

Original Article

Impact of inflammatory factors on coronary CT calcification score: correlation with plaque stability and evaluation of diagnostic efficiency

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Abstract: Objective: To investigate the correlation between inflammatory cytokines (IL-1 β , IL-6, and IL-10) and plaque stability as assessed by coronary computed tomography angiography (CTA) calcium scoring. Methods: A retrospective study was conducted in 148 patients who underwent coronary angiography in the Department of Cardiovascular Medicine of our hospital from January 2022 to December 2024. Patients were classified into a coronary heart disease (CHD) group (n = 102) and a non-CHD group (n = 46). Coronary CTA calcium scores, biochemical parameters, serum cytokine levels (IL-1 β , IL-6, IL-10), and plaque stability-related markers - including lipoprotein-associated phospholipase A2 (Lp-PLA2), lectin-like oxidized low-density lipoprotein receptor-1 (LOX-1), cathepsin K (CTSK), and pentraxin 3 (PTX3) - were compared between the groups. Patients were further stratified according to the Synergy between PCI with TAXUS and Cardiac Surgery (SYNTAX) score into mild-to-moderate and severe coronary artery disease subgroups. Correlations among inflammatory cytokines, SYNTAX score, and plaque stability-related markers were assessed and compared between the groups. Receiver operating characteristic (ROC) curve analysis was performed to evaluate the predictive value of inflammatory cytokines for CHD. Results: Significant differences were observed in peripheral blood levels of IL-1 β , IL-6, and IL-10 between the CHD and non-CHD groups (all P < 0.05). IL-1 β and IL-6 were positively correlated with both SYNTAX scores and plaque stability-related markers. ROC analysis revealed that IL-1 β had an area under the curve (AUC) of 0.754 (95% CI: 0.631-0.877, P < 0.05) with an optimal cutoff of 4.86, yielding a sensitivity of 68.6% and specificity of 89.7%. IL-6 showed an AUC of 0.746 (95% CI: 0.631-0.860, P < 0.05) with a cutoff of 6.74, corresponding to a sensitivity of 82.9% and specificity of 66.7%. Conclusion: Inflammatory factors are important contributors to the development and progression of CHD. Their levels are significantly correlated with coronary calcium scores and plaque stability-related markers, and they demonstrate favorable diagnostic performance, suggesting potential clinical utility.

Keywords: Coronary heart disease, inflammatory factors, plaque stability-related markers, diagnostic performance, correlation analysis

Introduction

Cardiovascular diseases (CVDs) have long been a major focus of public health research, as they represent a growing global threat to human health [1, 2]. According to the World Health Organization, CVDs remain one of the leading causes of death worldwide, accounting for more than 10 million deaths annually [3, 4]. Among these, coronary heart disease (CHD) is the most prevalent subtype and has shown a worrying trend toward earlier onset in younger populations [5]. Evidence indicates that the severity of coronary artery disease is directly associated with adverse cardiovascular out-

comes, highlighting the critical importance of assessing coronary artery calcification in risk stratification and patient management [6, 7].

Accumulating research supports the view that coronary atherosclerosis is a chronic inflammatory disease, in which cytokines such as interleukin (IL)-1 β , IL-6, and IL-10 play pivotal roles in disease initiation and progression [8, 9]. Moreover, plaque stability is closely associated with the occurrence of acute coronary syndromes. However, studies investigating the interplay between inflammatory cytokines, plaque stability, and coronary artery calcification remain limited [10]. Therefore, this study explores

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the correlations of IL-1 β , IL-6, and IL-10 with plaque stability and coronary calcium scores, aiming to provide a foundation for improving the overall diagnosis and treatment of CHD.

General information and methods

General information

This retrospective study included 148 patients who were admitted to the Department of Cardiovascular Medicine at our hospital between January 2022 and December 2024 and underwent coronary angiography. Patients were divided into a coronary heart disease (CHD) group (n = 102) and a non-CHD group (n = 46) according to the presence or absence of CHD. Additionally, based on a fractional flow reserve computed tomography (FFRCT) cutoff value of 0.80, patients were further stratified into high-FFRCT and low-FFRCT groups.

