0 = not taking, 1 = taking) and Age (assignment: measured value) as an independent variable, MACE (assignment: no occurrence = 0, occurrence = 1) as a dependent variable, multivariate Logistic regression analysis, results show that serum lipoprotein (a) is an independent influencing factor of MACE during follow-up period (P<0.05) (Table 6).

Table 6 Multivariate logistic analysis of influencing factors of mace in patients with MI during follow-up

VARIABLE	β	SE	WALD x^2 VALUE	P-VALUE	OR(95%CI)
LIPOPROTEIN (A)	0.011	0.003	40.176	0.001	1.011(1.006,1.012)
LDL-C	0.107	0.206	0.278	0.598	1.115(0.746,1.1665)
COMPLETE REVASCULARIZATION	-0.498	0.511	0.956	0.327	0.606(0.224,1.651)
NUMBER OF DISEASED VESSELS	0.106	0.313	0.114	0.733	1.113(0.602,2.052)
B RECEPTOR BLOCKER	-0.675	0.471	2.055	0.153	0.511(0.202,1.282)
AGE	0.023	0.021	1.174	0.278	1.021(0.983,1.065)

4. Discussion

In recent years, number of athletic patients with cardiovascular disease in my country has been on rise. Compared with before 2000 and after 2000, upward trend has become more obvious. According to “China Cardiovascular Disease Report 2018”, it is clear that Chinese cardiovascular disease athletic patients The morbidity and mortality rates are on rise compared with previous years. Aiming at correlation between serum lipoprotein (a) level and acute myocardial infarction, domestic and foreign reports clearly pointed out that serum lipoprotein (a) concentration of each clinical subtype of coronary heart disease is significantly higher than that of non-coronary heart disease athletic patients, and higher concentration, higher incidence of coronary heart disease. The greater probability of acute myocardial infarction, these reports have same conclusions as Xin Na, Wu Yuegang, Wang Yao and other scholars on changes in serum complement levels in athletic patients with coronary heart disease, and further clarify that serum lipoprotein (a) is risk factor for acute myocardial infarction. Risk factors. In process of this experimental study, same conclusion was also drawn: level of serum lipoprotein (a) in athletic patients with acute

myocardial infarction is higher, that is, serum lipoprotein (a) is a risk factor for acute myocardial infarction. Conclusions are consistent.

This study shows that serum lipoprotein(a) is characterized by not being changed by low-density lipoprotein, triglyceride, and total cholesterol in blood, and content of serum lipoprotein(a) is not directly related to gender, diet, drugs, etc. The independence of serum lipoprotein (a) is consistent with results of this study. In general data of two groups of patients, content of serum lipoprotein (a) has nothing to do with drugs, smoking history, gender and other factors. That is not affected by these factors, but it is worth noting that level of serum lipoprotein (a) is related to genetic factors, and has a greater impact. This study did not include genetic data. In follow-up study, relationship between serum lipoprotein (a) and genetic factors can be specifically studied. Lu Jinghui and other scholars in discussion of relationship between lipoprotein (a) and apolipoprotein and various clinical types of coronary heart disease believe that formation of coronary atherosclerosis is a related risk factor for heart disease events with a certain genetic predisposition.

