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The value of non-invasive myocardial work parameters combined with cardiac multi-dimensional parameters in predicting the risk of coronary heart disease

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Abstract: *To investigate the application value of a comprehensive model integrating non-invasive myocardial work (MW) parameters and multi-dimensional cardiac parameters in risk prediction and early screening of coronary heart disease (CHD) , a prospective study was conducted involving 141 patients with CHD. Patients were divided based on coronary angiography (CAG) results: the CHD group (n=84, coronary stenosis ≥50%) and the non-CHD group (n=57, coronary stenosis <50%). Parameters showing intergroup differences were identified by univariate analysis. Independent risk factors for CHD were determined using multivariate binary logistic regression, which were then used to construct a multi-dimensional combined prediction model. Predictive performance was evaluated using receiver operating characteristic (ROC) curves, area under the curve (AUC), sensitivity, specificity, and optimal cut-off values. The DeLong test was used to compare AUC differences between the combined model and single parameters. Univariate analysis showed that the CHD group had significantly lower levels of global constructive work (GCW), global work efficiency (GWE), global work index (GWI), global longitudinal strain (GLS), left ventricular ejection fraction (LVEF), and high-density lipoprotein cholesterol (HDL-C), and significantly higher levels of global wasted work (GWW), epicardial adipose tissue (EAT), fasting blood glucose (FBG), triglycerides (TG), triglyceride-glucose (TyG) index, Gensini score, metabolic syndrome (Mets), and smoking history compared to the non-CHD group (all P<0.05). Multivariate logistic regression confirmed that GCW, GWE, EAT, and Mets were significantly associated with the occurrence of CHD. ROC curve analysis revealed that among individual parameters, EAT had the best predictive ability (AUC=0.760). The combined model incorporating the six above-mentioned parameters achieved an AUC of 0.940 (95% CI: 0.897–0.982), with a sensitivity of 89.3% and specificity of 89.5%, outperforming all single parameters, with an AUC significantly higher than that of EAT.*

Keywords: *noninvasive myocardial work technology, metabolic syndrome, triglyceride-glucose, coronary heart disease*

1. Introduction

The pathological essence of coronary heart disease (CHD) is a chronic inflamzmatory response and fibroproliferative process mediated by abnormal lipid deposition, establishing it as a major global public health concern[1-4] . With rapid socioeconomic development and significant lifestyle changes in China, the number of affected adults has exceeded ten million, and the prevalence continues to rise. Given the lack of specific early clinical symptoms and the high risk of severe adverse cardiovascular events such as myocardial infarction, developing efficient and accurate non-invasive tools for early screening and risk stratification has become an urgent priority in both clinical management and disease prevention.

Conventional echocardiographic parameters, such as left ventricular ejection fraction (LVEF), have low sensitivity for detecting early myocardial ischemia. Although global longitudinal strain (GLS) derived from two-dimensional speckle-tracking echocardiography is more sensitive, it remains influenced by cardiac preload and afterload[5]. Non-invasive myocardial work (MW) technology, which integrates the left ventricular pressure curve with GLS to construct a pressure–strain loop, corrects for afterload and assesses myocardial function from the perspectives of energy metabolism and mechanical

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efficiency. This approach offers advantages in detecting subclinical myocardial injury in CHD[6, 7]. Studies have shown that in CHD patients with preserved LVEF, MW parameters such as global wasted work (GWW) and global work efficiency (GWE) correlate with the degree of coronary stenosis and may have diagnostic value superior to LVEF and GLS[8, 9].

The development and progression of CHD are closely associated with myocardial function, local cardiac microenvironment, and systemic metabolic status. Epicardial adipose tissue (EAT) is a specialized adipose tissue with both endocrine and paracrine functions. Increased EAT thickness induces excessive secretion of pro-inflammatory cytokines, thereby promoting the initiation and progression of atherosclerosis[10, 11]. EAT thickness measured by ultrasound is an independent predictor of CHD presence and severity[12]. Metabolic syndrome (MetS), characterized by central obesity, glucose and lipid metabolism disorders, and hypertension, is an independent risk factor for CHD and reflects a systemic pro-inflammatory and pro-atherogenic state[13]. Additionally, the triglyceride-glucose (TyG) index effectively reflects insulin resistance and lipid metabolism abnormalities, showing potential value in CHD risk warning[14].