Inclusion criteria: 1. age between 18 and 80 years; 2. completion of coronary angiography at our hospital; 3. availability of complete and well-documented hospitalization records; 4. first-time hospitalization for examination. Exclusion criteria: 1. previous treatment for coronary artery disease; 2. presence of valvular or congenital heart disease; 3. concomitant vascular diseases; 4. infection within the past two weeks; 5. history of rheumatic immune system or hematologic diseases; 6. presence of tumors. This study was approved by the Ethics Committee of Changxing People's Hospital.

Methods

Clinical data collection: General information and routine laboratory data were collected from all participants, including complete blood count parameters (hemoglobin, white blood cells, platelets) and coagulation indicators (prothrombin time). Serum levels of liver and kidney function markers (serum albumin, alanine aminotransferase (ALT), aspartate aminotransferase (AST), creatinine, blood urea nitrogen (BUN), and total bilirubin) were measured using an automated biochemical analyzer (Roche, Switzerland). Serum inflammatory factors were measured by ELISA. The main procedures were as follows: 5 mL of fasting venous blood was collected from each subject in the morning, centrifuged at 3500 rpm (radius 10 cm) for 5 minutes. Serum C-reactive protein (CRP) levels were measured using an automated im-

munoassay analyzer (Centaur XP, Siemens, Germany). Serum IL-1 β , IL-6, and IL-10 levels were quantified using ELISA kits according to the manufacturer's instructions.

Coronary examination method

All patients underwent coronary computed tomography angiography (CCTA) in the supine position with arms elevated above the head to ensure that the heart was centered within the scanning field. The scan range extended from 1 cm below the tracheal carina to 2 cm below the left diaphragm. Scanning was performed using prospective ECG-gated axial acquisition with CARE Dose 4D activated. The reference mAs was set to 180, with a delay time of 3 seconds. Images were reconstructed with a slice thickness of 0.75 mm, an increment of 0.4 mm, and a B26f kernel in the cardiac window. Additional scanning parameters included 128 \times 0.6 mm slices, a rotation time of 0.3 s, automatic heart rate estimation, and automatic pitch. For contrast tracking, a region of interest (ROI) was placed in the descending aorta with an attenuation threshold of 150 Hounsfield units. Contrast administration protocols and filtered back projection reconstruction were adjusted according to the patient's body weight. Tube voltage was set at 100 kV for patients weighing < 60 kg, 120 kV for those weighing 60-90 kg, and 140 kV for those > 90 kg. A conventional contrast injection protocol was used, with a total contrast dose of 370 mg/kg followed by 40 mL of normal saline. The injection duration was standardized to 12 seconds, with flow rates of 4.8 mL/s for patients < 60 kg, 5.4 mL/s for those 60-90 kg, and 5.8 mL/s for those > 90 kg. Filtered back projection (FBP) reconstruction was used for image processing. Optimal image sets from either diastolic or systolic phases were selected for analysis. Specialized software was used to calculate the new coronary blood flow parameter, fractional flow reserve computed tomography (FFRCT) values and to quantify plaque characteristics, including plaque length, minimal lumen area, total plaque volume, and volumes of lipid, fibrous, and calcified plaque components.

SYNTAX score assessment

The synergy between PCI with taxus and cardiac surgery (SYNTAX) score is a validated tool for

assessing the complexity of coronary artery lesions, primarily used to guide revascularization strategies (PCI or CABG) in patients with multivessel or left main coronary artery disease. The coronary SYNTAX score was calculated using the 16-segment method, incorporating stenosis severity, lesion characteristics (e.g., bifurcation, calcification), and specific classifications (e.g., total occlusion) [11]. Lesions with a diameter ≥ 1.5 mm and stenosis $\geq 50\%$ were included in the scoring system. Scores of 0-22 were classified as mild, 23-32 as moderate, and ≥ 33 as severe. Given the limited sample size, patients were stratified into two groups according to the SYNTAX results: mild coronary artery disease and moderate-to-severe coronary artery disease.

Primary outcomes

The primary outcomes of this study were the correlations among coronary artery calcification scores, inflammatory factors, and plaque stability-related markers between the two groups. In addition, differences in plaque characteristics and inflammatory factor expression were analyzed according to fractional flow reserve computed tomography (FFRCT) levels in patients with CHD. According to FFRCT values, CHD patients were further divided into two subgroups: a high-FFRCT group (FFRCT > 0.80 , $n = 62$) and a low-FFRCT group (FFRCT ≤ 0.80 , $n = 40$).