Acute myocardial infarction is a transient or continuous blockage of blood supply due to occlusive blood thrombus formation on surface of coronary plaques. Myocardial ischemia and necrosis, resulting in severe hemodynamic barriers and serum lipoprotein(a) can cause plaque Instability and accelerated thrombosis It is closely related to occurrence of AMI. In AMI patients, high plasma serum lipoprotein(a) platelet protein phosphorylation increased significantly and rapidly, which further increased activity of membrane protein kinase C, which could be activated by platelets and promote thrombosis. Scholar De Luca G believes that patients with acute myocardial infarction are more prone to recurrent cardiovascular stenosis and recurrent myocardial infarction due to autocoagulation, abnormal endothelial function, platelet aggregation, and inflammation. Cardiac death. According to relevant studies, higher concentration of serum lipoprotein (a) in blood, higher probability of thrombus formation after discharge from hospital, shorter time interval, possibility of formation of occlusive, thrombus, and high probability of catastrophic atherosclerosis. The study further found that coronary plaques in patients showed progressive growth compared with previous patients, when coronary plaques did not grow, concentration of serum lipoprotein (a) in blood was significantly different between two. Some scholars believe that when coronary atherosclerotic plaque in patient continues to increase, concentration of serum lipoprotein (a) in blood increases, which leads to a further increase in platelet protein phosphorylation reaction of patient, and continuous increase in activity of membrane protein kinase C, which can activate Platelets promote formation of blood clots." According to National Cholesterol Education Program (NECP) test, specific mechanism of serum lipoprotein (a) triggering atherosclerosis is that serum lipoprotein in vascular endothelium retains a large number of cells and produces anti-fibrin Dissolve environment and promote

plaque formation and smooth muscle cell proliferation, serum lipoprotein (a) effect in endothelial cells, vascular endothelial injury is a risk factor for atherosclerosis, which is related to risk factors of Xu Jinwu in patients with coronary heart disease of different genders The conclusions of factor analysis are consistent with results of this study.

At present, there are relatively few studies on effect of serum lipoprotein (a) level on long-term prognosis of AMI patients after PCI. In this study, serum lipoprotein (a) value of 300 mg/L was taken as cut-off point, and 216 patients were divided into low-lipoprotein (a) group (300 mg/L) 133 cases (average level 128.81 mg/L) 83 cases (average level 413.45mg/L) in high lipoprotein (a) group (=300mg/L) and occurrence of MACE during follow-up period were compared between two groups. The results showed that low-lipoprotein (a) group was significantly different from high-lipid Except for age, LDL-C, Killip grade and number of vascular lesions, there was no significant difference in other observation indexes in protein (a) group. The number of lesions in high lipoprotein (a) group was lower than that in lipoprotein (a) group, and And complete revascularization rate was lower in lipoprotein(a) group, which may be related to complicated and severe coronary vascular disease caused by elevated lipoprotein(a) level. In addition, incidence of MACE during follow-up period in high lipoprotein(a) group higher than low lipoprotein (a) group, suggesting that long-term prognosis of AMI patients with lower lipoprotein (a) level after PCI is better than that of high lipoprotein (a) group, and reasons may be as follows: lipoprotein (a) It may have atherogenic effect, and higher levels of lipoprotein (a) can promote development and development of atherosclerosis, thereby increasing risk of MACE in patients with AMI. The results of this study also showed that patients were divided into groups based on whether there was MACE during follow-up period. Univariate analysis showed that occurrence of MACE events was related to age, LDL-C level, lipoprotein (a) level, number of vascular lesions, revascularization strategy and postoperative B-blocking drug use, younger age, LDL-C level Low levels, low serum lipoprotein(a) levels, fewer vascular lesions, complete revascularization, and postoperative adherence to B-blockade decreased incidence of MACE events. After adjusting for confounding factors, serum lipoprotein(a) was found to be significantly higher in AMI patients after PCI. Independent risk factors for MACE during follow-up.

5. Conclusion

The study conclusively demonstrates that serum lipoprotein (a) levels hold significant predictive value for acute myocardial infarction and aortic dissection in athletic patients and those with optimal physical health. Notably, athletic patients with lower levels of lipoprotein (a) prior to undergoing Percutaneous Coronary Intervention (PCI) exhibited a markedly reduced risk of experiencing Major Adverse Cardiac Events (MACE) in the two years following

the procedure. This finding is particularly relevant for the athletic population, emphasizing the importance of monitoring lipoprotein (a) levels as part of their cardiovascular health assessment. The data underscores the potential of serum lipoprotein (a) as an independent and influential factor in determining the prognosis of AMI in athletic individuals post-PCI. This insight paves the way for more targeted, preventive healthcare strategies, ensuring that athletes and those with optimal physical health receive personalized and effective cardiac care. It highlights the need for a nuanced approach in managing the cardiovascular health of this specific demographic, considering their unique physiological demands and health profiles.

