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A multimodal nomogram for predicting disease progression in diabetic patients with coronary artery disease: integrating clinical, ultrasound, and angiographic data

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Abstract

Objective The long-term prognosis of diabetic patients with coronary artery disease (CAD) is influenced by various clinical variables and biomarkers. This study aimed to develop and validate a prognostic model that integrates clinical, echocardiographic, and angiographic data to predict disease progression.

Methods We retrospectively analyzed 396 diabetic CAD patients with a 3-year follow-up starting from their first coronary angiography. Outcome variables included recurrent myocardial infarction, unstable angina rehospitalization, heart failure, ischemic stroke, cardiovascular death, and all-cause death. Non-progression was defined as the absence of these events. Variables included clinical data, echocardiographic parameters, coronary angiography results, and biomarkers. A multivariate Cox regression model was developed, incorporating key factors (coronary lesion number, myocardial infarction history, ejection fraction, and creatinine).

Results Multivariate analysis identified the number of obstructed coronary arteries, history of myocardial infarction, ejection fraction, and creatinine level as independent predictors of disease progression. The model showed good predictive performance, with AUC values of 0.742, 0.782, and 0.816 at 3, 6, and 9 months, respectively. The C-index was 0.669 (95% CI: 0.5959–0.7196) in the training set and 0.695 (95% CI: 0.5781–0.7436) in the validation set, reflecting consistent predictive performance. Calibration curves showed excellent agreement between predicted and observed outcomes.

Conclusion We developed and validated a practical nomogram integrating clinical, biochemical, and imaging data to predict short-term disease progression in diabetic patients with CAD. This tool may assist clinicians in early risk stratification and individualized management planning.

Keywords Diabetic coronary artery disease, Disease progression, Coronary lesion, Creatinine, Echocardiography

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Introduction

Coronary artery disease (CAD) is a leading cause of death worldwide, particularly among diabetic patients [1–3]. Diabetes mellitus exacerbates the progression of CAD, leading to increased rates of cardiovascular events and mortality [4]. The inadequate control of risk factors in diabetic patients results in a higher burden of atherosclerotic plaques, causing ischemia and subsequent acute cardiac injury [5]. Prolonged exposure to hyperglycemia and dyslipidemia causes progressive and irreversible damage to the coronary vasculature, further complicating the management of CAD in diabetic patients [6].

Despite advancements in diagnostic and therapeutic approaches, such as the development of novel antiplatelet therapies and the widespread use of percutaneous coronary interventions (PCI) [7], predicting the progression of CAD in diabetic patients remains a significant challenge due to the complex interplay of clinical, imaging, and genetic factors. Traditionally, CAD risk stratification relies on clinical parameters such as age, blood pressure, cholesterol levels, and glycemic control [5, 8]. However, these factors alone are insufficient to predict disease progression, particularly in diabetic patients where traditional risk factors may not fully capture the disease's complexity [9, 10]. Ultrasound imaging, including measures of coronary flow, plaque characteristics, and vascular reactivity, has shown promise in providing valuable insights into the structural and functional aspects of CAD [11]. Furthermore, angiographic data, such as the extent of coronary artery stenosis, is a key determinant of disease severity and prognosis [12]. However, these modalities are typically used in isolation, and a comprehensive approach that integrates multiple types of data is lacking.

Therefore, the most effective approach to mitigating the impact of CAD in this population is through the development of a comprehensive prognostic model that restores the ability to predict disease progression and thereby prevents further cardiovascular events [13, 14]. Timely prediction and intervention decrease cardiac morbidity and mortality and are the primary therapeutic strategies for treating CAD in diabetic patients [15]. However, current models often rely predominantly on a single dimension of patient information—such as clinical scores based largely on symptomatology and limited laboratory indices, or angiographic grading systems focusing solely on anatomical lesion complexity—without accounting for other critical factors. For instance, commonly used clinical prediction tools emphasize clinical and laboratory parameters but fail to incorporate detailed imaging findings, while imaging-based scoring systems like the SYNTAX score highlight anatomical severity but overlook metabolic or functional parameters [16]. This narrow focus leads to an incomplete understanding of disease progression mechanisms and can leave clinicians uncertain about optimal intervention timing or the selection of appropriate therapeutic strategies [17]. Despite continuous refinements in risk stratification methodologies, a truly comprehensive prognostic tool that seamlessly integrates clinical, echocardiographic, angiographic, and biomarker data remains elusive. Such a multimodal model would not only deepen insight into disease dynamics in diabetic CAD patients but also guide more effective and personalized clinical decision-making. Thus, there is a great need to define novel mechanisms of disease progression that could be targeted for the treatment of CAD in diabetic patients. Recent advances in statistical prognostic modeling offer exciting opportunities to improve disease prediction by combining multimodal data [18–20]. This approach has been particularly useful in creating predictive models that incorporate clinical, imaging, and angiographic data to better assess the risk of disease progression in various cardiovascular conditions [21, 22].