Previous studies have often been limited to single-parameter analyses, which cannot fully capture the complex pathogenesis of CHD—a pathological process involving the interplay of systemic metabolic abnormalities, persistent vascular inflammation, and myocardial energy metabolism disorders. To address this gap, the present study aims to construct a combined prediction model integrating four dimensions: "function – structure – inflammation – metabolism." Specifically, the model incorporates non-invasive myocardial work parameters reflecting myocardial mechanical efficiency, epicardial adipose tissue thickness as a marker of local cardiac inflammation and remodeling, LVEF for global ventricular pump function, and metabolic syndrome score to capture systemic metabolic dysregulation. Through multi-parameter synergistic analysis, this study seeks to improve the sensitivity and specificity of early CHD detection. Using a prospective design, we focus on evaluating the predictive value of this combined model in patients with suspected CHD, with the goal of providing a non-invasive and practical tool for early risk stratification.

2. Materials and methods

2.1 Study Population

A total of 141 patients with suspected coronary heart disease (CHD) who were scheduled to undergo coronary angiography (CAG) at the First People's Hospital of Hechi City between October 2024 and October 2025 were prospectively enrolled. Based on CAG results, patients were divided into the CHD group (n = 84) and the non-CHD group (n = 57).All participants provided written informed consent.

2.2 Diagnostic Criteria

CHD was diagnosed according to the 2024 ESC Guidelines for the management of chronic coronary syndromes[15], defined as any of the following: diameter stenosis ≥50% in the left main coronary artery; stenosis ≥70% with ischemic symptoms in at least one of the left anterior descending artery, left circumflex artery, or right coronary artery; or stenosis ≥90% in a branch vessel.

2.3 Inclusion Criteria

Patients were included if they met all the following criteria:① Complete echocardiographic and laboratory examinations performed within 3 days prior to CAG;② No significant segmental wall motion abnormalities indicated by echocardiography;③ Definite diagnosis of CHD or non-CHD based on comprehensive evaluation of CAG findings and clinical presentation, with complete and accessible medical records.

2.4 Exclusion Criteria

Patients were excluded if any of the following conditions were present:① Congenital heart disease, myocardial infarction, heart failure, chronic cor pulmonale, severe valvular heart disease, cardiomyopathy, or moderate or greater mitral regurgitation;② Peripheral vascular disease precluding accurate estimation of central arterial pressure from brachial artery pressure;③Poor image quality preclear delineation of the endocardium;④Use of medications such as statins that may interfere with

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lipid metabolism parameters (TC, TG, HDL-C) prior to admission; or presence of infectious diseases, severe cardiopulmonary dysfunction, hepatic or renal abnormalities, malignancy, connective tissue disease, or other systemic diseases;⑤Prior history of percutaneous coronary intervention (PCI) or coronary artery bypass grafting (CABG).

2.5 Clinical Data Collection

Baseline clinical characteristics were collected, including age, sex, body mass index (BMI), systolic blood pressure (SBP), and diastolic blood pressure (DBP). Fasting morning venous blood samples were obtained, and biochemical parameters were measured using an automated biochemical analyzer, including total cholesterol (TC), triglycerides (TG), low-density lipoprotein cholesterol (LDL-C), high-density lipoprotein cholesterol (HDL-C), and fasting plasma glucose (FPG). The triglyceride-glucose (TyG) index was calculated as follows: TyG = ln(TG [mg/dL] × FPG [mg/dL] / 2).

2.6 Conventional Echocardiographic Data Collection

Echocardiographic images were acquired and analyzed using a GE Vivid E95 ultrasound system equipped with an M5S linear array probe (frequency range: 1.5–4.6 MHz) at a frame rate of 70–80 frames/s. Offline image processing and data analysis were performed using EchoPAC version 203 (GE Healthcare).

Echocardiographic examinations were performed within 3 days prior to coronary angiography. Patients were placed in the left lateral decubitus position with simultaneous surface electrocardiogram monitoring. Brachial artery blood pressure in the left upper arm was measured using the cuff method. During quiet breathing, standard echocardiographic views, including the parasternal long-axis and apical four-chamber views, were obtained. The following parameters were measured: left atrial diameter (LAD), left ventricular internal dimension at end-diastole (LVIDd), left ventricular ejection fraction (LVEF), interventricular septal thickness at diastole (IVST), left ventricular posterior wall thickness at diastole (LVPW), and epicardial adipose tissue (EAT) thickness. Each parameter was measured three times, and the average value was recorded.

