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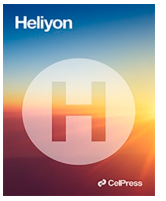

Research article

Different levels of lipids, Hb1Ac and cytokines among patients with coronary artery disease

Xue Jiang, Xin-ying Guo, Jie Zhang, Guo-yong Zhang, Zheng Ma, Cai-xia Guo *

Cardiovascular Center, Beijing Tongren Hospital, Capital Medical University, No. 1 Dongjiaomin Lane, Dongcheng District, Beijing, 100730, PR China

A R T I C L E I N F O

Keywords:
Dyslipidemia
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A B S T R A C T

Background: Different risk factors are responsible for the occurrence of coronary artery disease (CAD). Among these, the main factors are dyslipidemia, dysglycaemia, and endothelial inflammation. The aim of the study was to analyze the levels of lipids, glucose, and cytokine in patients with different coronary heart diseases.

Methods: A total of 2147 patients diagnosed with coronary atherosclerosis, stable angina, unstable angina, acute non-ST-segment elevation infarction (NSTEMI) and acute ST-segment-elevation myocardial infarction (STEMI) at the Cardiovascular Center of Beijing Tongren Hospital from February 2022 to April 2023. The data were gathered from the medical record system. Nonparametric Wilcoxon test was used for statistical analysis of continuous variables, and chi-square test was used for statistical analysis of categorical variables among multiple groups.

Results: Compared with coronary atherosclerosis group, acute myocardial infarction group showed a significant increase in IL-6 level ($p < 0.001$). Compared with stable angina group, acute myocardial infarction group showed a significant increase in IL-6 and decrease in INF-γlevels ($p < 0.001$). Compared with unstable angina group, acute myocardial infarction group showed a significant increase in IL-6 level and decrease in IL-17, as well as INF-γlevels ($p < 0.001$). Compared with NSTEMI group, the proportion of younger, males, glycemic-lowering drugs, as well as the levels of TC, LDL-C in STEMI group increased significantly, while the proportion of hypertension, IFG/IGT/DM, hyperlipidemia and Hb1Ac level decreased significantly. STEMI group showed a significant increase in IL-2 and IL-6 levels ($p < 0.05$).

Conclusions: The levels of lipids, Hb1Ac, IL-2 and IL-6 were varying in patients with different stages of coronary heart diseases.The data would contribute to a deeper understanding of the roles of lipids, glucose, and inflammation in the occurrence and development of CAD.

1. Background

Ischemic heart disease remains a huge burden on individuals and society all over the world. Coronary atherosclerosis is the fundamental and critical pathobiological process responsible for driving cardiovascular events, including stable angina, unstable angina, acute non-ST-segment-elevation myocardial infarction (NSTEMI) and acute ST-segment-elevation myocardial infarction (STEMI) [1]. Both genetic and environmental factors contribute to the occurrence and development of coronary atherosclerosis,

* Corresponding author.
E-mail address: cxguo@ccmu.edu.cn (C.-x. Guo).

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including hypertension, hyperlipidemia and hyperglycemia [1–3].

Dyslipidemia including high total cholesterol (TC), triglyceride (TG), low-density lipoprotein cholesterol (LDL-C), lipoproteion(a) [Lp(a)] and low high-density lipoprotein cholesterol (HDL-C), is an important step in the occurrence and development of coronary atherosclerosis. Lipid levels reduction is the most important steps to prevent progression and plaque rupture of coronary atherosclerosis [1]. Ranging from asymptomatic, stable angina to unstable angina, acute myocardial infarction, the lipid levels are different. In addition, DM is the major risk factor responsible for the development of coronary atherosclerosis. Previous studies have shown that in non-diabetic patients with myocardial infarction or stable angina, glycosylated hemoglobin (HbA1c) level correlate with coronary artery disease (CAD) severity. No correlation was found between admission glucose or fasting glucose levels and CAD severity [4]. Further research is needed to analyze the levels of lipids and glucose levels in different disease states of coronary heart disease.

