

The development of a coronary heart disease risk prediction model based on non-invasive myocardial work parameters and lipid metabolism indicators

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Abstract

Objective The aim is to develop a new risk prediction model for coronary heart disease (CHD) by utilizing Non-invasive Myocardial Work (MW) parameters and lipid metabolism indicators, so as to identify and manage populations at high risk of CHD at an early stage.

Methods Patients with suspected CHD who were admitted to the Affiliated Hospital of Youjiang Medical University for Nationalities from October 2024 to June 2025 and scheduled to undergo Coronary Angiography (CAG) were prospectively collected. They were divided into the CHD group and the non-CHD group according to the CAG results. Logistic regression was used to identify factors related to CHD, and R-Studio was employed to construct a nomogram model for predicting CHD based on independent predictors.

Results Pearson correlation analysis and multivariate logistic regression analysis showed that the Atherogenic Index of Plasma (AIP), Global Work Index (GWI), Global Wasted Work (GWW), and Left Ventricular Posterior Wall (LVPW) at End - Diastole were independent risk factors associated with CHD. Based on the results of multivariate logistic regression, the CHD risk prediction model constructed using R-Studio showed an average AUC of 0.836 and a C-index of 0.847 in the validation set, indicating that the model can well distinguish between high-risk and low-risk populations for CHD. After calibration, the Mean Absolute Error (MAE) of the calibration curve was 0.017, which further verified the robustness and reliability of its discriminative efficacy. Decision curve analysis showed that when the threshold probability was in the range of 10%-95%, the model had a relatively high clinical net benefit value.

Conclusion This study established a CHD risk prediction model based on non-invasive myocardial work parameters and lipid metabolism indicators, which can effectively identify high-risk populations of CHD. It provides a non-invasive, portable and accurate CHD risk assessment tool for most medical institutions in China, assists clinicians in making correct clinical decisions, and thus improves the prognostic effect.

1. Introduction

CHD is a chronic immune-inflammatory and fibroproliferative disease driven by lipids. When blood lipids remain at a high level, they can activate the serum complement system in the body, producing factors that are harmful to vascular endothelial cells, damaging vascular barrier function, and thereby causing lipid deposition[1]. CHD is a major threat to global health. Therefore, how to early identify people at high risk of CHD for timely and effective intervention has become an urgent problem to be solved[2].

Previous studies have shown that inflammatory markers such as High-sensitivity C-reactive protein (hs-CRP) and interleukins, as well as traditional blood lipid indicators like High-Density Lipoprotein Cholesterol (HDL-C) and Low-Density Lipoprotein Cholesterol (LDL-C), can be used for early screening and risk prediction of CHD, but their sensitivity is still insufficient.[3] In recent years, multiple studies have indicated [3] that the AIP calculated using Triglyceride (TG) and HDL-C, is an independent risk factor for atherosclerotic cardiovascular diseases. Its predictive value is superior to that of individual traditional blood lipid indicators, reflecting the balance between atherogenic and anti-atherogenic processes in the body.

In terms of medical imaging, traditional echocardiographic parameters such as Left Ventricle Ejection Fraction (LVEF) have insufficient sensitivity to detect abnormal myocardial activity in patients with early-stage CHD, and abnormalities only appear when myocardial damage is relatively severe. Although Two-dimensional Speckle Tracking can detect myocardial dysfunction relatively well, it is susceptible to the influence of load, leading to errors in the results. As a new technology for evaluating myocardial function, MW technology breaks through traditional

limitations. By integrating myocardial longitudinal strain and blood pressure data, it can sensitively detect myocardial metabolic abnormalities in patients with coronary artery stenosis $\geq 50\%$ at an early stage, provide quantitative assessment of left ventricular function, and offer a new perspective for early screening[4, 5].

In China, in the early screening for CHD in the outpatient departments of most medical institutions, most outpatient doctors usually assess the risk of early CHD based on patients' symptoms, medical history, electrocardiogram, relevant serological indicators, and echocardiography. For experienced outpatient doctors, it is not difficult to assess people at high risk of CHD through the above methods. However, for doctors with limited clinical experience, it is sometimes difficult to accurately assess the risk of CHD in suspected patients only through the above indicators. If the incidence of CHD in the patient is underestimated and the patient is not admitted to the hospital for further diagnosis and treatment in a timely manner, the opportunity for early intervention may be missed, leading the patient to develop acute coronary syndrome. If the risk of CHD in the patient is overestimated and the patient is admitted to the hospital blindly, it will inevitably result in the waste of medical resources and increase the economic burden on the patient.

To address the above-mentioned clinical dilemmas, various clinical scoring systems for CHD are often used as screening tools in outpatient settings. Currently, pre-test probability models related to CHD include the Diamond and Forrester models, among others[6]. However, these risk assessment tools were developed for Western populations and have certain limitations in terms of universal applicability, making them not entirely suitable for Asian populations. Therefore, there is an urgent need in current clinical practice for a CHD risk assessment tool that is applicable to Asian populations, particularly the Chinese population. Moreover, this tool should assist clinicians in quickly and effectively identifying individuals at high risk of CHD, enabling timely and standardized management of such populations.

In light of the above, this study aims to construct a functional-metabolic dual-dimensional nomogram model capable of early predicting the risk of CHD by utilizing MW parameters and lipid metabolism indicators. By quantifying the synergistic diagnostic value of both for CHD, it is intended to provide a more comprehensive non-invasive prediction tool for clinical practice.