Statistical analysis

Statistical analyses were performed using SPSS 26.0 software. Continuous variables were expressed as mean \pm standard error of the mean (Mean \pm SEM) and were analyzed using t-tests for normally distributed data. Between-group comparisons were conducted using independent samples t-tests. Categorical data were presented as rates (percentages) and were analyzed using the chi-square (χ^2) test. Receiver operating characteristic (ROC) curve analysis was employed to evaluate the predictive value of serum IL-1 β , IL-6, and IL-10 for CHD. Pearson correlation analysis was used to assess the relationships between inflammatory factors, SYNTAX scores, and plaque stability-related markers. A p -value less than 0.05 was considered statistically significant.

Results

Baseline characteristics

There were no significant differences between the CHD and non-CHD groups in terms of age, body mass index (BMI), prevalence of hypertension, smoking history, alcohol consumption, chronic obstructive pulmonary disease (COPD), history of stroke, or hyperlipidemia, indicating that the two groups were comparable. Detailed data are presented in **Table 1**.

Liver and kidney function

No statistically significant differences were observed between the CHD and non-CHD groups in liver function indicators (AST, ALT) or kidney function indicators (serum creatinine, BUN) (all $P > 0.05$). Detailed data are presented in **Figure 1**.

Peripheral blood inflammatory factor levels

Serum levels of IL-1 β , IL-6, and IL-10 differed significantly between the CHD and non-CHD groups. Patients in the CHD group exhibited elevated IL-1 β and IL-6 levels and reduced IL-10 levels compared to the non-CHD group, with all differences reaching statistical significance (all $P < 0.05$). Detailed data are shown in **Figure 2**.

Predictive value of peripheral blood inflammatory factors for CHD and their association with plaque stability-related markers and SYNTAX scores

Significant differences were observed in peripheral blood levels of IL-1 β , IL-6, IL-10, and plaque stability-related markers (Lp-PLA2, LOX-1, CTSK, and PTX3) among patients with varying SYNTAX scores. Specifically, patients with mild-to-moderate coronary artery disease exhibited lower IL-1 β and IL-6 levels and higher IL-10 levels compared to those in the severe coronary artery disease group, with all differences reaching statistical significance (all $P < 0.05$). Correlation analysis demonstrated that IL-1 β and IL-6 were positively associated with SYNTAX scores and plaque stability-related markers (all $P < 0.05$), whereas IL-10 showed negative correlations with both ($P < 0.05$).

ROC curve analysis indicated optimal cutoff values for predicting CHD at 3.25 ng/mL for IL-1 β , 3.15 ng/mL for IL-6, and 1.55 ng/mL for IL-10.

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Table 1. Baseline characteristics of patients in the CHD and non-CHD groups

Characteristic	CHD group (n = 102)	No-CHD (n = 46)	Statistical value	P
Age	58.9±6.2	59.2±6.3	0.271	0.787
Gender (Male/Female)	56/46	30/16	1.386	0.239
BMI (kg/m ²)	25.3±1.9	25.5±2.2	0.564	0.574
History of hypertension	65	30	0.031	0.861
History of smoking	32	15	0.022	0.881
COPD	10	9	2.700	0.100
History of stroke	7	3	0.077	0.781
History of atrial fibrillation	9	4	0.083	0.773
FPG	5.85±0.84	5.87±0.75	0.139	0.890
TC	4.64±0.92	4.40±0.98	1.429	0.155
TG	1.82±1.09	1.87±0.94	0.269	0.788
HDL.C	1.08±0.25	1.02±0.27	1.318	0.190
LDL.C	2.73±0.82	2.69±0.89	0.267	0.790
History of antihypertensive drug use	60	28	0.297	0.586
History of anticoagulant drug use	65	22	3.308	0.069
History of lipid-lowering drug use	43	25	1.897	0.168
Inner diameter of the left atrium	41.5±3.8	42.0±4.0	0.729	0.467
Left ventricular ejection fraction	56.6±3.5	55.9±4.0	1.076	0.283
Left indoor diameter	43.8±2.7	44.7±2.9	1.834	0.069
BNP	258.4±40.0	252.7±43.5	0.781	0.436

Note: BMI, body mass index; COPD, chronic obstructive pulmonary disease; FPG, fasting plasma glucose; TC, total cholesterol; TG, triglycerides; HDL.C, high-density lipoprotein cholesterol; LDL.C, low-density lipoprotein cholesterol; BNP, b-type natriuretic peptide.