Inflammation plays a crucial role in the development and progression of coronary atherosclerosis. Research has shown that concentrations of IL-6 are associated with CAD characteristics, indicating that inflammation may contribute to pathways in CAD pathophysiology [5]. Vascular endothelial activation and dysfunction is the first and most important step in atherosclerosis development [6]. Simultaneous transcriptomic analysis of psoriatic lesion skin and atherosclerotic plaque found that INF-γ and TNF-α synergistically drive endothelial inflammation and endothelial dysfunction [7]. Further research is needed to clarify the levels of cytokine levels in different disease states of CAD.

The aim of the present study was to analyze the levels of lipids, glucose and cytokine levels in different disease states of CAD, including coronary atherosclerosis, stable angina, unstable angina, NSTEMI and STEMI.

2. Methods

2.1. Study population

The present study was a a single-center retrospective study. Hospitalized patients diagnosed as coronary atherosclerosis, stable angina pectoris and acute coronary syndrome in the cardiovascular center of Beijing Tongren Hospital from February 2022 to April 2023 were included, regardless of age, complications, treatment and whether it was the first event. Refusal to sign informed consent forms, as well as patients with other types of heart disease, severely impaired liver or kidney function, malignancy, autoimmune diseases, active infections, and those taking anti-inflammatory drugs or immunosuppressants were excluded. Coronary atherosclerosis was characterized according to luminal obstructive stenosis between 30 % and 50 % [8]. Stable angina occurs when the myocardial oxygen supply cannot meet the demand [9]. Acute coronary syndrome (ACS) describes the range of myocardial ischemic states that includes unstable angina, NSTEMI, or STEMI [10]. A total of 2147 patients with ischemic heart disease were included in the present study. According to the principles mentioned in the Declaration of Helsinki, the ethics committees of Beijing Tongren Hospital approved the research protocol (approval number: TREC2022-KY081). Written informed consent were obtained from all participants (or guardians of participants) in the present study.

2.2. Clinical data collection

The clinical information of enrolled patients were collected through electronic medical record system. The subsequent data were gathered from the medical record system, including age, sex; medical history of hypertension, IFG/IGT/DM and hyperlipidemia; TC, TG, HDL-C, LDL-C, Lp(a), glucose, high-sensitivity C-reactive protein (hsCRP), Hb1Ac, interleukin-1 beta (IL-1β), Interleukin-10 (IL-10), interleukin-12p70 (IL-12p70), Interleukin-17 (IL-17), Interleukin-2 (IL-2), Interleukin-4 (IL-4), Interleukin-5 (IL-5), Interleukin-6 (IL-6), Interleukin-8 (IL-8), Interferon-alpha (INF-α), interferon-gamma (INF-γ), tumor necrosis factor-alpha (TNF-α).

2.3. Laboratory analyses

Fasting blood specimens from 1814 patients were collected using EDTA anticoagulation tubes at admission. Applying standard Beijing Tongren Hospital assays on fresh samples to measure TC, TG, HDL-C, LDL-C, Lp(a), FPG, hsCRP, Hb1Ac, IL-1β, IL-10, IL-12p70, IL-17, IL-2, IL-4, IL-5, IL-6, IL-8, INF-α, INF-γ, TNF-α. Beckman Coulter (Beckman Coulter Ireland, Inc, USA) and Test kit for detecting the levels of lipid panel, glucose, etc [Menelaus Medical Equipment (Shanghai) Co., Ltd, China]. Tosoh Automated Glycohemoglobin Analyzer HLC-723G8 and G8 Elution Buffer HSI No.1(s) were used to detect the level of Hb1Ac. A reagent kit based on the principle of multiple microsphere flow cytometry immunofluorescence luminescence for detecting cytokines (Qingdao Ruisikaier Biotechnology Co., Ltd, China).

2.4. Statistical analyses

Continuous variables in clinical characteristic data were presented as medians (interquartile ranges). Categorical variables in clinical characteristic data were presented as percentages. Nonparametric Wilcoxon test was used for statistical analysis of continuous variables, and chi-square test was used for statistical analysis of categorical variables among multiple groups. Two-sided $p < 0.05$ was considered statistically significant. The above statistical analyses were conducted using SPSS Statistics version 21.0 (IBM Corp., Armonk, NY, USA).