2. Materials and Methods

2.1 General Information

A total of 129 suspected CHD patients (80 in the CHD group and 49 in the non-CHD group) who were scheduled to undergo CAG examination in our hospital from October 2024 to June 2025 were included. All patients were informed of the study in advance and signed the informed consent form. The diagnosis of CHD was made in accordance with the relevant diagnostic criteria: left main stem diameter stenosis $\geq 50\%$, or stenosis $\geq 70\%$ in at least one of the left anterior descending artery, left circumflex artery, or right coronary artery with ischemic symptoms, or branch vessel stenosis $\geq 90\%$. The diagnostic criteria were in line with those specified in the 2024 ESC Guidelines for the management of chronic coronary syndromes [7].

Inclusion criteria: Echocardiography and relevant laboratory tests were performed within 3 days before CAG; Echocardiography showed no obvious segmental wall motion abnormalities; The condition was comprehensively analyzed, and combined with CAG, the diagnosis was clearly confirmed as CHD or non-CHD, with complete medical records.

Exclusion criteria: ☐ Patients with congenital heart disease, myocardial infarction, heart failure, chronic pulmonary heart disease, as well as those with severe valvular disease, cardiomyopathy, or mitral regurgitation of moderate degree or above; ☐ Patients with peripheral vascular diseases where the brachial artery cannot truly reflect central arterial pressure, etc.; ☐ Patients with poor image quality that cannot clearly distinguish the endocardium; ☐ Patients who have taken drugs including statins that may affect Total Cholesterol (TC), TG, HDL-C, etc. before the visit; those with complicated infectious diseases; severe cardiopulmonary insufficiency; hepatic or renal insufficiency; malignant tumors; connective tissue diseases; ☐ Patients who have previously undergone Percutaneous Coronary Intervention (PCI) or coronary artery bypass grafting.

This study was approved by the Ethics Committee of the Affiliated Hospital of Youjiang Medical University for Nationalities (Approval No.: YYFY-2024-248).

2.2.Measure

Collection of patients' general data

The general data collected include age, gender, Body Mass Index (BMI), and blood pressure (including Systolic Blood Pressure [SBP] and Diastolic Blood Pressure [DBP]).

Collection of patients' serological index tests

Fasting peripheral venous blood was collected from the cubital vein in the early morning, and blood biochemical indicators were detected using a fully automatic biochemical analyzer, including: Fasting Plasma Glucose (FPG), TC, TG, HDL-C, and LDL-C. Additionally, the AIP and Triglyceride-Glucose (TyG) were calculated, where $AIP = \log(TG/HDL-C)$ and $TyG = \ln(TG [mg/dL] \times FPG [mg/dL] / 2)$.

Collection of echocardiographic parameters

A GE Vivid E95 color ultrasound diagnostic instrument with an M5S linear array probe (probe frequency: 1.5–4.6 MHz, frame rate: 70–80 frames/s) was used, equipped with an Echo PAC (Version 203) workstation for offline image analysis and processing. Patients underwent echocardiography to collect images within 3 days before CAG.

First, connect the surface electrocardiogram and measure the brachial artery blood pressure of the left upper limb using the cuff method. The patient was placed in the left lateral decubitus position, and conventional echocardiographic parameters were collected under the state of quiet breathing, including standard parasternal left ventricular long-axis view and apical four-chamber view, to obtain Left Atrial diameter (LA), Left Ventricular End Diastolic Dimension (LVEDD), LVEF, Interventricular Septal Thickness at diastole (IVST), LVPW, and Epicardial Adipose Tissue (EAT). The patient was instructed to hold their breath at the end of expiration, and dynamic images of standard apical four-chamber, three-chamber, and two-chamber views were collected for 3–5 cardiac cycles. All images were standard views with clear display of endocardial boundaries.

The collected dynamic images were imported into the Echo PAC (Version 203) workstation for offline analysis and processing. According to the prompts, the region of interest was created by automatically tracking and manually correcting the traced endocardial boundaries. The software displayed the results obtained from automatic tracking, and the Global Longitudinal Strain (GLS) value of the left ventricle was acquired. Entering the myocardial work mode, the brachial artery cuff pressure was inputted to obtain the left ventricular pressure-strain loop, and the GWI, GCW, GWE and GWW were directly derived.

CAG

The patient was placed in a supine position. Routine disinfection was performed, and a sterile drape was applied. The right radial artery was used as the puncture path. After local anesthesia of the puncture site with 1% lidocaine hydrochloride, the right radial artery was punctured using the Seldinger technique, and a sheath was inserted. Unfractionated heparin was injected into the sheath. A 5F Radial TIG multi-purpose angiography catheter was used to perform conventional angiography of the left and right coronary arteries. The coronary artery conditions were evaluated by two senior interventional physicians. CHD was diagnosed if there was $\geq 50\%$ diameter stenosis of the left main stem, or $\geq 70\%$ stenosis of at least one of the left anterior descending artery, left circumflex artery, or right coronary artery with ischemic symptoms, or $\geq 90\%$ stenosis of branch vessels.

2.3.Data Analysis

Data were processed using SPSS 24.0 software. Measurement data conforming to a normal distribution were expressed as mean $\pm$ standard deviation ($\bar{x} \pm s$), and comparisons between groups were performed using independent sample t-test; those not conforming to a normal distribution were expressed as median (interquartile range). Comparisons of counting data were conducted using Fisher's exact test and Pearson's chi-square test. To avoid the impact of excessive correlation between variables on the reliability of results, Pearson correlation analysis was used to assess bivariate correlations. Univariate and multivariate Logistic regression analyses were applied to identify independent risk factors for the occurrence of CHD. Based on the independent risk factors identified by logistic analysis, a CHD risk prediction nomogram model was constructed using R-Studio. To evaluate the discriminative ability of the model, a five-fold cross-validation method was used to plot the ROC curve. For calibration, the study plotted the calibration curve using the Bootstrap method (with 1000 repeated samplings). Finally, Decision Curve Analysis (DCA) was employed to evaluate the clinical benefit of the model.

3. Result

3.1.Comparison of general data and echocardiographic parameters.