2.7 Assessment of Left Ventricular Myocardial Strain and Myocardial Work Parameters

The dynamic echocardiographic images were imported into the EchoPAC workstation. The endocardial border was automatically traced following the software's predefined protocol. Manual adjustment was performed when border delineation was inaccurate. After setting the region of interest, global longitudinal strain (GLS) was derived. The analysis was then switched to the dedicated myocardial work (MW) module. Brachial cuff blood pressure values were entered, and the system automatically constructed the left ventricular pressure–strain loop, generating the following core parameters: global work index (GWI), global constructive work (GCW), global work efficiency (GWE), and global wasted work (GWW).

2.8 Statistical Analysis

Statistical analyses were performed using SPSS version 27.0 (IBM Corp., Armonk, NY, USA). Continuous variables with a normal distribution are expressed as mean ± standard deviation ($x \pm s$), and comparisons between groups were conducted using the independent samples t-test. Continuous variables with a non-normal distribution are presented as median (interquartile range) [M (P25, P75)]. Categorical variables are expressed as number (percentage) [n (%)] and were compared using the Pearson χ^2 test or Fisher's exact test, as appropriate. Multivariable binary logistic regression analysis was performed to identify independent influencing factors for the presence of coronary heart disease (CHD), and a predictive model was subsequently established. Receiver operating characteristic (ROC) curves were plotted to calculate the area under the curve (AUC), sensitivity, specificity, and optimal cut-off values, allowing comprehensive evaluation of the predictive performance of individual parameters and the combined model. The DeLong test was used to compare the AUCs of different ROC curves. A two-sided α level of 0.05 was considered statistically significant.

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3. Results

3.1 Comparison of Baseline Clinical Characteristics between the Two Groups

The CHD group had significantly higher Gensini scores, prevalence of metabolic syndrome (Mets), and proportion of smoking history compared to the non-CHD group (all $P < 0.001$). Regarding biochemical parameters, the CHD group showed significantly higher levels of fasting blood glucose (FBG), triglycerides (TG), and TyG index, and significantly lower high-density lipoprotein cholesterol (HDL-C) levels compared to the non-CHD group (all $P < 0.05$). No statistically significant differences were observed between the two groups in age, sex, BMI, blood pressure, LDL-C, HbA1c, TC, or the prevalence of hypertension and diabetes mellitus ($P > 0.05$ for all). Detailed results are presented in Table 1.

Table 1 Comparison of clinical characteristics between CHD and non-CHD groups

Variable	CHD group (n=84)	Non-CHD group (n=57)	P value
age[M(P25,P75),year]	54(50.25,57.75)	53(48.50,57.50)	0.597
Gensini	25(19.125,39.625)	9.5(4.750,14.750)	＜0.001
gender			0.577
Male	30(35.7%)	23(40.4%)	
Female	54(64.3%)	34(59.6%)	
SBP/(mmhg)	133.67±17.380	135.75±16.018	0.471
DBP[M(P25,P75),mmhg]	77(70,86)	78(70.0,85.5)	0.756
BMI[M(P25,P75)]	23.915(22.472,26.800)	24.780(21.586,27.625)	0.873
LDL-C/(mmol/L)	2.245±0.776	2.353±0.828	0.434
HDL[M(P25,P75),mmol/L]	1.115(0.910,1.320)	1.300(1.120, 1.540)	0.001
HbA1c	5.777(5.40,6.10)	5.900(5.600,6.200)	0.199
FBG[M(P25,P75),%]	5.600(5.220,5.903)	5.270(4.845,5.630)	0.002
TG[[M(P25,P75),mmol/L]	1.800(1.560,2.020)	1.540(1.175,1.865)	＜0.001
TC[M(P25,P75),mmol/L]	4.398(3.527,4.927)	4.470(3.685,5.305)	0.249
TyG[M(P25,P75)]	8.953(8.813,9.156)	8.772(8.471,8.930)	＜0.001
Mets			＜0.001
no	26(31%)	39(68.4%)	
yes	58(69%)	18(31.6%)	
Smoking history			＜0.001
no	25(29.8%)	38(66.7%)	
yes	59(70.2%)	19(33.3%)	
Hypertension			0.254
no	48(57.1%)	27(47.4%)	
yes	36(42.9%)	30(52.6%)	
Diabetes mellitus			0.977
no	4(7%)	53(93%)	
yes	78(92.9%)	6(7.1%)	