3. Results

3.1. Clinical characteristics

Table 1 shows the clinical characteristics of four groups, including coronary atherosclerosis, stable angina, unstable angina and acute myocardial infarction groups. Compared to coronary atherosclerosis group, stable angina group tended to be a higher proportion of males, smoker, drinker, blood pressure-lowering drugs, hypertension,IFG/IGT/DM, hyperlipidemia, a lower TC, HDL-C, LDL-C and a higher Hb1Ac ($p < 0.05$). Compared to coronary atherosclerosis group, unstable angina group tended to be a higher proportion of males, smoker, antiplatelet agents, lipid-lowering drugs, blood pressure-lowering drugs, glycemic-lowering drugs, IFG/IGT/DM, hyperlipidemia, a lower TC, HDL-C, LDL-C, and a higher glucose, Hb1Ac ($p < 0.001$). Compared to coronary atherosclerosis group, acute myocardial infarction group tended to be younger, a higher proportion of males, smoker, antiplatelet agents, blood pressure-lowering drugs, glycemic-lowering drugs, a lower proportion of hyperlipidemia, a lower HDL-C and a higher glucose, hsCRP, Hb1Ac ($p < 0.001$). Compared to stable angina group, unstable angina group tended to be a higher proportion of antiplatelet agents ($p < 0.001$). Compared to stable angina group, acute myocardial infarction group tended to be younger, a higher proportion of males, smoker, antiplatelet agents, a lower proportion of hypertension, hyperlipidemia, a lower HDL-C and a higher TC, TG, LDL-C, glucose and hsCRP ($p < 0.05$).

Table 2 shows the 12 cytokine levels of four groups, including coronary atherosclerosis, stable angina, unstable angina and acute myocardial infarction groups. Compared with coronary atherosclerosis group, acute myocardial infarction group showed a significant increase in IL-6 level ($p < 0.001$). Compared with stable angina group, acute myocardial infarction group showed a significant increase in IL-6 and decrease in INF-γlevels ($p < 0.001$). Compared with unstable angina group, acute myocardial infarction group showed a significant increase in IL-6 level and decrease in IL-17, as well as INF-γlevels ($p < 0.001$).

Table 3 shows the clinical characteristics of NSTEMI group and STEMI group. Compared to NSTEMI group, the proportion of

Table 1
Clinical characteristics of coronary atherosclerosis, stable angina, unstable angina and acute myocardial infarction.

Characteristics	coronary atherosclerosis (n = 285)	stable angina (n = 269)	unstable angina (n = 944)	acute myocardial infarction (n = 649)	*p*-value
Demographic characteristics					
Age, years, median (IQR), years	64.0(58.0,70.0)	66.0(59.0,71.0)	65.0(58.0,71.0)	61.0(52.0,70.0)	<0.001[a,b,c]
Male, n (%)	109(38.2)	163(60.6)	590(62.5)	510(78.6)	<0.001[a,b,c,d,f]
Smoker, n (%)	85(29.8)	126(46.8)	445(47.1)	429(66.1)	<0.001[a,b,c,d,f]
Drinker, n (%)	60(21.1)	85(31.6)	257(27.2)	185(28.5)	0.035[f]
Discharge medication, n (%)					
Antiplatelet agents	241(84.6)	235(87.4)	923(97.8)	617(95.1)	<0.001[a,b,c,d,e]
Lipid-lowering Drugs	271(95.1)	262(97.4)	926(98.1)	614(94.6)	0.001[c,d]
Blood pressure-lowering Drugs	195(68.4)	234(87.0)\	866(91.7)	595(91.7)	<0.001[a,d,f]
Glycemic-lowering Drugs	41(14.4)	61(22.7)	267(28.3)	191(29.4)	<0.001[a,d]
Medical history, n (%)					
Hypertension	194(68.1)	213(79.2)	716(75.8)	403(62.1)	<0.001[b,c,f]
IFG/IGT/DM	76(26.7)	101(37.5)	421(44.6)	221(34.1)	<0.001[c,d,f]
Hyperlipidemia	138(48.4)	171(63.6)	557(59.0)	242(37.3)	<0.001[a,b,c,d,f]
Laboratory test					
TC(mM), median (IQR)	4.6(3.9,5.4)	3.9(3.4,4.7)	4.1(3.4,4.9)	4.4(3.7,5.2)	<0.001[b,c,d,f]
TG (mM), median (IQR)	1.4(0.9,1.8)	1.2(0.9,1.8)	1.3(0.9,1.8)	1.4(1.0,2.1)	0.004[b,c,]
HDL-C (mM), median (IQR)	1.3(1.1,1.6)	1.2(1.0,1.4)	1.2(1.0,1.4)	1.0(0.9,1.2)	<0.001[a,b,c,d,f]
LDL-C (mM), median (IQR)	2.6(2.0,3.3)	2.1(1.7,2.7)	2.3(1.7,3.0)	2.7(2.0,3.4)	<0.001[b,c,d,f]
Lp(a)(mg/dl), median (IQR)	13.6(5.9,28.5)	14.7(6.0,30.7)	13.5(5.6,33.5)	16.2(6.9,32.6)	0.074
glucose(mM), median (IQR)	5.6(5.1,6.5)	5.8(5.2,6.9)	5.9(5.2,7.4)	6.5(5.6,8.2)	<0.001[a,b,c,d]
hsCRP (mg/L), median (IQR)	1.1(0.6,2.7)	1.3(0.6,3.1)	1.3(0.6,3.2)	4.4(1.8,13.9)	<0.001[a,b,c]
Hb1Ac(%), median (IQR)	6.0(5.6,6.6)	6.2(5.8,6.9)	6.3(5.8,7.3)	6.1(5.7,7.1)	<0.001[a,d,f]