A total of 129 subjects were included in this study, including 80 cases in the CHD group and 49 cases in the non-CHD group. Through comparative analysis of clinical data and ultrasonic parameters (**Tables 1 and 2**), there were statistically significant differences in LVEF, IVST, GLS, GWI, GCW, GWW, GWE, LVPW, EAT, HDL-C, and AIP between the two groups ($P < 0.05$). Compared with the non-CHD group, the CHD group had higher levels of LVEF, GLS, GWI, GCW, GWE, and AIP, while lower levels of IVST, GWW, LVPW, EAT, and HDL-C.

Table 1.Comparison of patients' clinical data

Characteristic	CHD (n=80)	non-CHD (n=35)	p-value
age[M(P25,P75),year]	54(50,57)	53(48,56.5)	0.464
gender			0.604
Male	51(63.8%)	29(36.3%)	
Female	29(59.2)	20(40.8)	
SBP/(mmhg)	133.48±17.64	137.53±15.54	0.188
DBP[M(P25,P75),mmhg]	76.5(70,86)	78(70,86)	0.961
BMI[M(P25,P75)]	23.98(22.60,26.80)	24.78(21.81,27.63)	0.793
LDL-C/(mmol/L)	2.25±0.79	2.28±0.87	0.377
HDL-C[M(P25,P75),mmol/L]	1.13(0.88,1.32)	1.36(1.13,1.57)	<0.001
FBG[M(P25,P75),%]	5.25(4.83,5.89)	5.27(4.91,5.87)	0.806
TG[[M(P25,P75),mmol/L]	1.76(1.42,2.14)	1.81(1.18,2.12)	0.301
TC[M(P25,P75),mmol/L]	4.40(3.53,4.92)	4.56(3.87,5.35)	0.188
AIP[M(P25,P75)]	0.18(0.10,0.37)	0.10(-0.01,0.22)	0.003
TyG[M(P25,P75)]	8.88(8.68,9.32)	8.97(8.47,9.15)	0.536

SBP Systolic Blood Pressure; DBP diastolic blood pressure; BMI body mass index; LDL-C Low Density Lipoprotein Cholesterol; HDL-C High-Density Lipoprotein Cholesterol; FBG fasting plasma glucose; TG Triglyceride; TC total cholesterol; AIP Atherogenic Index of Plasma; TyG triglyceride-glucose.

Table 2. Comparison of patients' ultrasonic parameters

Characteristic	CHD (n=80)	non-CHD (n=35)	p-value
LVEF/(mm)	61.38±5.82	63.59±6.11	0.042
LVEDD[M(P25,P75),mm]	48(46,51)	47(45.5,50)	0.184
IVST/(mm)	10.69±1.74	9.82±1.52	0.005
LVPW/[M(P25,P75)]mm]	9.9(9.2,10.8)	9.10(8.75,9.95)	<0.001
LA[M(P25,P75),mm]	29.65(27.63,31.60)	30(28.35,33)	0.261
EAT[M(P25,P75),mm]	5.12(4.39,6.35)	4.45(3.99,5.31)	0.018
GLS[M(P25,P75),%]	16.50(14.50,17.88)	18.5(16,19.75)	<0.001
GWI[M(P25,P75),mmHg%]	1620(1482.25,1855.25)	1940(1721.75,2193.5)	<0.001
GCW[M(P25,P75),mmHg%]	1984.75(1800.63,2188)	2344(2092.5,2606.75)	<0.001
GWW[M(P25,P75),mmHg%]	209.50(155.25,263.38)	141(96.75,200)	<0.001
GWE[M(P25,P75),mmHg%]	91.50(88.00,93.00)	90.5(93,94)	0.002

LVEF Left Ventricle Ejection FractionLVEDD Left Ventricular End Diastolic DimensionIVST interventricular septal thickness at diastoleLVPW Left Ventricular Posterior WallLA left atrialdiameterEAT epicardial adipose tissueGLS global longitudinal strainGWI global work indexGCW global constructive workGWE global work efficiencyGWW global waste work.

3.2.Screening of risk factors for CHD

To avoid including redundant variables in the final model, this study conducted a correlation analysis based on the results of univariate analysis. As shown in **Table 3**, variable pairs with $|r| \geq 0.7$ were screened out: GWI-GCW and GWW-GWE. One of them was randomly retained and the other was eliminated, with GWI and GWW being retained. After eliminating redundant variables, the remaining variables were LVEF, IVST, GLS, GWI, GWW, LVPW, EAT, AIP, and HDL-C. The correlation coefficient matrix of the remaining variables was recalculated to ensure that $|r| < 0.7$ for all variables in the **Table 4**. Finally, the variance inflation factor (VIF) of the remaining variables was calculated using multicollinearity test, and the VIF of all variables was < 5 , as shown in **Table 5**.

Based on the results of univariate analysis, Pearson correlation analysis, and combined with professional expertise, multivariate Logistic regression analysis was performed with factors showing statistically significant differences between groups as independent variables (AIP, GWI, GWW, LVPW) and the occurrence of CHD as the dependent variable. The results showed that AIP, GWI, GWW, and LVPW were independent risk factors for coronary artery stenosis in patients ($P < 0.1$) (**Table 6**).

AIP[OR 10.312,95%CI(0.747~142.397),p=0.082],

GWI[OR 0.997,95%CI(0.997~0.995),p=0.002],

GWW[OR 1.006,95%CI(1.006~1.013),p=0.051],

LVPW[OR 2.398,95%CI(1.303~4.415),p=0.005].