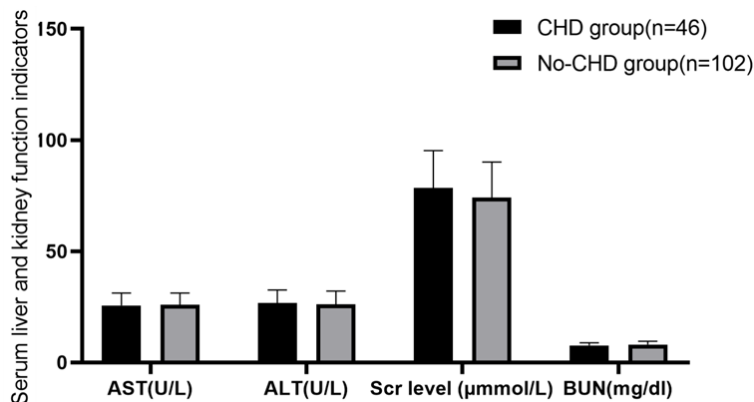


Figure 1. Comparison of liver and kidney functions between CHD and no-CHD patients. CHD, coronary heart disease.

Detailed data are presented in **Tables 2-4** and **Figures 3-6**.

Correlations between plaque characteristics, inflammatory cytokines, and inflammatory response indexes in patients with different FFRCT levels

CHD patients were stratified into a high-FFRCT group (n = 62) and a low-FFRCT group (n = 40). Compared with the high-FFRCT group, patients

in the low FFRCT group had significantly greater plaque length, total plaque volume, and lipid plaque volume, along with a smaller minimum lumen area. No statistically significant differences were observed in fibrous plaque or calcified plaque volumes between the two groups. Peripheral blood levels of IL-1 β and IL-6 were significantly higher in the low-FFRCT group than those in the high-FFRCT group (both P < 0.05). Correlation analysis further revealed that plaque length, total plaque volume, and lipid

plaque volume were positively associated with IL-1 β and IL-6, but negatively associated with IL-10. Conversely, the minimum lumen area was negatively correlated with IL-1 β and IL-6, but positively correlated with IL-10 (all P < 0.05). Detailed results are shown in **Tables 5-7**.

Discussion

CHD is one of the most prevalent cardiovascular diseases worldwide [12, 13]. Revasculari-

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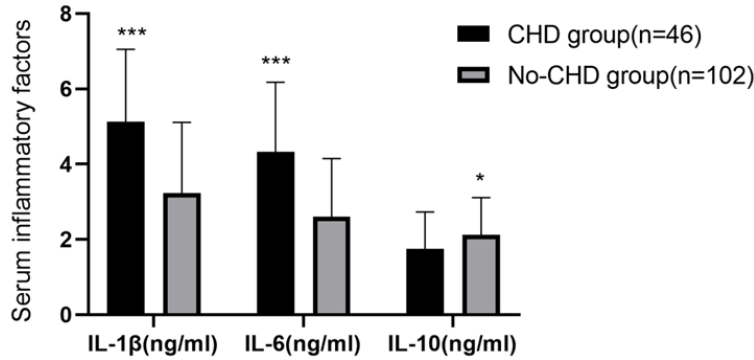


Figure 2. Comparison of peripheral serum inflammatory cytokine levels between CHD and no-CHD patients. Compared with the no-CHD group, *** $P < 0.001$, * $P < 0.05$. CHD, coronary heart disease.

Table 2. Peripheral blood inflammatory factor levels in patients with different SYNTAX scores

Cytokine	Mild-moderate group (n = 69)	Severe group (n = 33)	Statistical value	P
IL-1β (ng/ml)	4.65±1.64	6.03±1.59	-4.014	< 0.001
IL-6 (ng/ml)	3.78±1.69	5.19±1.54	-4.054	< 0.001
IL-10 (ng/ml)	1.80±0.73	1.41±0.69	2.568	0.012

Note: IL-1β, interleukin-1 beta; IL-6, interleukin-6; IL-10, interleukin-10.

Table 3. Diagnostic performance of inflammatory factors for coronary heart disease

Cytokine	Sensitivity	Specificity	AUC (95% CI)
IL-1β	77.50%	43.5%	0.767 (0.683-0.851)
IL-6	73.5%	41.3%	0.746 (0.666-0.826)
IL-10	71.6%	47.8%	0.708 (0.619-0.797)

Note: IL-1β, interleukin-1 beta; IL-6, interleukin-6; IL-10, interleukin-10.