Continuous data are presented as median (interquartile range, IQR), and categorical variables are presented as %.
TC: total cholesterol; TG: triglyceride; HDL-C: high-density lipoprotein cholesterol; LDL-C: low-density lipoprotein cholesterol; Lp(a): lipoproteion(a); glucose: fasting plasma glucose; hsCRP: high-sensitivity C-reactive protein; Hb1Ac: glycosylated hemoglobin.

[a] Acute myocardial infarction vs. coronary atherosclerosis.
[b] Acute myocardial infarction vs. stable angina.
[c] Acute myocardial infarction vs.unstable angina.
[d] Unstable angina vs. coronary atherosclerosis.
[e] Unstable angina vs. stable angina.
[f] Stable angina vs. coronary atherosclerosis.

Table 2
12 cytokine levels of coronary atherosclerosis, stable angina, unstable angina and acute myocardial infarction.

Characteristics	coronary atherosclerosis (n = 102)	stable angina (n = 89)	unstable angina (n = 429)	acute myocardial infarction (n = 420)	*p*-value
IL-1β (pg/ml), median (IQR)	2.4(0,6.5)	2.8(0,5.8)	1.9(0,5.9)	0.8(0,4.5)	0.056
IL-10 (pg/ml), median (IQR)	0.9(0.8,1.3)	1.0(0.8,1.3)	1.0(0.8,1.4)	1.1(0.8,1.4)	0.120
IL-12p70 (pg/ml), median (IQR)	1.3(0.9,1.9)	1.3(1.0,1.9)	1.3(0.9,1.8)	1.2(0.9,1.8)	0.769
IL-17 (pg/ml), median (IQR)	2.3(1.8,3.4)	2.5(1.8,3.7)	2.5(1.7,4.1)	2.2(1.6,3.2)	0.024[c]
IL-2 (pg/ml), median (IQR)	1.3(0.7,2.2)	1.2(0.5,2.4)	1.5(0.5,2.5)	1.3(0.6,2.1)	0.967
IL-4 (pg/ml), median (IQR)	0.9(0.6,1.4)	0.8(0.6,1.3)	0.9(0.5,1.5)	0.9(0.5,1.3)	0.356
IL-5 (pg/ml), median (IQR)	2.9(1.0,5.2)	3.4(1.4,5.6)	3.3(1.6,5.6)	2.7(1.4,4.8)	0.149
IL-6 (pg/ml), median (IQR)	2.6(0.6,5.2)	2.7(1.1,5.4)	3.1(1.1,6.5)	14.2(5.7,35.7)	<0.001[a,b,c]
IL-8 (pg/ml), median (IQR)	1.0(0,3.8)	1.6(0.3,4.7)	2.1(0.3,5.0)	1.9(0.2,6.8)	0.199
INF-α (pg/ml), median (IQR)	0.7(0,2.5)	1.0(0,3.6)	0.8(0,2.9)	0.9(0,2.7)	0.533
INF-γ (pg/ml), median (IQR)	2.8(0.3,6.8)	3.6(1.9,7.2)	3.2(0.7,6.5)	2.3(0.2,4.9)	0.001[b,c]
TNF-α (pg/ml), median (IQR)	1.1(0,2.2)	1.0(0,2.1)	0.9(0,2.6)	0.8(0,2.0)	0.151