Table 3.Correlation analysis

	LVEF	IVST	GLS	GWI	GCW	GWW	GWE	LVPW	EAT	HDL-C	AIP
LVEF	1										
IVST	-.211*	1									
GLS	.174*	-0.162	1								
GWI	.222*	0.001	.633**	1							
GCW	.223*	-0.069	.665**	.875**	1						
GWW	-0.027	0.098	-.484**	-.315**	-.209*	1					
GWE	0.074	-0.095	.613**	.498**	.432**	-.917**	1				
LVPW	-.193*	.687**	-0.113	0.063	-0.019	0.031	0.022	1			
EAT	-0.118	.344**	-.209*	-.196*	-.267**	.195*	-.237**	.178*	1		
HDL-C	0.138	0.118	.204*	.240**	0.153	-.224*	.207*	0.111	0.008	1	
AIP	0.005	-0.076	-0.133	-.189*	-.204*	0.104	-0.082	0.013	0.122	-.648**	1

*. Correlation is significant at the 0.05 level (two-tailed).

**. Correlation is significant at the 0.01 level (two-tailed).

LVEF Left Ventricle Ejection Fraction, IVST interventricular septal thickness at diastole, GLS global longitudinal strain, GWI global work index, GCW global constructive work, GWE global work efficiency, GWW global waste work, LVPW Left Ventricular Posterior Wall, EAT epicardial adipose tissue, HDL-C High-Density Lipoprotein Cholesterol, AIP Atherogenic Index of Plasma.

|r| < 0.3 indicates a weak correlation; 0.3 ≤ |r| < 0.7 indicates a moderate correlation; |r| ≥ 0.7 indicates a strong correlation.

Table 4. Correlation analysis of remaining variables after removing redundant variables

	LVEF	IVST	GLS	GWI	GWW	LVPW	EAT	AIP	HDL-C
LVEF	1								
IVST	-.211*	1							
GLS	.174*	-.162	1						
GWI	.222*	.001	.633**	1					
GWW	-.027	.098	-.484**	-.315**	1				
LVPW	-.193*	.687**	-.113	.063	.031	1			
EAT	-.118	.344**	-.209*	-.196*	.195*	.178*	1		
AIP	.005	-.076	-.133	-.189*	.104	.013	.122	1	
HDL	.138	.118	.204*	.240**	-.224*	.111	.008	-.648**	1

*. Correlation is significant at the 0.05 level (two-tailed).

**. Correlation is significant at the 0.01 level (two-tailed).

LVEF Left Ventricle Ejection Fraction, IVST interventricular septal thickness at diastole, GLS global longitudinal strain, GWI global work index, GWW global waste work, LVPW Left Ventricular Posterior Wall, EAT epicardial adipose tissue, HDL-C High-Density Lipoprotein Cholesterol, AIP Atherogenic Index of Plasma.

|r| < 0.3 indicates a weak correlation; 0.3 ≤ |r| < 0.7 indicates a moderate correlation; |r| ≥ 0.7 indicates a strong correlation.

Table 5.Multicollinearity test

Variable	VIF	1-VIF
LVEF	1.175	0.851
IVST	2.191	0.456
GLS	2.053	0.487
GWI	1.843	0.543
GWW	1.381	0.724
LVPW	2.026	0.494
EAT	1.262	0.792
HDL	2.008	0.498
AIP	1.907	0.524

LVEF Left Ventricle Ejection Fraction; IVST interventricular septal thickness at diastole; GLS global longitudinal strain; GWI global work index; GWW global waste work; LVPW Left Ventricular Posterior Wall; EAT epicardial adipose tissue; HDL-C High-Density Lipoprotein Cholesterol; AIP Atherogenic Index of Plasma.

VIF = 1 indicates that there is no multicollinearity among variables; $1 < \text{VIF} < 5$ suggests that there is a certain degree of multicollinearity, but it usually does not have a serious impact on the regression results; $5 \leq \text{VIF} < 10$ shows that there is a moderate degree of multicollinearity, which requires attention. $\text{VIF} \geq 10$ means that there is severe multicollinearity, which will have a significant impact on the estimation and interpretation of regression coefficients, and measures need to be taken to deal with it.

Table 6. Multivariate logistic regression analysis of influencing factors for CHD

Characteristic	β	SE	Wald χ^2	OR	95%CI	p-value
AIP	2.333	1.339	3.034	10.312	0.747~142.397	0.082
GWI	-0.003	0.001	9.704	0.997	0.997~0.995	0.002
GWW	0.006	0.003	3.812	1.006	1.006~1.013	0.051
LVPW	0.875	0.311	7.892	2.398	1.303~4.415	0.005

AIP Atherogenic Index of Plasma; GWI global work index; GWW global waste work; LVPW Left Ventricular Posterior Wall.

3.3 Construction of the CHD Risk Prediction Nomogram Model

Based on the four independent risk factors (GWI, GWW, LVPW, and AIP), a CHD risk prediction nomogram model was constructed using R-Studio. As shown in **Figure 1**, the variables included in this nomogram model are GWI, GWW, LVPW, and AIP. The impact of each variable on the occurrence of CHD is reflected in their respective row lengths and corresponding scores. The total score of the model is obtained by summing the scores of each variable, with a higher score indicating a greater contribution to the risk of CHD. GWI represents the overall systolic capacity of the myocardium; the lower the value, the higher the base score on the Points axis, corresponding to a higher risk of CHD. GWW represents the energy consumed by the myocardium; the higher the value, the higher the base score on the Points axis, corresponding to a higher risk of CHD. LVPW is the thickness of the left ventricular wall at the end of

diastole; the higher the value, the higher the base score on the Points axis, corresponding to a higher risk of CHD. The higher the AIP value, the higher the base score on the Points axis, corresponding to a higher risk of CHD, suggesting that abnormal lipid metabolism can exacerbate the risk of CHD. For predictive assessment, sum the base scores on the Points axis corresponding to the above risk factors, find the corresponding total score on the Total Points axis after summation, and finally refer to the "The risk of CHD" axis to read the predicted risk of CHD.