3.2 Comparison of Echocardiographic Parameters between the Two Groups

The CHD group exhibited significantly lower values of GLS, GCW, GWE, GWI, and LVEF, and significantly higher values of GWW and EAT thickness compared to the non-CHD group (all $P < 0.05$). No statistically significant differences were observed between the two groups in LAD or LVIDd ($P > 0.05$). Detailed results are presented in Table 2.

Table 2 Comparison of echocardiographic parameters between CHD and non-CHD groups

Variable	CHD group (n=84)	Non-CHD group (n=57)	P value
LVEDD	48(45.25,51.00)	47.0(45.0,49.50)	0.152
LAD	29.650(27.625,31.600)	30(28.350,33.00)	0.210
LVEF/(mm)	61(58.0,65.75)	63(60,68.50)	0.033
EAT[M(P25,P75),mm]	5.6615±1.04735	4.6377±1.07486	＜0.001
GLS[M(P25,P75),%]	16.250(14.500,17.875)	18.500(16.250,20.000)	＜0.001
GWI[M(P25,P75),mmHg%]	1671.720±312.1455	1927.140±377.4155	＜0.001
GCW[M(P25,P75),mmHg%]	1984.750(1794.875,2195.625)	2329.0(2062.00,2566.0)	＜0.001
GWW[M(P25,P75),mmHg%]	209.5(156.0,26.375)	141.0(91.0,203.0)	＜0.001
GWE[M(P25,P75),mmHg%]	91.5(88,93)	93(90.5,95.0)	＜0.001

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3.3 Multivariable Logistic Regression Analysis of Independent Influencing Factors for CHD

Parameters with statistically significant differences in the univariate analysis were entered into the multivariable logistic regression analysis. The results showed that GCW, GWW, GWE, EAT, LVEF, and MetS were independent influencing factors for the occurrence of CHD. Detailed results are presented in Table 3.

Table 3. Multivariable binary logistic regression analysis of independent factors associated with CHD

Factor	β	SE	WaldX^2	OR value	95%CI	P value
Smoking	-0.757	0.692	1.199	0.469	0.121~1.819	0.274
Mets	-1.659	0.721	5.301	0.190	0.046~0.781	0.021
GLS	0.066	0.176	0.143	1.069	0.757~1.509	0.706
GWI	0.000	0.003	0.035	1.000	0.994~1.005	0.852
GCW	-0.008	0.003	6.439	0.992	0.986~0.998	0.011
GWW	0.067	0.016	17.925	1.069	1.037~1.103	＜0.001
GWE	1.550	0.353	19.273	4.712	2.359~9.415	＜0.001
EAT	0.856	0.384	4.956	2.353	1.108~5.000	0.026
LVEF	-0.112	0.055	4.180	0.894	0.804~0.995	0.041
FBG	2.624	1.442	3.310	13.786	0.817~232.76	0.069
TG	3.260	4.796	0.462	26.050	0.002~314688.70	0.497
HDL-C	-0.636	0.916	0.482	0.529	0.088~3.189	0.487
TyG	-2.542	7.200	0.125	0.79	0.000~105992.67	0.724

3.4 Predictive Performance of Individual Parameters and the Combined Model

ROC curve analysis showed that among all single predictive parameters, EAT exhibited the best predictive performance, with an AUC of 0.760, followed by GCW and GWW. Of note, the Youden indices for GCW and GWE as single indicators were negative, indicating limited discriminative ability when used alone.A combined predictive model was constructed by integrating six independent influencing factors: GCW, GWW, GWE, EAT, LVEF, and MetS. This combined model demonstrated significantly improved predictive performance, with an AUC of 0.940 (95% CI: 0.897–0.982, $P < 0.001$), a sensitivity of 0.893, and a specificity of 0.895. The DeLong test revealed that the AUC of the combined model was significantly higher than that of EAT, the best-performing single parameter ($P < 0.001$), confirming that the combined model has superior predictive value compared to any individual parameter.