Continuous data are presented as median (interquartile range, IQR).
IL-1β: interleukin-1 beta; IL-10: Interleukin-10; IL-12p70: interleukin-12p70; IL-17: Interleukin-17; IL-2: Interleukin-2; IL-4: Interleukin-4; IL-5: Interleukin-5; IL-6: Interleukin-6; IL-8: Interleukin-8; INF-α: Interferon-alpha; INF-γ: interferon-gamma; TNF-α: tumor necrosis factor-alpha.
d: unstable angina vs. coronary atherosclerosis: e: unstable angina vs. stable angina; f: stable angina vs. coronary atherosclerosis.
[a] Acute myocardial infarction vs. coronary atherosclerosis.
[b] Acute myocardial infarction vs. stable angina.
[c] Acute myocardial infarction vs.unstable angina.

Table 3
Clinical characteristics of non-ST-segment-elevation myocardial infarction (NSTEMI) and ST-segment-elevation myocardial infarction (STEMI).

Characteristics	NSTEMI (n = 283)	STEMI (n = 366)	p-value
Demographic characteristics			
Age, years, median (IQR), years	63.0(54.0,72,0)	59.0(49.0,68,0)	<0.001
Male, n (%)	210(74.2)	300(82.0)	0.017
Smoker, n (%)	182(64.3)	247(67.5)	0.397
Drinker, n (%)	91(32.2)	94(25.7)	0.070
Discharge medication, n (%)			
Antiplatelet agents	269(95.1)	348(95.1)	0.987
Lipid-lowering Drugs	270(95.4)	344(94.0)	0.428
Blood pressure-lowering Drugs	259(91.5)	336(91.8)	0.897
Glycemic-lowering drugs	95(33.6)	96(26.2)	0.042
Medical history, n (%)			
Hypertension	204(72.1)	199(54.4)	<0.001
IFG/IGT/DM	124(43.8)	97(26.5)	<0.001
Hyperlipidemia	129(45.6)	113(30.9)	<0.001
Laboratory test			
TC(mM), median (IQR)	4.3(3.6,5.2)	4.5(3.8,5.3)	0.032
TG (mM), median (IQR)	1.4(1.0,2.0)	1.4(1.0,2.2)	0.772
HDL-C (mM), median (IQR)	1.0(0.9,1.2)	1.0(0.8,1.2)	0.340
LDL-C (mM), median (IQR)	2.5(2.0,3.3)	2.8(2.2,3.4)	0.004
Lp(a) (mg/dl), median (IQR)	16.1(6.3,34.0)	16.4(7.0,32.3)	0.930
glucose (mM), median (IQR)	6.2(5.4,8.4)	6.6(5.8,8.0)	0.069
hsCRP (mg/L), median (IQR)	3.7(1.4,13.7)	4.8(2.2,14.0)	0.058
Hb1Ac (%), median (IQR)	6.3(5.8,7.7)	6.1(5.7,6.8)	0.001

Continuous data are presented as median (interquartile range, IQR), and categorical variables are presented as %.
TC: total cholesterol; TG: triglyceride; HDL-C: high-density lipoprotein cholesterol; LDL-C: low-density lipoprotein cholesterol; Lp(a): lipoproteion(a); glucose: fasting plasma glucose; hsCRP: high-sensitivity C-reactive protein; Hb1Ac: glycosylated hemoglobin.

younger, males, glycemic-lowering drugs, as well as the levels of TC, LDL-C in STEMI group increased significantly, while the proportion of hypertension, IFG/IGT/DM, hyperlipidemia, and Hb1Ac decreased significantly ($p < 0.05$).

Table 4 shows the 12 cytokine levels of NSTEMI group and STEMI group. Compared to acute NSTEMI group, STEMI group showed a significant increase in IL-2 and IL-6 levels ($p < 0.05$).

Table 4
12 cytokine levels of non-ST-segment-elevation myocardial infarction (NSTEMI) and ST-segment-elevation myocardial infarction (STEMI).