3.4 Evaluation of Clinical Performance and Clinical Applicability of the CHD Risk Prediction Nomogram Model

To evaluate the discriminative ability of the model, a five-fold cross-validation method was used to plot the ROC curve (**Figure 2**). The results showed that the model had an average AUC of 0.836 in the validation set, with a 95% CI of 0.759–0.825 and a C-index of 0.864, indicating that the model has good discriminative ability. After determining the optimal cut-off value based on the Youden index, the model had an average sensitivity of 0.838 and specificity of 0.776, which further confirmed the reliability of its classification performance.

In terms of calibration, the study plotted a calibration curve (**Figure 3**) using the Bootstrap method (with 1000 repeated samplings) and calculated a Mean Absolute Error (MAE) of 0.017, indicating a good fit between the model's predicted probabilities and the actual observed rates. In addition, the results of the Hosmer-Lemeshow goodness-of-fit test were $X^2 = 8.492$, $df = 8$, $p\text{-value} = 0.387$, which reached statistical non-significance, suggesting that the model has a good calibration degree with no obvious overfitting.

To evaluate the clinical application value of this model under different risk thresholds, a further DCA was performed (**Figure 4**). The results showed that when the risk threshold range was between 0.05 and 0.8, the net benefit brought by the CHD risk prediction nomogram model was higher than that of the "treat-all" and "treat-none" strategies. This indicates that the model has good clinical practicability within a relatively wide range of risk thresholds and can provide an effective auxiliary decision-making basis for the early intervention and management of high-risk populations with CHD.

In summary, the CHD risk prediction nomogram model constructed in this study has good discriminative ability, calibration consistency and clinical decision-making value, and thus has certain potential for clinical promotion.

4. Discussion

This study is the first to introduce AIP and non-invasive myocardial work parameters into the risk prediction model for CHD. By adding new indicators, the predictive performance of the model for CHD has been improved, enabling a more comprehensive and accurate assessment. The nomogram risk prediction model finally constructed in this study makes the prediction and analysis of clinical events more visual and graphical[8]. All parameters in this model are collectible and easy-to-operate indicators. Their extensive implementation at the primary level is conducive to carrying out disease risk stratification with large sample sizes, avoiding the omission of high-risk groups, and saving unnecessary expenses. The DCA curve analysis in this study also proves the practicability and effectiveness of the model. The model conforms to the "lipid-vessel-myocardium" cascade injury chain theory in the pathophysiological mechanism of coronary heart disease, further indicating that lipid metabolism disorder can drive the development of atherosclerosis[9].

The model developed in this study is simple to use, accurate and reliable, and features visualization. It can be used for CHD risk stratification in populations still at moderate to high risk. Compared with the simplicity of traditional prediction models, such as relying solely on serological indicators or imaging methods, the prediction model in this study overcomes the limitation of restricted universality, thus meeting the needs of large-scale population screening

at the primary level. According to the data from the Global Burden of Disease Study 2021, ischemic heart disease, especially CHD, remains the leading risk factor for death and disease burden worldwide.[10]Moreover, the continuous increase in the number of CHD patients and the disease burden is mainly caused by metabolic factors such as hypertension, hyperglycemia, high BMI, and LDL-C.[11–15]Previous studies have shown that age, gender, blood pressure, BMI, and age are risk factors for CHD, but they cannot accurately assess the risk level of CHD.

TG is the most abundant lipid in human adipose tissue. High levels of TG can lead to lipotoxicity, thereby causing the occurrence and progression of inflammation[16].HDL-C contains hundreds of lipids and proteins, which are known to perform antioxidant and anti-inflammatory functions in the regulation of metabolic diseases, including diabetes[17]. An elevated TG/HDL-C ratio has been shown to be associated with an increased risk of cardiovascular events related to cardiometabolic disorders. Particularly in middle- and low-risk populations with stable angina pectoris and no known CHD, a higher TG/HDL-C ratio has been confirmed as an independent predictor of coronary atherosclerotic events and progression[18, 19].This finding is consistent with the predictive value of AIP[20]. AIP combines TG and high-density lipoprotein cholesterol (HDL-C) levels. It not only reflects the ratio of TG to HDL-C but also represents the size of lipoprotein particles. Compared with high TG levels or low HDL-C levels, AIP can more accurately reflect the pathogenicity and specificity of dyslipidemia[21, 22].

A subsequent meta-analysis further confirmed that an elevated AIP is a potential prognostic marker for adverse cardiovascular events in patients with CAD, and that increased levels of AIP are consistently associated with an elevated risk of adverse events[23].Subsequent meta-analyses have further confirmed that an elevated AIP is a potential prognostic marker for adverse cardiovascular events in patients with CAD, and that increased AIP levels are consistently associated with an elevated risk of adverse events. The results of this study support the findings of the aforementioned meta-analyses: the CHD group not only showed significantly lower HDL-C levels than the non-CHD group, but also had a significant elevation in AIP. More importantly, this study confirmed that a significant elevation in AIP is an independent predictor of CHD, which is consistent with previous research conclusions[24].In summary, AIP can sensitively identify lipid metabolism disorders in the early stage, and its elevation is significantly associated with an increased risk of adverse cardiovascular events. This indirectly indicates the core role of lipoprotein phenotypic imbalance in driving the progression of atherosclerosis.