In this study, we propose a multimodal nomogram that integrates clinical, ultrasound, and angiographic data to predict disease progression in diabetic patients with CAD.

Each data source provides a unique dimension of prognostic insight: clinical metrics (such as past myocardial infarction history or comorbidities) capture baseline risk and systemic disease burden; echocardiographic parameters (like ejection fraction and ventricular dimensions) reflect cardiac function and remodeling; and angiographic data (including the number and severity of obstructed coronary arteries) directly quantify the structural extent of atherosclerotic disease. By combining these complementary domains, we create a more holistic and precise representation of an individual patient's cardiovascular risk. Our analysis reveals a previously unrecognized multimodal activation of the prognostic model, highlighting that such integration reduces the uncertainty of disease progression, decreases the risk of cardiovascular events, and improves the accuracy of risk prediction. This comprehensive approach holds promise for informing tailored therapeutic strategies and ultimately improving outcomes in diabetic patients with CAD.

Materials and methods

Study population

A total of 453 diabetic patients with coronary artery disease (CAD) who underwent selective coronary angiography (CAG) from June 2022 to November 2023 were initially included in this retrospective cohort study. Patient data were collected from the Department of Cardiology, the Fourth Affiliated Hospital of China Medical University. The exclusion criteria were as follows: patients with concurrent cardiac conditions (e.g., valvular heart disease, pericarditis, or significant systemic

diseases affecting prognosis) ($n = 23$); those with a history of coronary artery bypass grafting (CABG) or percutaneous coronary intervention (PCI) within the past 6 months ($n = 10$); patients unable to comply with follow-up or with incomplete follow-up data, as well as those with severe cognitive dysfunction or other psychological conditions that might interfere with participation ($n = 9$); and patients who could not provide informed consent ($n = 15$). As a result, 396 patients met the inclusion criteria and were included in the final analysis. The non-progression group consisted of patients who did not experience any of these events during the follow-up period. Of these 396 patients, 70% were randomly assigned to the training set and 30% to the validation set to develop and validate the prognostic model. The progression group included patients with recurrent myocardial infarction, unstable angina requiring rehospitalization, heart failure, ischemic stroke, cardiovascular death, or all-cause mortality. The non-progression group consisted of patients who did not experience any of these events during the follow-up period. (Fig. 1).

Model construction and assessment

To predict disease progression in diabetic patients with coronary artery disease (CAD), we developed a multimodal nomogram integrating clinical, ultrasound, and angiographic data. Clinical data included demographic information (age, sex, duration of diabetes, comorbidities), laboratory test results (e.g., blood glucose levels, lipid profile), and clinical history (e.g., previous myocardial infarction, hypertension, smoking history). Ultrasound data include ascending aorta diameter, ventricular septum thickness, left ventricular systolic diameter, left ventricular diastolic diameter, left ventricular stroke volume, and EF value. Our EF is measured using the Simpson biplane method. The ultrasound equipment used in our study was the Philips EPIQ CVx cardiac ultrasound system (Philips Healthcare, Andover, MA, USA). Angiographic data included the number and location of coronary artery lesions (e.g., left anterior descending artery, circumflex artery, right coronary artery). Cox proportional hazards regression analysis was employed to identify prognostic factors for disease progression. Variables with $P \leq 0.10$ in univariate analysis were included in multivariate Cox regression analysis. To ensure robust predictor selection and minimize the risk of overfitting, stepwise selection was applied during the multivariate analysis. This method helps identify the most relevant variables while balancing model complexity and predictive accuracy. Based on the multivariable analysis results, we constructed prognostic models for 3-month, 6-month, and 9-month outcomes. The model's performance was evaluated using the area under the receiver operating characteristic curve (AUC) and concordance index (C-index) to assess the goodness-of-fit and discriminatory ability of the nomogram. Calibration of the nomogram was assessed using calibration curves, and decision curve analysis (DCA) was performed to evaluate its clinical utility. Kaplan-Meier methods and log-rank tests were used to calculate and compare differences in disease progression between groups. All internal validation was performed using bootstrapping with 200 resamples to assess the robustness and stability of the selected variables and model performance.

Statistical analyses

Statistical analyses were performed using R (version 4.3.1) for Windows. Baseline characteristics were analyzed using the compareGroups package, which automatically selects appropriate statistical tests based on variable type and distribution—typically applying t-tests or Mann–Whitney U tests for continuous variables, and chi-square or Fisher' s exact tests for categorical variables. The summary table, including descriptive statistics and *p*-values for group comparisons, was formatted using the CBCgrps package. The nomogram, decision curve analysis, and calibration curves were generated using the rms package in R. Survival analysis was conducted using Kaplan-Meier methods, and differences in disease progression were compared using the log-rank test, with the survminer and survival packages in R. The concordance index (C-index) was selected as the primary metric for model performance, as it reflects the model's ability to correctly rank patients by risk while accounting for censoring and varying follow-up times, which are inherent in time-to-event data. A *P*-value of < 0.05 was considered statistically significant.