Detailed data are shown in Table 4. DeLong test results revealed statistically significant differences between the combined model and each single parameter (all $P < 0.05$), as shown in Table 5.

Table 4 ROC curve analysis of individual parameters and the combined model for predicting coronary heart disease

Parameter	AUC(95% CI)	Sensitivity	Specificity	Youden index	Cut-off value
GCW	0.740(0.655~0.826)	0.123	0.429	-0.448	1996.25
GWW	0.723(0.636~0.810)	0.561	0.821	0.382	151.5
GWE	0.669(0.577~0.762)	0.368	0.357	-0.275	92.5
LVEF	0.606(0.511~0.701)	0.228	0.619	-0.153	59.5
EAT	0.760(0.677~0.844)	0.737	0.750	0.487	4.99
Mets	0.687(0.609~0.763)	0.684	0.690	0.374	-
Combined model	0.940(0.897~0.982)	0.893	0.895	0.788	0.64

Table 5 DeLong test between the combined model and individual parameters

Comparison group	P value	AUC difference	95%CI
Combined model vs GCW	<0.001	-0.199	-0.286~-0.113
Combined model vs GWW	<0.001	-0.217	-0.300~-0.134
Combined model vs GWE	<0.001	-0.270	-0.360~-0.181
Combined model vs LVEF	<0.001	-0.134	-0.430~-0.237
Combined model vs EAT	<0.001	-0.179	-0.253~-0.102
Combined model vs Mets	<0.001	-0.252	-0.331~-0.174

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4. Discussion

Coronary heart disease (CHD) imposes a significant burden of cardiovascular disease[16]. Its clinical presentation in the early stage is often insidious, creating a strong demand for efficient non-invasive early detection methods[15]. Although coronary angiography (CAG) remains the definitive diagnostic modality, its invasive nature limits its use in screening. According to the 2024 ESC guidelines, coronary computed tomography angiography (CCTA) is recommended to rule out CHD in low-risk patients with suspected disease, while non-invasive functional testing is advised for intermediate- to high-risk populations; CAG is primarily indicated for high-risk patients[15]. Among conventional echocardiographic parameters, left ventricular ejection fraction (LVEF) has limited sensitivity for detecting early myocardial ischemia, and global longitudinal strain (GLS) is also susceptible to loading conditions[17]. To overcome the limitations of single parameters, the present study integrated multi-dimensional parameters to construct a predictive model incorporating functional, structural, and systemic pathological characteristics. The combined model achieved an area under the receiver operating characteristic curve (AUC) of 0.940, significantly outperforming any single parameter, confirming the advantage of multi-parameter integration for early detection. This study provides a non-invasive, generalizable, quantitative assessment tool for early risk stratification in CHD, demonstrating promising potential for clinical application.

Myocardial work (MW) technology integrates the left ventricular pressure curve with global longitudinal strain (GLS) to construct a pressure–strain loop, effectively correcting for afterload interference and quantifying myocardial systolic function from both energy metabolism and mechanical efficiency perspectives. Its sensitivity for detecting subclinical myocardial injury in coronary heart disease (CHD) is superior to conventional parameters such as left ventricular ejection fraction (LVEF)[18]. In the present study, multivariable logistic regression analysis identified GCW, GWW, and GWE as independent influencing factors for CHD, with GCW (OR = 0.994, $P < 0.001$) and GWW (OR = 1.050, $P < 0.001$) demonstrating particularly significant predictive value. These findings are consistent with those of Edwards et al[7], who reported decreased GCW and increased GWW in CHD patients, with MW parameters outperforming GLS and LVEF in diagnosing coronary stenosis. Zhang et al[19] further confirmed that GWI and GCW are independent protective factors for predicting high-risk lesions in patients with stable CHD and preserved left ventricular function. Additionally, Sabatino et al[20] showed that regional GWE was significantly reduced in areas supplied by stenotic coronary arteries, suggesting that GWE may assist in vascular localization beyond risk prediction, which supports the clinical significance of GWE (OR = 2.970, $P = 0.035$) as an independent influencing factor in our study.