Early non-invasive quantification of myocardial function and structure in high-risk populations with CHD is a major clinical challenge. Previous studies have shown that there is a correlation between myocardial mechanical stress and the severity of coronary artery stenosis in CHD patients with preserved LVEF[25].Echocardiographic parameter LVPW and two-dimensional speckle tracking parameters have been confirmed as good indicators for identifying abnormal myocardial activity in CHD patients, and our study is consistent with the above-mentioned studies[26].However, two-dimensional speckle tracking technology is susceptible to afterload, which may lead to errors in the detection results. Compared with other echocardiographic parameters (including LVEF and two-dimensional speckle tracking), non-invasive myocardial work parameters can capture structural remodeling and impairment during myocardial ischemia earlier[27–29].Our research results show that the LVEF and GLS in the CHD group were not significantly lower than those in the non-CHD group, but the myocardial work performance was impaired.A previous study indicated that GWW and GWE are helpful for identifying the systolic function of the heart in patients with coronary artery stenosis and also reflect myocardial oxygen consumption [30, 31]. The results of our study are consistent with this conclusion, and the global left ventricular myocardial work parameters may have good clinical significance for screening suspected coronary heart disease.

Existing studies have shown that the CHD risk assessment system developed by Diamond and Forrester is based on age, gender, and some of the most basic clinical indicators [6]. However, this prediction system has certain

limitations: it may overestimate the risk of CHD onset and fail to incorporate some important influencing factors. Moreover, the process of obtaining parameters for these two traditional models is cumbersome and poorly operable, making them unavailable for popularization in the majority of outpatient and emergency departments in China, let alone in primary hospitals. Meanwhile, on the basis of domestic and foreign studies, this study is the first to introduce non-invasive myocardial function indicators and AIP into the prediction model, which has greatly improved the prediction accuracy and completeness of the model. In addition, after DCA analysis, the new model in this study can clearly demonstrate its applicability in clinical practice. The nomogram model constructed in this study includes a clinically practical risk scoring system. The parameters of this model are easily accessible, and it has a wide range of applications, which can be used in general hospitals and primary medical and health institutions. This convenient and fast prediction tool can help clinicians make more accurate preventive decisions, control the source of diseases, reduce the burden on the medical system, and at the same time address the current deficiencies in the universality and preventiveness of diagnostic systems.

However, this study has certain limitations. Firstly, the study samples were only obtained from a single-center regional hospital, with a limited sample size, and there was insufficient comparative verification with a wider range of medical institutions and a broader clinical population. Secondly, since the model only used the Bootstrap method for internal validation and did not adopt the verification method of multi-center external cohorts, the effect of clinical generalization still needs further exploration.

5. Conclusions

The nomogram model established in this study, which integrates indicators related to myocardial function and lipid metabolism, is a convenient and effective tool that can be used for early non-invasive screening of CHD, with an AUC value of 0.836. Whether applied in large hospitals or small community health care centers, this model can accurately predict the risk of high-risk patient groups and help clinicians make accurate judgments on patients' conditions, thereby optimizing clinical decisions and improving patient prognosis.

Declarations

Informed Consent Statement

Informed consent was obtained from all subjects involved in the study.

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Author Contribution

SWF and MXH: Writing, reviewing, editing, and validation. CCC,TFL: Writing, reviewing, editing, and funding acquisition. YMH and KH: Data collection and analysis. SHF and LTW: Writing, reviewing, and editing.

Data Availability

Data are contained within the article.