Rusult

Patient characteristics

A total of 396 diabetic patients with coronary artery disease (CAD) were included in the analysis, all of whom had complete baseline clinical, laboratory, and ultrasound data (Fig. 1). The clinical characteristics of these patients are summarized in Table 1.

All *p*-values comparing the training and validation cohorts were greater than 0.05, indicating that there were no statistically significant differences in baseline characteristics between the two groups. The median age of the cohort was 65.00 years (IQR: 57.75–70.25), with 63.38% ($n = 251$) being male and 36.62% ($n = 145$) female. Hypertension was present in 76.26% of patients ($n = 302$), and 27.27% ($n = 108$) had a history of myocardial infarction.

Additionally, 42.93% ($n = 170$) reported a history of smoking. Multi-vessel disease was prevalent, with 70.20% ($n = 278$) exhibiting lesions in multiple coronary arteries.

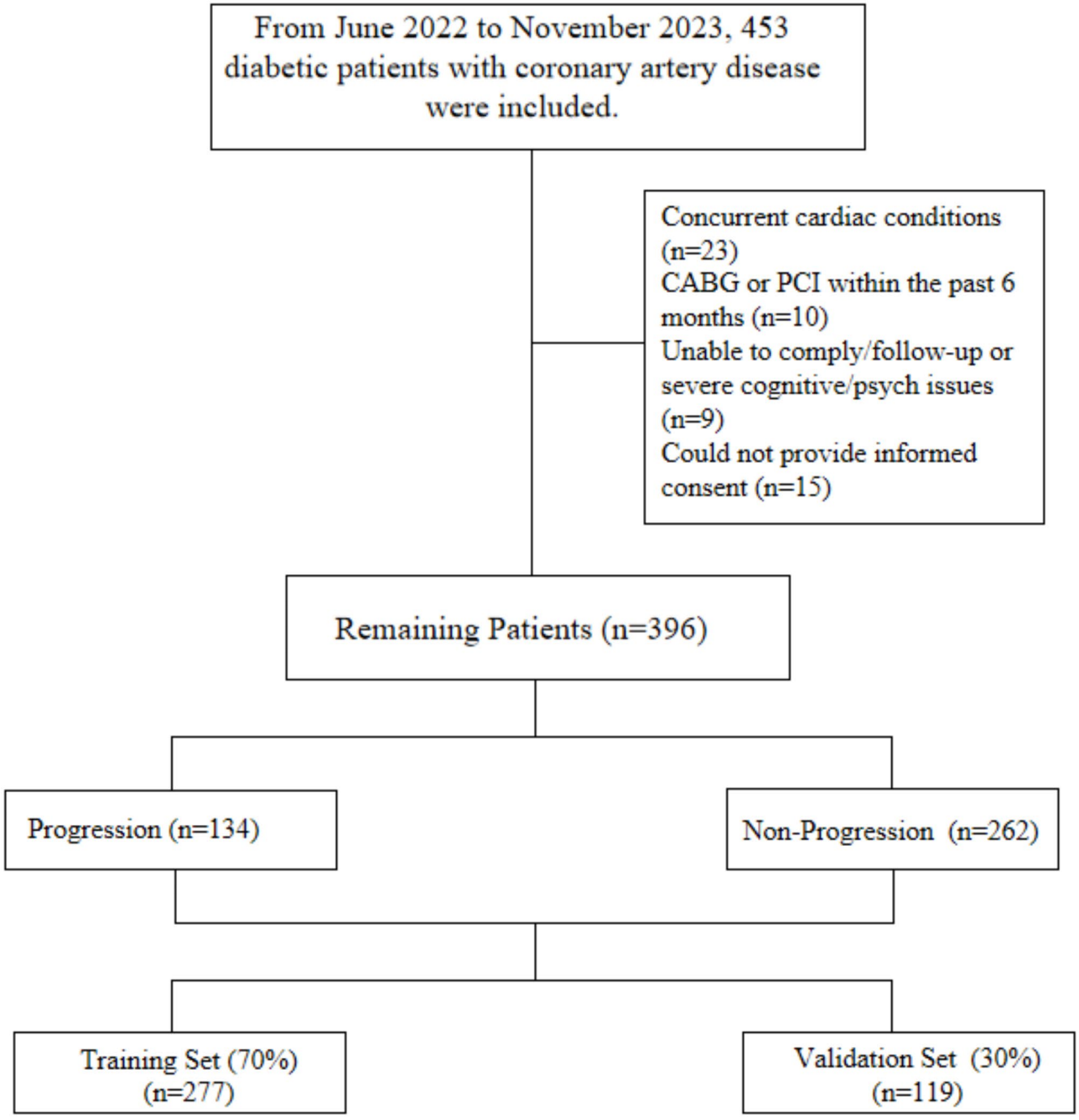

Fig. 1 Flowchart

Prognostic factors for disease progression

Univariate Cox regression analysis identified eight baseline parameters significantly associated with disease progression (DP) at a threshold of $P < 0.2$, including the number of coronary lesions, history of myocardial infarction, serum potassium levels, creatinine (Cr), blood urea nitrogen (BUN), ejection fraction (EF), left ventricular end-systolic diameter (LVDs), and left ventricular end-diastolic diameter (LVDd).