Characteristics	NSTEMI (n = 178)	STEMI (n = 242)	p-value
IL-1β (pg/ml), median (IQR)	0.8(0,4.7)	0.8(0,4.2)	0.205
IL-10 (pg/ml), median (IQR)	1.0(0.8,1.5)	1.1(0.9,1.4)	0.977
IL-12p70 (pg/ml), median (IQR)	1.3(0.8,1.9)	1.2(0.9,1.7)	0.491
IL-17 (pg/ml), median (IQR)	2.2(1.7,3.1)	2.2(1.5,3.2)	0.688
IL-2 (pg/ml), median (IQR)	1.1(0.4,2.0)	1.5(0.8,2.2)	0.036
IL-4 (pg/ml), median (IQR)	0.9(0.5,1.3)	0.9(0.5,1.3)	0.718
IL-5 (pg/ml), median (IQR)	3.2(1.6,5.0)	2.5(1.2,4.7)	0.119
IL-6 (pg/ml), median (IQR)	8.8(3.4,23.6)	20.0(9.4,42.3)	<0.001
IL-8 (pg/ml), median (IQR)	1.7(0.1,5.1)	2.1(0.2,8.4)	0.193
INF-α (pg/ml), median (IQR)	1.1(0,2.7)	0.8(0,2.7)	0.334
INF-γ (pg/ml), median (IQR)	2.6(0.2,4.8)	2.1(0.2,4.9)	0.723
TNF-α (pg/ml), median (IQR)	0.9(0,2.2)	0.7(0,1.8)	0.242

Continuous data are presented as median (interquartile range, IQR).
IL-1β: interleukin-1 beta; IL-10: Interleukin-10; IL-12p70: interleukin-12p70; IL-17: Interleukin-17; IL-2: Interleukin-2; IL-4: Interleukin-4; IL-5: Interleukin-5; IL-6: Interleukin-6; IL-8: Interleukin-8; INF-α: Interferon-alpha; INF-γ: interferon-gamma; TNF-α: tumor necrosis factor-alpha.

4. Discussion

We found that there are differences in clinical characteristics, Hb1Ac and lipid and cytokine levels among patients with coronary artery disease. Compared to coronary atherosclerosis group, stable angina group showed a significant decrease in TC, HDL-C, LDL-C and increase in Hb1Ac, unstable angina group showed a significant decrease in TC, HDL-C, LDL-C and increase in glucose, Hb1Ac, acute myocardial infarction group showed a significant decrease in HDL-C and increase in IL-6, glucose, hsCRP, Hb1Aclevel. Compared to stable angina group, unstable angina group tended to be a higher proportion of antiplatelet agents, acute myocardial infarction group showed a significant decrease in HDL-C, INF-γand increase in TC, TG, LDL-C, IL-6, glucose, hsCRP. Compared with unstable angina group, acute myocardial infarction group showed a significant increase in IL-6 level and decrease in IL-17, as well as INF-γlevels. Compared to acute NSTEMI group, STEMI group showed a significant increase in IL-2 and IL-6 levels.

Current evidence confirms that high plasma glucose level is the mediator of poor prognosis of myocardial infarction. Previous studies have shown that patients with elevated blood glucose in patients with STEMI indicate adverse outcome [11,12]. The non-diabetic patients undergoing primary percutaneous coronary intervention for STEMI were included, the results showed that fasting glucose was an independent risk factor for heart failure and left ventricular systolic dysfunction [11]. The study found that elevated admission Hb1Ac level is associated with poor prognosis in nondiabetic patients with STEMI [11,13]. In addition, studies have shown that the higher the plasma glucose level of patients, the lower their left ventricular ejection fraction (LVEF), the more severe their heart failure, and the longer their hospital stay. A study suggests a negative correlation between admission blood glucose and LVEF, as well as a positive correlation with Troponin-I level and length of hospital stay [14]. We observed the differences in Hb1Ac between NSTEMI and STEMI patients, our data showed that the Hb1Ac level of STEMI patients is lower than NSTEMI patients, which is consistent with the results of clinical characteristics, the proportion of IFG/IGT/DM in STEMI patients is lower than that in NSTEMI patients.

High LDL-C and Lp(a) levels form the cornerstone approach of cardiovascular risk [15–17]. In STEMI, a lower LDL-C level was associated with worse outcomes for death during hospitalization, within 30-days and 12-months, but not in NSTEMI [18]. High Lp(a) level is an independent predictor of coronary artery disease [19]. We found that the TC and LDL-C levels of STEMI patients are higher than NSTEMI patients.