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Figures

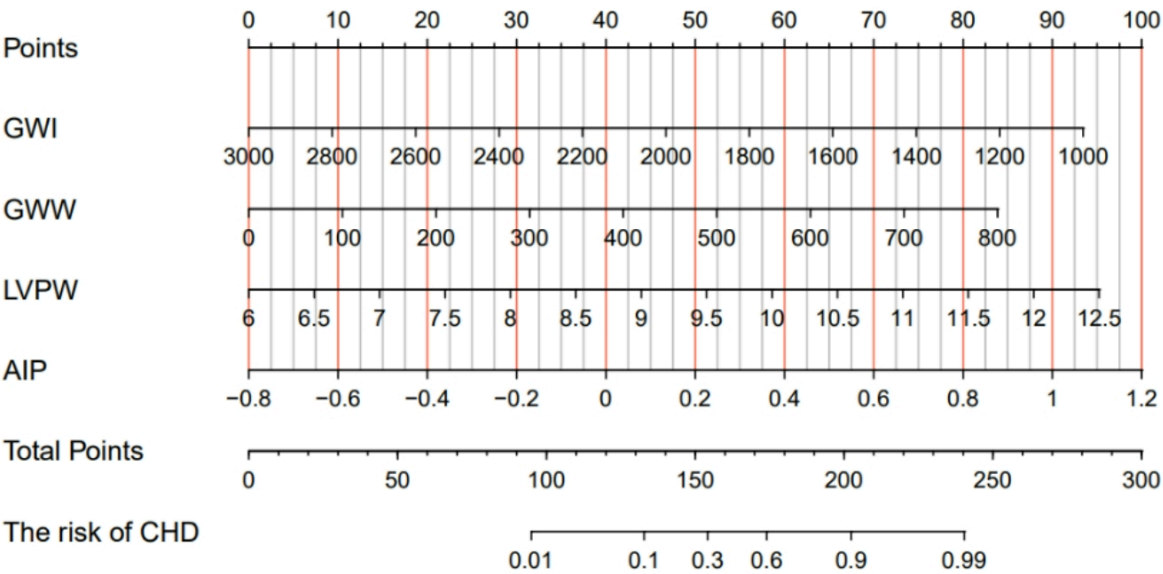


Figure 1
Nomogram model for predicting CHD risk

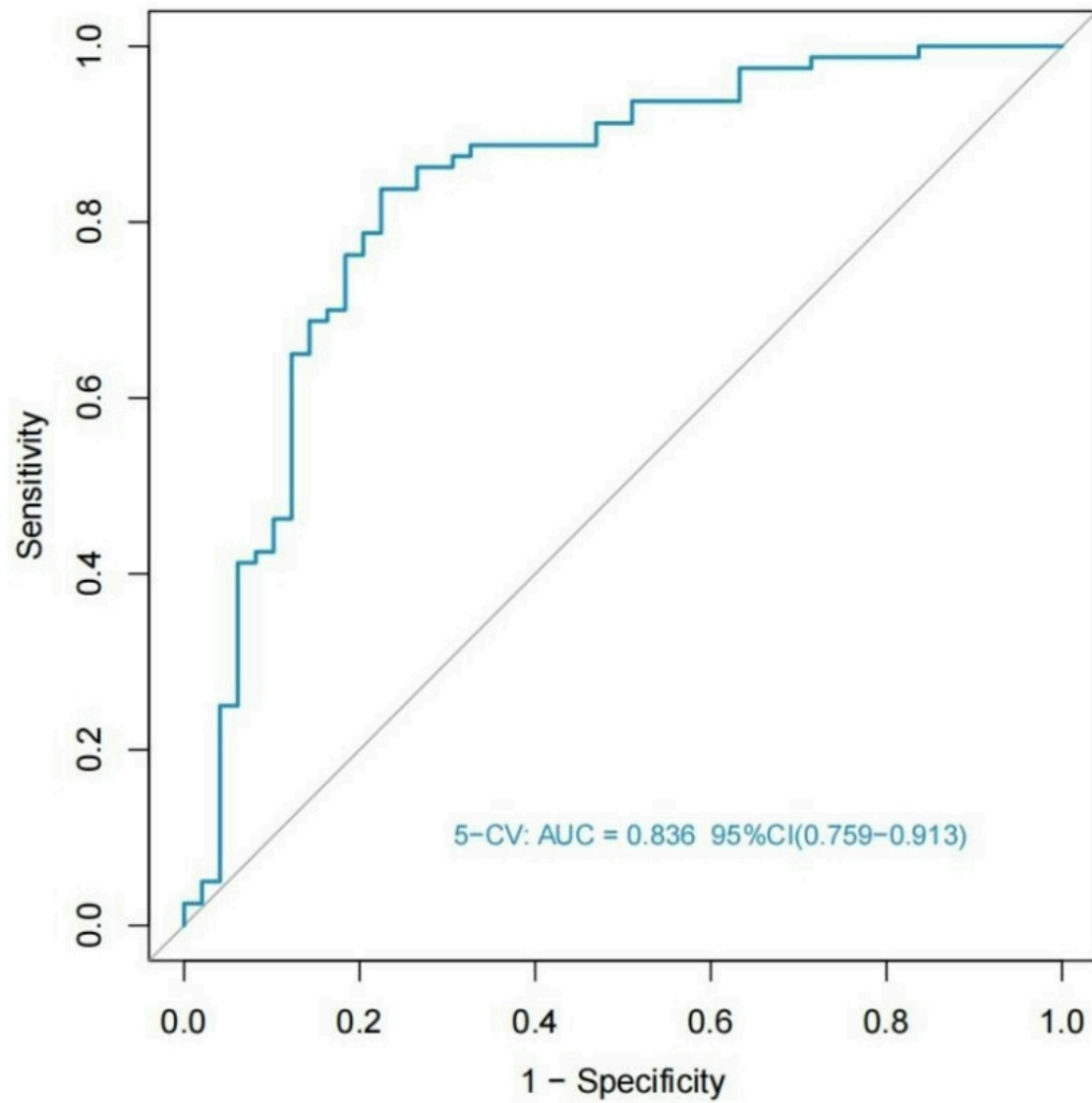


Figure 2

Receiver operating characteristic curve of the nomogram model for predicting the risk of CHD