Multivariate Cox regression analysis ($N = 396$) further revealed four independent predictors of DP: the number of coronary lesions, history of myocardial infarction, creatinine (Cr), and ejection fraction (EF) (Table 2; Fig. 2). We assessed potential multicollinearity among these predictors using Variance Inflation Factor (VIF) analysis, with all values < 2 (lesions: 1.07; MI: 1.02; Cr: 1.41; EF: 1.25), indicating no significant collinearity. Additionally, pairwise interaction terms were tested and found to be non-significant. These predictors reflect the combined

Table 1 Baseline characteristics of patients with diabetes mellitus combined with coronary heart disease

	[ALL] *N* = 396	Validation *N* = 119	Train *N* = 277	*p*.overall
genders:				1
females	145 (36.62%)	44 (36.97%)	101 (36.46%)	
Male	251 (63.38%)	75 (63.03%)	176 (63.54%)	
years	65.00 [57.75;70.25]	64.00 [57.00;70.00]	65.00 [58.00;71.00]	0.293
Progression:				0.312
no	284 (71.72%)	90 (75.63%)	194 (70.04%)	
yes	112 (28.28%)	29 (24.37%)	83 (29.96%)	
time	36.00 [15.75;36.00]	36.00 [36.00;36.00]	36.00 [12.00;36.00]	0.131
AO	34.00 [32.00;36.00]	34.00 [32.00;36.00]	34.00 [32.00;36.00]	0.739
IVS	9.00 [8.00;10.00]	9.00 [8.00;10.00]	9.00 [8.00;10.00]	0.323
LVDd	48.00 [45.00;51.00]	48.00 [45.00;51.00]	48.00 [45.00;51.00]	0.87
LVDs	31.00 [27.75;35.00]	32.00 [28.00;36.00]	30.00 [27.00;35.00]	0.219
SV	49.00 [43.00;58.00]	50.00 [44.00;58.50]	49.00 [43.00;58.00]	0.452
EF:				0.886
< 50%	37 (9.34%)	12 (10.08%)	25 (9.03%)	
≤ 50%	359 (90.66%)	107 (89.92%)	252 (90.97%)	
TC	3.33 [2.78;4.23]	3.29 [2.76;4.21]	3.33 [2.79;4.28]	0.782
HDL	0.80 [0.69;0.94]	0.81 [0.66;0.94]	0.80 [0.70;0.94]	0.896
LDL	1.88 [1.37;2.59]	1.81 [1.37;2.62]	1.88 [1.36;2.59]	0.896
CK	70.50 [49.00;111.25]	72.00 [50.50;115.50]	70.50 [49.00;107.00]	0.341
CKMB	16.40 [12.10;21.40]	16.40 [11.95;19.55]	16.40 [12.40;21.60]	0.396
BUN	5.30 [4.41;6.26]	5.30 [4.48;6.12]	5.30 [4.39;6.38]	0.898
Cr	69.00 [60.00;79.05]	69.00 [60.00;77.00]	69.00 [60.00;81.00]	0.466
K	3.96 [3.74;4.14]	3.94 [3.68;4.16]	3.96 [3.77;4.13]	0.553
Height	168.00 [162.00;171.25]	168.00 [162.00;170.00]	168.00 [162.00;172.00]	0.739
Weight	70.00 [65.00;75.00]	70.00 [65.00;74.00]	70.00 [65.00;75.00]	0.638
Hypertension:				0.562
no	94 (23.74%)	31 (26.05%)	63 (22.74%)	
yes	302 (76.26%)	88 (73.95%)	214 (77.26%)	
smoke:				0.328
no	226 (57.07%)	63 (52.94%)	163 (58.84%)	
yes	170 (42.93%)	56 (47.06%)	114 (41.16%)	
Alcoholism:				0.638
no	333 (84.09%)	98 (82.35%)	235 (84.84%)	
yes	63 (15.91%)	21 (17.65%)	42 (15.16%)	
MI:				0.467
no	288 (72.73%)	90 (75.63%)	198 (71.48%)	
yes	108 (27.27%)	29 (24.37%)	79 (28.52%)	
Lesion:				0.235
1	118 (29.80%)	30 (25.21%)	88 (31.77%)	
>=3	278 (70.20%)	89 (74.79%)	189 (68.23%)	