Inflammation plays a crucial role in the development and progression of coronary atherosclerosis. We observed the differences in cytokine levels between NSTEMI and STEMI patients, our data showed that the IL-2 and IL-6 levels of STEMI patients is significantly higher than NSTEMI patients. A study suggests that the IL-2 complex may serve as a promising therapeutic approach to attenuate adverse remodeling after myocardial infarction [20]. More importantly, the latest research indicate that IL-2R is independently associated with the occurrence of severe coronary artery calcification in CAD patients. The study reveal that targeting the IL-2/IL-2R pathway may be effective in preventing or treating CAD [21]. Further mechanism research has found that IL-2 improve heart function following myocardial infarction through enhancing angiogenesis in mice [22]. In addition, plasma IL-6 level has been reported to be elevated in acute myocardial infarction patient [23]. Further research has found that circulating IL-6 is associated with central obesity, hypertension and insulin resistance. Research reveals that IL-6 participates in the pathogenesis of coronary heart disease through a combination of autocrine, paracrine and endocrine mechanisms[24]. Research shows that IL-17-driven inflammation is associated with clinical instability in patients with CAD [25], this study found that the level of IL-17 in patients with unstable angina were significantly higher than those in patients with acute myocardial infarction, indicating that IL-17 may be involved in the progression of unstable plaques.

The aim of this study was to elucidate the levels of lipids, glucose, and cytokine levels in different stages of CAD. The research results will contribute to a deeper understanding of the roles of lipids, glucose, and inflammation in the occurrence and development of CAD, providing direction for future research on specific molecular mechanisms and laying a theoretical foundation for the development of prevention and diagnosis strategies for CAD. This study has limitations, Firstly, the sample size is small, and future multicenter large sample studies can be conducted. Secondly, this study is a retrospective study and lacks follow-up data. Prospective cohort

studies can be conducted in the future. Thirdly, the item of information important for coronary heart disease were not collected, BMI. Further research will be conducted in the future by supplementing this indicator. Finally, due to the correlation between genes and race, the results obtained in this study are difficult to generalize widely.

5. Conclusions

In conclusion, compared to NSTEMI patients, STEMI patients tended to be a higher TC, LDL-C, IL-2, IL-6 levels and a lower Hb1Ac.

CRediT authorship contribution statement

Xue Jiang: Writing – original draft, Data curation. **Xin-ying Guo:** Data curation. **Jie Zhang:** Data curation. **Guo-yong Zhang:** Data curation, Writing – review & editing, Data curation, Conceptualization. **Zheng Ma:** Data curation. **Cai-xia Guo:** Writing – review & editing, Data curation.

Ethics approval and consent to participate

According to the principles mentioned in the Declaration of Helsinki, the ethics committees of Beijing Tongren Hospital approved the research protocol. Written informed consent were obtained from all participants (or guardians of participants) in the present study.

Consent for publication

All authors have approved this manuscript.

Availability of data and materials

The data that support the fndings of this study are available from the corresponding author upon reasonable request.

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Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

List of abbreviations

NSTEMI	acute non-ST-segment-elevation myocardial infarction
STEMI	acute ST-segment-elevation myocardial infarction
Impaired fasting glycaemia	IFG
impaired glucose tolerance	IGT
DM	DM
TC	total cholesterol
TG	triglyceride
LDL-C	low-density lipoprotein cholesterol
Lp(a)	lipoproteion(a)
HDL-C	low high-density lipoprotein cholesterol
HbA1c	glycosylated hemoglobin
CAD	coronary artery disease
IL-6	Interleukin-6
hsCRP	high-sensitivity C-reactive protein
IL-1β	interleukin-1 beta
IL-10	Interleukin-10
IL-12p70	interleukin-12p70
IL-17	Interleukin-17
IL-2	Interleukin-2
IL-4	Interleukin-4
IL-5	Interleukin-5

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IL-8	Interleukin-8
INF-α	Interferon-alpha
INF-γ	interferon-gamma
TNF-α	tumor necrosis factor-alpha
ACS	Acute coronary syndrome
LVEF Left	ventricular ejection fraction

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