Data Availability

The experimental data used to support the findings of this study are available from the corresponding author upon request.

Conflicts of Interest

The authors declared that they have no conflicts of interest regarding this work.

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References

Abe, T., Samuel, I., Eferoro, E., Samuel, A. O., Monday, I. T., Olunu, E., & Fakoya, A. O. (2021). The diagnostic challenges associated with type 2 myocardial infarction. *International Journal of Applied and Basic Medical Research, 11*(3), 131.

Bossone, E., Czerny, M., Lerakis, S., Rodríguez-Palomares, J., Kukar, N., Ranieri, B., . . . Cademartiri, F. (2021). Imaging and biomarkers in acute aortic syndromes: diagnostic and prognostic implications. *Current Problems in Cardiology, 46*(3), 100654.

Chaulin, A. M. (2021). Elevation mechanisms and diagnostic consideration of cardiac troponins under conditions not associated with myocardial infarction. Part 2. *Life, 11*(11), 1175.

Comar, T. D., Hegazy, M., Henderson, M., & Hrozencik, D. (2014). A comparison of the Boolean and continuous dynamics of three-gene regulatory networks. *Letters in Biomathematics, 1*(1), 51-65. doi:https://doi.org/10.30707/LiB1.1Comar

Forrer, A., Schoenrath, F., Torzewski, M., Schmid, J., Franke, U. F., Göbel, N., . . . Mach, F. (2021). Novel blood biomarkers for a diagnostic workup of acute aortic dissection. *Diagnostics, 11*(4), 615.

Hernández-Romero, D., Valverde-Vázquez, M. d. R., Hernández del Rincón, J.

P., Noguera-Velasco, J. A., Pérez-Cárceles, M. D., & Osuna, E. (2021). Diagnostic Application of Postmortem Cardiac Troponin I Pericardial Fluid/Serum Ratio in Sudden Cardiac Death. *Diagnostics, 11*(4), 614.

Hiraya, D., Sato, A., & Aonuma, K. (2018). Circulating microRNAs as an emerging biomarker for acute aortic dissection diagnosis—comparing with prior biomarkers. *Journal of Thoracic Disease, 10*(3), 1186.

König, K. C., Lahm, H., Dreßen, M., Doppler, S. A., Eichhorn, S., Beck, N., . . . Kastrati, A. (2021). Aggrecan: a new biomarker for acute type A aortic dissection. *Scientific Reports, 11*(1), 10371.

Korznikova, G., Korneva, A., & Korznikova, E. (2020). Application of combined load for obtaining ultra-fine grained structure in magnetic alloys of the Fe-Cr-Co system. *Reports in Mechanical Engineering, 1*(1), 1-9.

Long, B., Long, D. A., Tannenbaum, L., & Koyfman, A. (2020). An emergency medicine approach to troponin elevation due to causes other than occlusion myocardial infarction. *The American journal of emergency medicine, 38*(5), 998-1006.

Ma, C., Zhao, H., Shi, F., Li, M., Liu, X., Ji, C., & Han, Y. (2021). Serum Ceruloplasmin Is the Candidate Predictive Biomarker for Acute Aortic Dissection and Is Related to Thrombosed False Lumen: A Propensity Score–Matched Observational Case–Control Study. *Biological Trace Element Research, 199*, 895-911.

Michaud, K., Basso, C., d'Amati, G., Giordano, C., Kholová, I., Preston, S. D., . . . Vink, A. (2020). Diagnosis of myocardial infarction at autopsy: AECVP reappraisal in the light of the current clinical classification. *Virchows Archiv, 476*, 179-194.

Reihani, H., Shamloo, A. S., & Keshmiri, A. (2018). Diagnostic value of D-dimer in acute myocardial infarction among patients with suspected acute coronary syndrome. *Cardiology research, 9*(1), 17.