Note: Values are presented as median (interquartile range) or *n* (%), as appropriate. p.overall values represent statistical comparisons between the training and validation sets. AO: Aortic Diameter; IVS: Interventricular Septum; LVDd: Left Ventricular Diastolic Diamete; LVDs: Left Ventricular Systolic Diameter; SV: Stroke Volume; EF: Ejection Fraction; TC: Total Cholesterol; HDL: High-Density Lipoprotein; LDL: Low-Density Lipoprotein; CK: Creatine Kinase; CKMB: Creatine Kinase Isoenzyme MB; BUN: Blood Urea Nitrogen; Cr: Creatinine; K: Potassium; MI: History of myocardial infarction; smoke: smoking history; Alcoholism: drinking history; Lesion: Number of coronary lesions

impact of anatomical, functional, and biochemical factors on disease progression in diabetic patients with CAD.

Development and validation of the prognostic nomogram

Based on the four independent predictors identified in the multivariate Cox regression, a nomogram was constructed to estimate the 3-, 6-, and 9-month disease progression risk in diabetic patients with CAD (Fig. 3A).

The nomogram provides a practical tool for clinicians to identify high-risk patients and tailor therapeutic strategies accordingly.

Patients were stratified into high- and low-risk groups based on the median score, with the low-risk group

Table 2 Univariate and multivariate COX regression of factors associated with disease progression

characteristics	Univariate COX regression			multivariate COX regression			VIF
	OR	CI	P	OR	CI	P	
Lesion	1.997	1.171–3.404	0.011	2.259	1.304–3.915	0.004	1.07
MI	1.657	1.062–2.587	0.026	1.623	1.035–2.544	0.035	1.02
Alcoholism	0.812	0.431–1.532	0.521				
smoke	1.11	0.719–1.713	0.638				
Hypertension	1.003	0.601–1.674	0.99				
K	2.233	1.16–4.302	0.016				
Cr	1.01	1.006–1.013	< 0.001	1.006	1.002–1.01	0.002	1.41
BUN	1.116	1.069–1.165	< 0.001				
CKMB	0.998	0.989–1.007	0.622				
CK	0.999	0.998–1.001	0.31				
LDL	0.838	0.666–1.054	0.131				
HDL	0.506	0.18–1.426	0.198				
TC	0.875	0.726–1.054	0.161				
EF	0.259	0.153–0.439	< 0.001	0.265	0.148–0.477	< 0.001	1.25
SV	1.015	0.997–1.033	0.104				
LVDs	1.049	1.02–1.078	0.001				
LVDd	1.066	1.03–1.104	< 0.001				
IVS	1.064	0.92–1.23	0.402				
AO	1.005	0.945–1.07	0.866				
years	1.004	0.983–1.026	0.699				
genders	1.104	0.703–1.733	0.668				

Note: AO: Aortic Diameter; IVS: Interventricular Septum; LVDd: Left Ventricular Diastolic Diamete; LVDs: Left Ventricular Systolic Diameter; SV: Stroke Volume; EF: Ejection Fraction; TC: Total Cholesterol; HDL: High-Density Lipoprotein; LDL: Low-Density Lipoprotein; CK: Creatine Kinase; CKMB: Creatine Kinase Isoenzyme MB; BUN: Blood Urea Nitrogen; Cr: Creatinine; K: Potassium; MI: History of myocardial infarction; smoke: smoking history; Alcoholism: drinking history; Lesion: Number of coronary lesions; CI: Confidence Interval; OR: Odds Ratio; VIF: Variance Inflation Factor;

VIF values were calculated to assess multicollinearity among predictors retained in the final multivariate model. All VIF values were < 2, indicating no significant multicollinearity

demonstrating significantly better survival probabilities (Fig. 3B).

The nomogram's predictive accuracy was evaluated in both the training and validation sets. In the training set, the model demonstrated good discriminative ability, with AUC values of 0.680 (3 months), 0.702 (6 months), and 0.705 (9 months) (Fig. 4A).

In the validation set, the AUC values were 0.742, 0.782, and 0.816, respectively, confirming the model's external generalizability (Fig. 4B).

The concordance index (C-index) was 0.669 (95% CI: 0.5959–0.7196) in the training set and 0.695 (95% CI: 0.5781–0.7436) in the validation set.

Calibration curves indicated excellent agreement between predicted and observed outcomes in both training and validation sets at 3, 6, and 9 months (Fig. 5A-F).

Discussion

In this study, we developed and validated a multimodal nomogram that integrates clinical, ultrasound, and angiographic data to predict disease progression in diabetic patients with coronary artery disease (CAD). Our findings demonstrated that a comprehensive approach incorporating the number of obstructed coronary arteries, previous myocardial infarction history, creatinine (Cr) levels, and left ventricular ejection fraction (EF) significantly enhances the accuracy of short- and intermediate-term risk predictions. By contrast, conventional risk stratification models that rely solely on clinical or laboratory parameters frequently fail to fully capture the complex interplay of pathophysiological factors contributing to CAD progression in the diabetic population [23, 24].