Table 4. Correlation comparison between inflammatory factors and plaque stabilizing factors

Classification	IL-1β		IL-6		IL-10	
	r	P	r	P	r	P
Lp-PLA2	0.887	0.012	0.772	0.010	-0.834	0.001
LOX-1	0.767	0.034	0.781	0.027	-0.861	0.002
CTSK	0.810	0.031	0.802	0.014	-0.855	0.001
PTX3	0.865	0.010	0.813	0.002	-0.817	0.017

Note: Lp-PLA2, lipoprotein-associated phospholipase A2; LOX-1, lectin-like oxidized low-density lipoprotein receptor-1; CTSK, cathepsin K; PTX3, pentraxin 3.

zation, through medical and surgical treatments, remains the mainstay of treatment [14]. Nevertheless, due to the acute onset and rapid progression of CHD, some patients continue to experience irreversible cardiac dysfunction despite receiving treatment. Early and effective

diagnosis of CHD is therefore critical to improving patient prognosis. At present, coronary angiography is considered the gold standard for CHD; however, as an invasive procedure, its clinical use is limited. Non-invasive imaging techniques combined with serological markers are increasingly favored in clinical practice [15]. Moreover, effective assessment of coronary atherosclerosis is particularly important, as unstable plaques are prone to hemorrhage or rupture, promoting platelet adhesion and aggregation, which can result in thrombus formation, coronary artery occlusion, and adverse cardiovascular events [16, 17]. Additionally, CHD patients often present with systemic atherosclerotic lesions, such as carotid artery atherosclerosis, which may contribute to acute neurological dysfunction [18]. These observations underscore the clinical significance of assessing plaque stability in CHD. Recent studies highlight the pivotal role of inflammation in coronary plaque formation, CHD progression, and plaque stability. Accordingly, investigating the role of inflammatory factors in CHD diagnosis and treatment holds significant clinical value [19, 20]. In this context, we conducted a retrospective analysis of patient data and serum biomarkers at our center to explore the utility of inflammatory factors in assessing coronary artery calcification, diagnosing CHD, and evaluating plaque stability.

This study investigated the relationship between inflammatory factors and coronary atherosclerosis. The results indicated that patients with CHD exhibited elevated peripheral levels of pro-inflammatory factors IL-6 and IL-1β, whereas levels of the anti-inflammatory cyto-