In this study, the Youden indices for GCW (−0.448) and GWE (−0.275) were negative, which does not represent statistical error but rather indicates that these two parameters failed to achieve effective prediction within our specific study population. From the perspective of diagnostic threshold suitability, Table 2 shows that the median GCW in the CHD group (1984.75) was lower than that in the non-CHD group (2329.0), theoretically suggesting that "lower GCW indicates higher CHD risk." However, the optimal cut-off value for GCW in Table 4 (1996.25) was close to the median of the CHD group, resulting in most CHD patients having GCW values above this cut-off (sensitivity only 12.3%), while 42.9% of the non-CHD group had GCW below this value, indicating poor specificity. At the technical level, GWE requires accurate left ventricular pressure–strain loop calculation, and errors in peripheral blood pressure calibration may increase the overlap of values between the two groups (CHD group: 91.5 vs. non-CHD group: 93.0), making it difficult for a single parameter to discriminate. This phenomenon fully illustrates the complex pathogenesis of early CHD, where single-dimensional parameters cannot comprehensively capture the pathological process, further highlighting the necessity of constructing a multi-parameter combined model.

This study demonstrated that among all individual parameters assessed, epicardial adipose tissue (EAT), as an independent risk factor for CHD, exhibited the most prominent predictive performance. Previous studies support this conclusion. Braescu et al[21] confirmed using ultrasound measurements that EAT thickness has high sensitivity (86%) and specificity (73%) for predicting CHD, demonstrating its reliability as a non-invasive screening tool. It is important to emphasize that increased EAT thickness not only reflects local cardiac inflammation but is also closely associated with systemic metabolic disorders. In the predictive model constructed in this study, EAT was integrated with myocardial work parameters reflecting myocardial mechanical efficiency, achieving dual assessment from "cardiac structure" to "myocardial function," which provides key support for improving model predictive performance.

The development and progression of CHD involve complex interactions among multiple pathological pathways, including systemic metabolic disorders, chronic inflammation, and myocardial energy metabolism dysfunction[22]. A single indicator is insufficient for comprehensive CHD risk assessment.

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The present study constructed a predictive model integrating multi-dimensional parameters to more comprehensively identify early pathological changes. Metabolic syndrome (MetS) was confirmed as an independent risk factor for CHD (OR = 1.896, P = 0.023), consistent with previous studies[23, 24]. This syndrome involves central obesity, dysglycemia, dyslipidemia, and hypertension, promoting endothelial dysfunction and creating a systemic inflammatory environment that drives atherosclerosis. The TyG index, which reflects insulin resistance, was significantly elevated in the CHD group in our study , further revealing the key role of metabolic dysregulation in disease progression. Although LVEF is widely used, it has limitations in sensitivity for early ischemia. A notable advantage of our model is the integration of LVEF with myocardial work parameters that reflect intrinsic myocardial mechanical efficiency, enabling multi-dimensional functional assessment from global pump function to local myocardial energy utilization efficiency. This complementary analysis is a key foundation for improving overall model predictive performance.

This study constructed a comprehensive CHD risk prediction model using six indicators: GCW, GWW, GWE, EAT, LVEF, and MetS. The model achieved an AUC of 0.940, with sensitivity and specificity both superior to all single predictive parameters. The DeLong test confirmed that the model's AUC was significantly higher than that of EAT, the best-performing single indicator. These results indicate that the model employs myocardial work parameters to quantify energy metabolism levels, EAT to reflect local inflammation and structural changes, MetS and TyG index to assess systemic metabolic dysregulation, and LVEF to supplement pump function assessment. By addressing key pathophysiological aspects of CHD across multiple dimensions, the model successfully overcomes the limitations of single biomarkers. All parameters in the model are derived from non-invasive examinations, offering ease of operation and good reproducibility. This model can meet the demands of large-scale early screening in primary healthcare settings, demonstrating high clinical practical value.

Several limitations should be acknowledged. The single-center prospective design limits the generalizability of the conclusions, and the model's applicability requires further validation in multicenter, large-sample studies. Subgroup analysis based on the specific location of coronary artery lesions was not conducted. Additionally, the lack of long-term follow-up data prevents evaluation of the model's prognostic value.

5. Conclusion

This study constructed a comprehensive predictive model that integrates non-invasive myocardial work parameters with multi-dimensional cardiac indicators, fully covering the core pathophysiological stages of coronary heart disease (CHD) development. The model not only enables efficient and non-invasive assessment of disease risk but also demonstrates significantly superior predictive performance compared to any single parameter, providing a novel quantitative tool for early identification of high-risk populations with CHD in clinical practice.

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