Studies have shown that diabetes promotes widespread intimal thickening, impaired collateral vessel formation, and rapid plaque progression, resulting in a greater degree of coronary artery obstruction [25, 26]. Cardiac function is largely dependent on the preservation of coronary vascular function [27]. The number of obstructed coronary arteries reflects the anatomical severity and extent of atherosclerosis [28, 29]. Therefore, the degree of coronary artery obstruction serves as an early and sensitive indicator of disease severity and progression in this population. A history of myocardial infarction (MI) indicates prior plaque rupture and myocardial injury [29], and diabetic patients are more likely to experience silent or atypical MIs due to autonomic neuropathy, resulting in delayed diagnosis and increased myocardial damage [30]. Moreover, diabetes is associated with impaired infarct healing and adverse remodeling, compounding the risk of future cardiovascular events [31]. Together, these factors

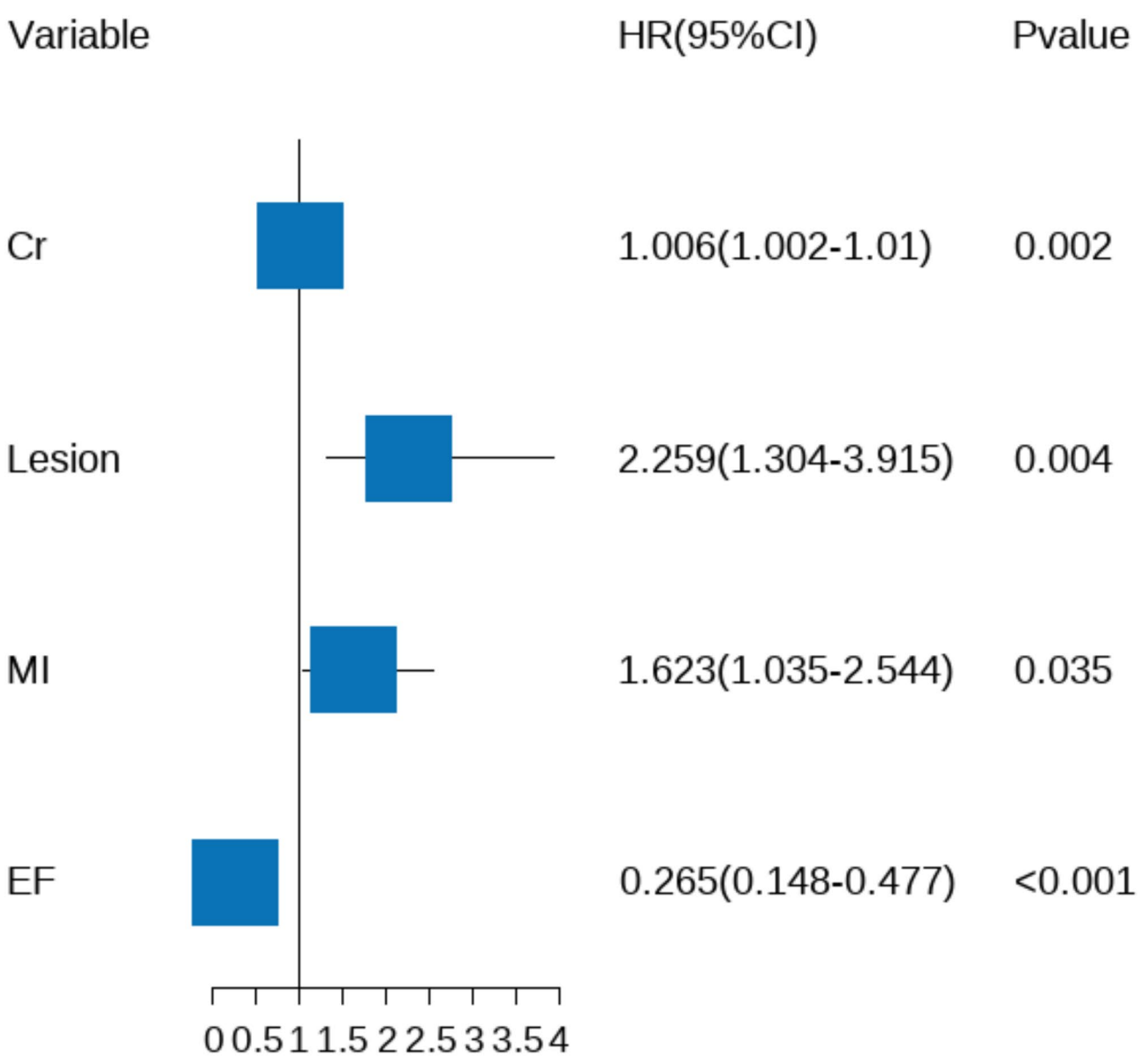

Fig. 2 Forest plot of multivariable cox regression analysis. This forest plot illustrates the hazard ratios (HR) and 95% confidence intervals (CI) for various cardiovascular risk factors assessed through multivariable Cox regression. The variables included are creatinine (Cr), number of coronary artery lesions (Lesion), history of myocardial infarction (MI), and ejection fraction (EF). Each factor's impact on cardiovascular outcomes is represented by the (square), with the area of the square corresponding to the factor's weight in the analysis, and the (line) extending to the left and right indicating the 95% CI. The (vertical line) at 1.0 serves as a reference for a neutral effect. Creatinine (Cr): The HR is 1.006 with a 95% CI of 1.002 to 1.01, suggesting a minor but statistically significant increase in risk with higher creatinine levels ($P = 0.002$). Coronary Artery Lesions (Lesion): With an HR of 2.259 and a 95% CI of 1.304 to 3.915, a higher number of lesions is associated with a significant increase in cardiovascular risk ($P = 0.004$). Myocardial Infarction (MI): Patients with a history of MI have an HR of 1.623, with a 95% CI of 1.035 to 2.544, indicating an increased risk compared to those without MI ($P = 0.035$). Ejection Fraction (EF): A lower EF is associated with a significantly reduced risk, as shown by an HR of 0.265 and a 95% CI of 0.148 to 0.477 ($P < 0.001$)

make prior MI a powerful marker of cumulative ischemic burden and disease instability in diabetic patients [32, 33]. Elevated serum creatinine levels serve as a marker of renal dysfunction, a common comorbidity in diabetes that promotes systemic inflammation, oxidative stress, and vascular calcification—factors that exacerbate coronary pathology. Reduced left ventricular ejection fraction represents impaired myocardial contractility, often a downstream consequence of chronic ischemia, infarction, and diabetic cardiomyopathy [34]. These predictors are not isolated but pathophysiologically linked.

Multivessel disease increases the likelihood of significant ischemia and infarction, which in turn impairs ventricular function (lower EF) [34]. Renal dysfunction not only amplifies systemic atherogenic processes but also worsens myocardial remodeling and neurohormonal