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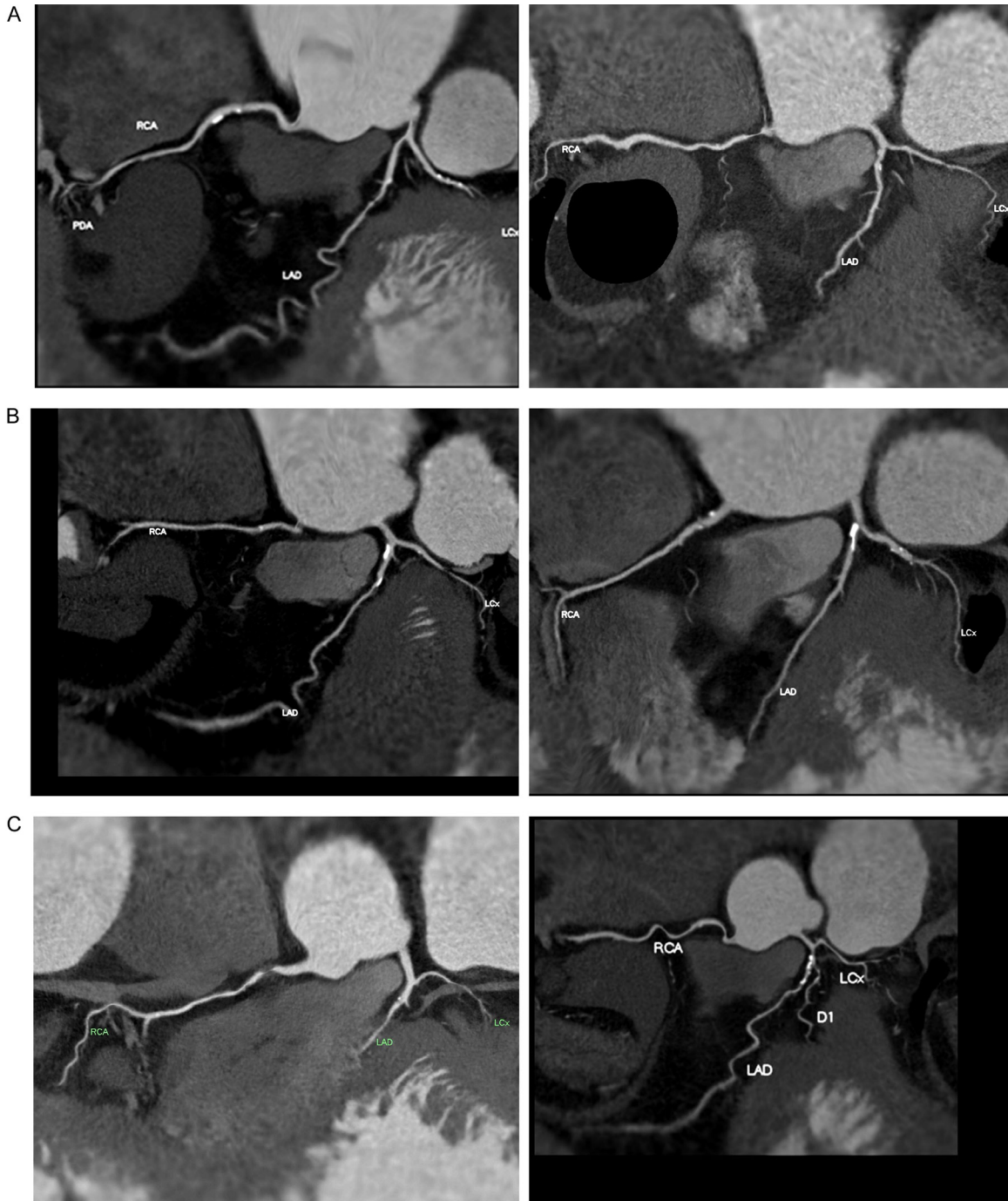


Figure 3. Representative imaging of coronary artery lesions of varying severity. A: Mild coronary artery disease; B: Moderate coronary artery disease; C: Severe coronary artery disease.

kine IL-10 were reduced compared to healthy controls. Correlation analysis revealed a positive association between SYNTAX scores and pro-inflammatory factors, and a negative association with IL-10. This may be explained by the fact that IL-6 is primarily produced by activated immune cells-including monocytes/macrophages

and T cells-as well as endothelial cells, vascular smooth muscle cells, and adipocytes, and functions as a key inflammatory mediator throughout atherosclerosis. IL-1 β plays a central role from the early stage of endothelial injury, promoting adhesion molecule expression, to foam cell formation, macrophage lipid uptake,

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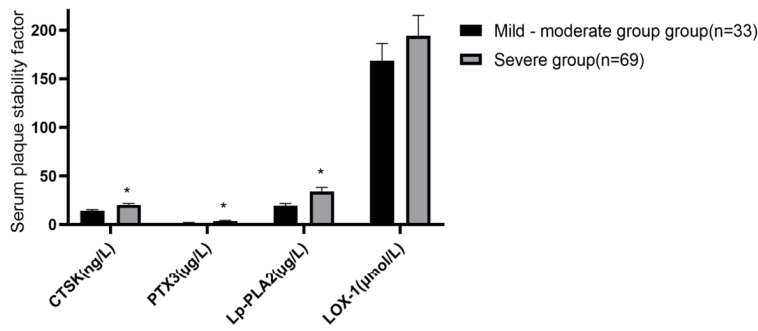


Figure 4. Impact of coronary artery lesion severity on plaque stability markers. Compared with the mild-moderate group, *P < 0.05.