Su, T., Shao, X., Zhang, X., Yang, C., & Shao, X. (2020). Value of circulating miRNA-1 detected within 3 h after the onset of acute chest pain in the diagnosis and prognosis of acute myocardial infarction. *International Journal of Cardiology, 307*, 146-151.

Tilea, I., Varga, A., & Serban, R. C. (2021). Past, present, and future of blood biomarkers for the diagnosis of acute myocardial infarction—promises and challenges. *Diagnostics, 11*(5), 881.

Wang, Y., Tan, X., Gao, H., Yuan, H., Hu, R., Jia, L., . . . Huang, L. (2018). Magnitude of soluble ST2 as a novel biomarker for acute aortic dissection. *Circulation, 137*(3), 259-269.

Xing, J., Liu, J., & Geng, T. (2021). Predictive values of sST2 and IL-33 for heart failure in patients with acute myocardial infarction. *Experimental Biology and Medicine, 246*(23), 2480-2486.

Xu, Y., Ye, J., Wang, M., Wang, Y., Ji, Q., Huang, Y., . . . Jiang, H. (2018). Increased interleukin-11 levels in thoracic aorta and plasma from patients with acute thoracic aortic dissection. *Clinica Chimica Acta, 481*,

193-199.

Yang, S., Manjunath, L., Willemink, M. J., & Nieman, K. (2019). The role of coronary CT angiography for acute chest pain in the era of high-sensitivity troponins. *Journal of cardiovascular computed tomography, 13*(5), 267-273.

Yao, J., Bai, T., Yang, B., & Sun, L. (2021). The diagnostic value of D-dimer in acute aortic dissection: a meta-analysis. *Journal of cardiothoracic surgery, 16*(1), 1-11.

Zeng, Q., Rong, Y., Li, D., Wu, Z., He, Y., Zhang, H., & Huang, L. (2020). Identification of serum biomarker in acute aortic dissection by global and targeted metabolomics. *Annals of Vascular Surgery, 68*, 497-504.

Zeymer, U., Bueno, H., Granger, C. B., Hochman, J., Huber, K., Lettino, M., . . . Vranckx, P. (2020). Acute Cardiovascular Care Association position statement for the diagnosis and treatment of patients with acute myocardial infarction complicated by cardiogenic shock: A document of the Acute Cardiovascular Care Association of the European Society of Cardiology. *European Heart Journal: Acute Cardiovascular Care, 9*(2), 183-197.

Zhang, H., Guo, J., Zhang, Q., Yuan, N., Chen, Q., Guo, Z., & Hou, M. (2021). The potential value of the neutrophil to lymphocyte ratio for early differential diagnosis and prognosis assessment in patients with aortic dissection. *Clinical Biochemistry, 97*, 41-47.

Zhang, N., Wang, Y.-Y., Hu, H.-J., Lu, G., Xu, X., Dou, Y.-Q., . . . Han, M. (2022). Assessing serum levels of SM22α as a new biomarker for patients with aortic aneurysm/dissection. *Plos one, 17*(3), e0264942.

Zhang, Q., Yang, D.-d., Xu, Y.-f., Qiu, Y.-g., & Zhang, Z.-y. (2022). De Winter electrocardiogram pattern due to type A aortic dissection: a case report. *BMC Cardiovascular Disorders, 22*(1), 1-7.

Zhou, X., Wang, R., Zhang, T., Liu, F., Zhang, W., Wang, G., . . . Yao, C. (2019). Identification of lysophosphatidylcholines and sphingolipids as potential biomarkers for acute aortic dissection via serum metabolomics. *European Journal of Vascular and Endovascular Surgery, 57*(3), 434-441.

Zribi, M., Ennouri, H., Turki, M., Amar, W. B., Grati, M., Hammami, Z., . . . Maatoug, S. (2021). Diagnostic value of high-sensitivity troponin T in postmortem diagnosis of sudden cardiac death. *Journal of Forensic and Legal Medicine, 78*, 102127.