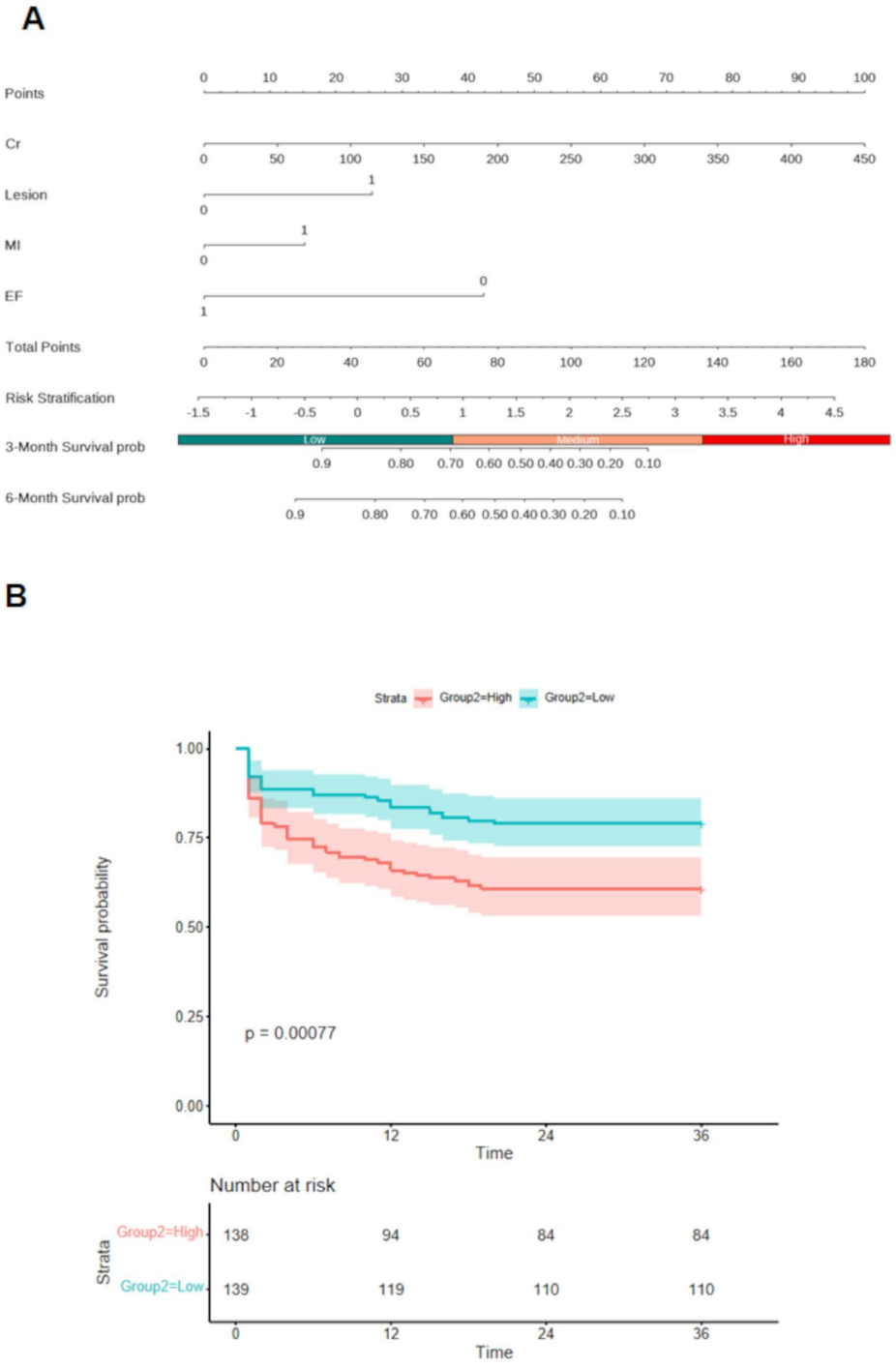

Fig. 3 **(A)** Nomogram integrating the number of obstructed coronary arteries (Lesion, defined as ≥ 50% stenosis), previous myocardial infarction (MI), creatinine (Cr) level, and left ventricular ejection fraction (EF) to estimate the 3-, 6-, and 9-month risk of disease progression in diabetic patients with coronary artery disease (CAD). To use the nomogram, draw vertical lines from each variable to the corresponding points scale, sum these points to obtain a total score, and then reference the probability scale at the bottom to determine the individual patient's predicted risk. **(B)** Kaplan–Meier survival curves for patients stratified into high- and low-risk groups according to