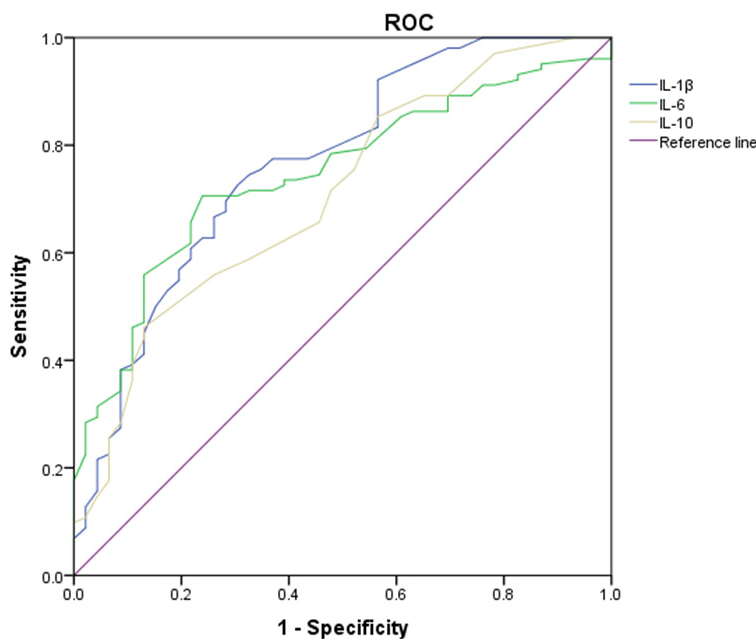


Figure 5. Receiver Operating Characteristic (ROC) curves of inflammatory factors for predicting coronary heart disease.

and plaque development leading to CHD. As a key cytokine of the innate immune system, IL-1 β is mainly secreted by activated macrophages, dendritic cells, and endothelial cells via activation of the NLRP3 inflammasome, an intracellular multiprotein complex, and serves as a primary initiator of vascular inflammation. Furthermore, IL-1 β strongly induces the production of other pro-inflammatory cytokines, such as IL-6 and TNF- α , and chemokines, generating an “inflammatory cascade amplification” that exacerbates vascular inflammation and aggravates coronary artery injury. In contrast, IL-10 is produced by regulatory T cells (Tregs), M2 macrophages, dendritic cells, and B cells. It exerts

anti-inflammatory effects by inhibiting macrophage release of IL-1 β and IL-6, thereby interrupting the inflammatory cascade amplification, and by promoting macrophage polarization toward the M2 phenotype, which reduces inflammation within atherosclerotic plaques. These findings are consistent with previous studies. Moreover, our analysis demonstrated that predictive models incorporating IL-6, IL-1 β , and IL-10 exhibited robust diagnostic performance, further confirming the significant association between inflammatory cytokines and CHD, in agreement with prior research [21-23]. These results suggest that assessing the expression levels of inflammatory cytokines may facilitate early prevention of CHD and provide a solid theoretical basis for its diagnosis.

Further analysis of the relationship between inflammatory factors and plaque stability-related markers revealed significant differences between patients with severe coronary artery stenosis and those with mild lesions. Correlation analysis indicated a positive association between pro-inflammatory cytokines and plaque stability-related markers, whereas anti-inflammatory cytokines were inversely correlated. Mechanistically, IL-6 can stimulate the production of matrix metalloproteinases (MMPs), which degrade collagen in the fibrous cap, thereby rendering plaques more vulnerable to rupture and increasing the risk of acute coronary syndromes, including myocardial infarction and unstable angina. IL-1 β is expressed within atherosclerotic plaques and directly promotes plaque rupture, precipitating acute cardiovascular events. In contrast, IL-10 exerts a stabilizing effect by antagonizing the expression of pro-inflammatory cytokines and reducing plaque-related markers, supporting previous research

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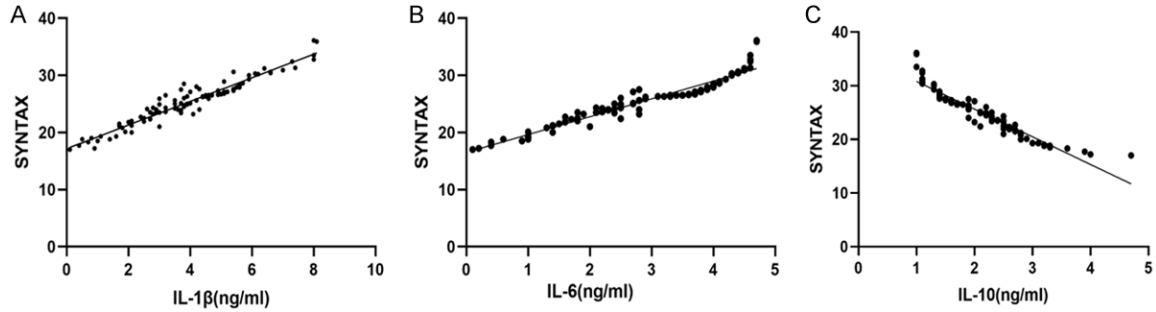


Figure 6. Correlation between coronary artery calcification SYNTAX score and IL-1 β (A), IL-6 (B), and IL-10 (C).