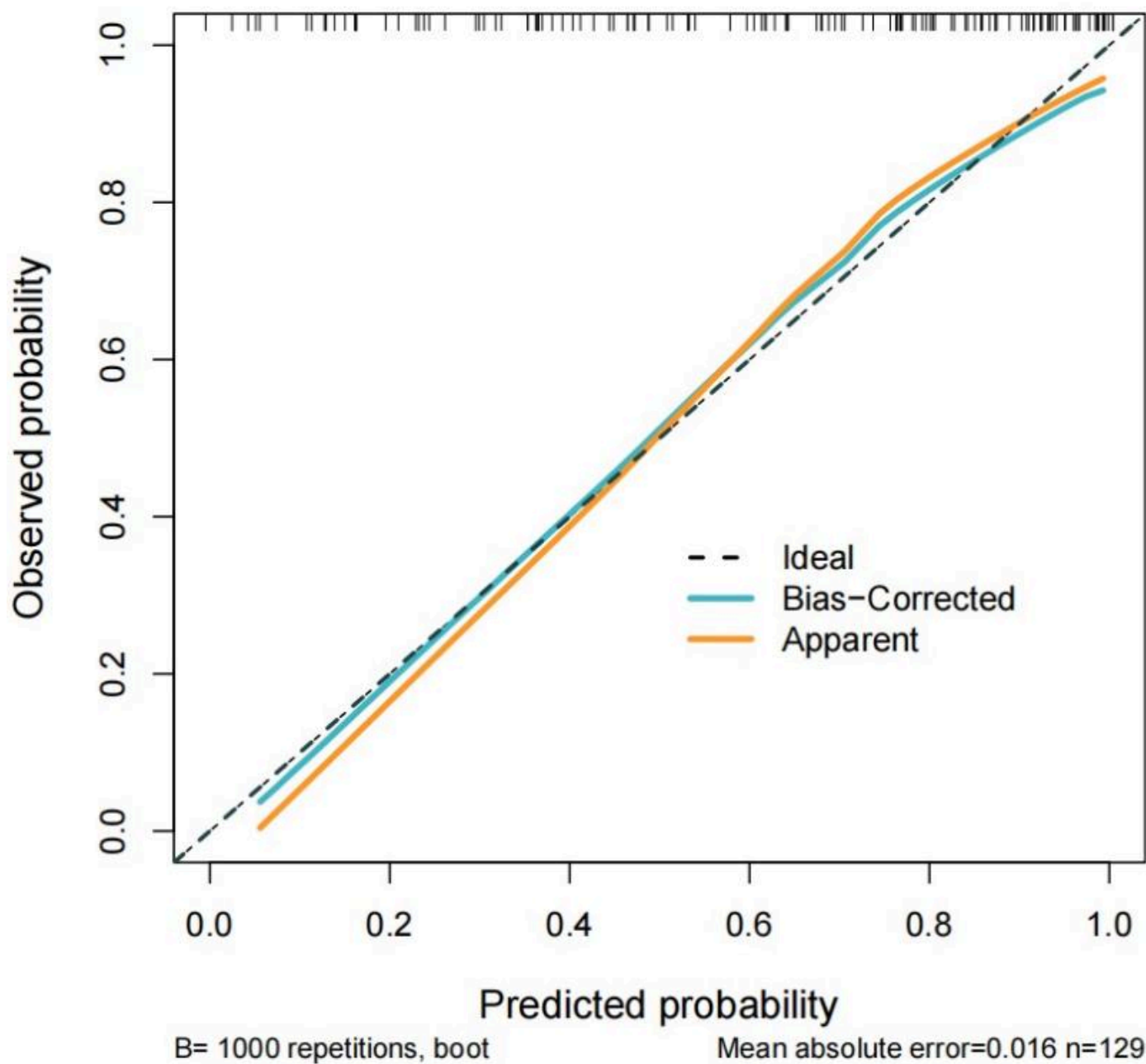


Figure 3

Calibration curve for the nomogram predicting the risk of confirmed CHD in patients with suspected coronary heart disease

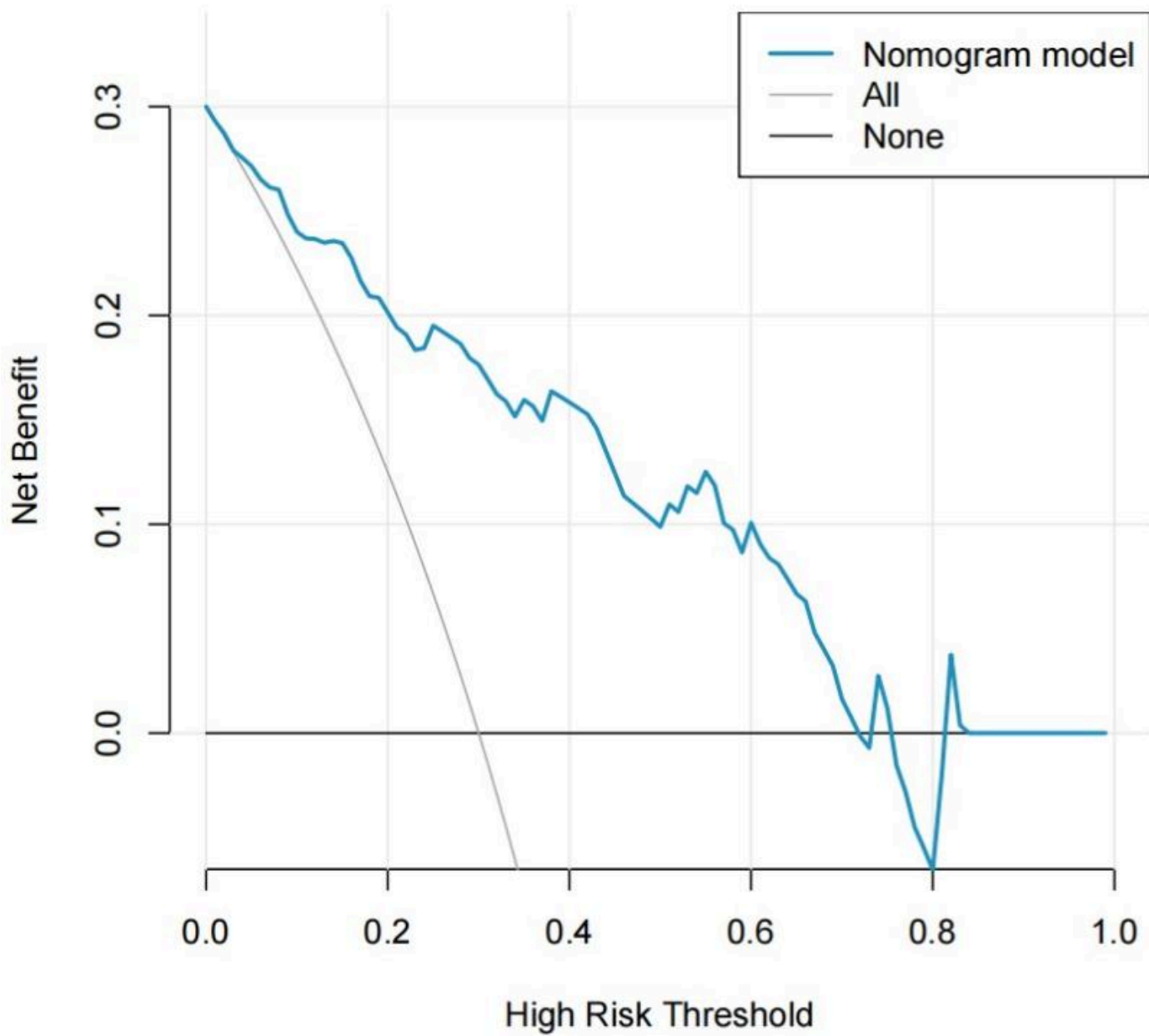


Figure 4

Decision curve for the nomogram predicting the risk of coronary heart disease