Table 5. Coronary plaque characteristics in patients with different FFRCT levels

Group	Plaque length	Minimum lumen area	Total plaque volume	Lipid plaque volume	Volume of fibrous plaque	Calcified plaque volume
Low-FFRCT group (n = 62)	21.46 \pm 1.53	69.50 \pm 4.54	181.64 \pm 6.89	21.22 \pm 3.83	127.53 \pm 17.00	8.91 \pm 1.43
High-FFRCT group (n = 40)	14.88 \pm 1.33	75.47 \pm 5.73	165.45 \pm 5.38	18.98 \pm 3.68	125.49 \pm 16.43	8.77 \pm 1.50
t	22.290	5.843	12.580	2.928	0.600	0.474
P	< 0.001	< 0.001	< 0.001	0.004	0.550	0.637

Note: FFRCT, fractional flow reserve derived from computed tomography.

Table 6. Inflammatory cytokine levels in patients with different FFRCT levels

Group	Case	IL-1 β	IL-6	IL-10
Low FFRCT group	62	5.89 \pm 0.74	4.26 \pm 0.51	1.06 \pm 0.29
High FFRCT group	40	4.26 \pm 0.61	3.50 \pm 0.42	1.56 \pm 0.27
t	-	11.610	7.858	8.731
P	-	< 0.001	< 0.001	< 0.001

Note: FFRCT, fractional flow reserve derived from computed tomography.

Table 7. Correlation between plaque characteristics and inflammatory factor indicator levels (r/P)

Plaque Characteristic	IL-6	IL-1 β	IL-10
Plaque length	0.669/0.032	0.724/0.028	-0.683/0.041
Total plaque volume	0.739/0.027	0.719/0.016	-0.737/0.036
Lipid plaque volume	0.747/0.011	0.727/0.011	-0.749/0.033
Minimum lumen area	-0.780/0.010	-0.721/0.023	0.748/0.029

Note: IL-1 β , interleukin-1 beta; IL-6, interleukin-6; IL-10, interleukin-10.

findings [24-26]. These results further confirm the relationship between inflammatory cytokines and plaque-stabilizing markers and may serve as an early warning indicator for the clinical prevention of adverse cardiovascular events.

Previous studies have demonstrated that coronary blood flow parameters derived from raw data obtained through coronary CT angiogra-

phy (CTA) can reliably reflect coronary hemodynamics [27]. Our study showed that patients with higher fractional flow reserve (FFR) exhibited lower coronary calcification and more favorable plaque characteristics compared to those with lower FFR. Additionally, we observed a correlation between plaque features and inflammatory factors. The underlying mechanism may be as follows: pro-inflammatory cytokines such as TNF- α and IL-1 β increase vascular endothelial permeability, facilitating the infiltration of low-density lipoprotein (LDL) into the vascular intima, where it undergoes oxidation to form oxidized LDL (ox-LDL). Ox-LDL is subsequently phagocytosed by macrophages, leading to

foam cell formation and contributing to the development of the lipid-rich necrotic core. Activated monocytes, T cells and other infiltrating plaques release matrix metalloproteinases (MMPs), which degrade collagen fibers in the fibrous cap, leading to cap thinning and an increased risk of plaque rupture. Eventually, the inflammatory environment impairs smooth muscle cell proliferation and collagen synthesis, further compromising plaque stability [28,

29]. This study has several limitations. First, the sample size was relatively small and derived from a single center, which may limit the generalizability of the findings. Second, this is a nested case-control study and the severity of patients' conditions varied. Therefore, multi-center studies with larger sample sizes are needed to further validate these conclusions. Additionally, this study did not evaluate the combined diagnostic efficacy of the three cytokines, as the individual markers already demonstrate strong predictive performance. Assessing the collective predictive value of these biomarkers represents an important avenue for future research to further strengthen the study's conclusions.

In conclusion, peripheral serum inflammatory cytokines IL-6, IL-1 β and IL-10 are closely associated with coronary atherosclerosis and plaque stability. Their combined assessment can increase the detection rate of CHD and demonstrates robust diagnostic value, supporting its potential clinical application.

Disclosure of conflict of interest

None.

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