the median nomogram-derived risk score. Patients in the high-risk group showed a significantly higher incidence of disease progression compared to those in the low-risk group (log-rank test, $P = 0.00077$)

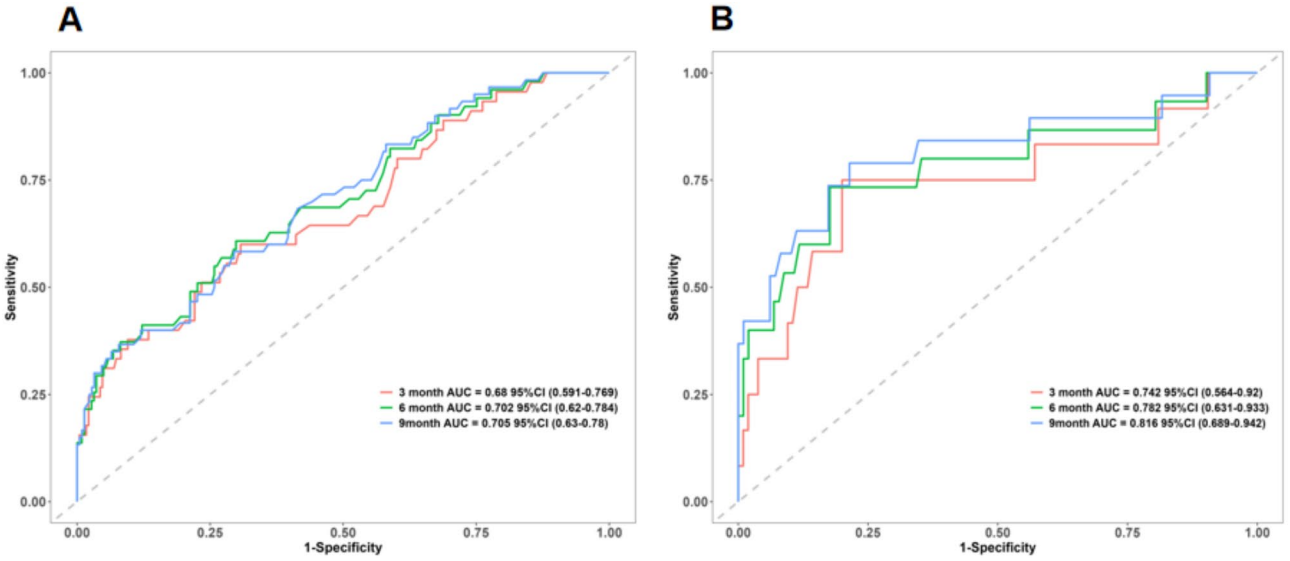

Fig. 4 **(A)** Receiver Operating Characteristic (ROC) curves for the training set evaluating the nomogram's performance in predicting disease progression at 3, 6, and 9 months. The robust Area Under the Curve (AUC) values indicate strong discriminative ability of the model. **(B)** ROC curves for the validation set demonstrating similarly strong AUC values at 3, 6, and 9 months, confirming the external generalizability and reproducibility of the nomogram's predictive performance

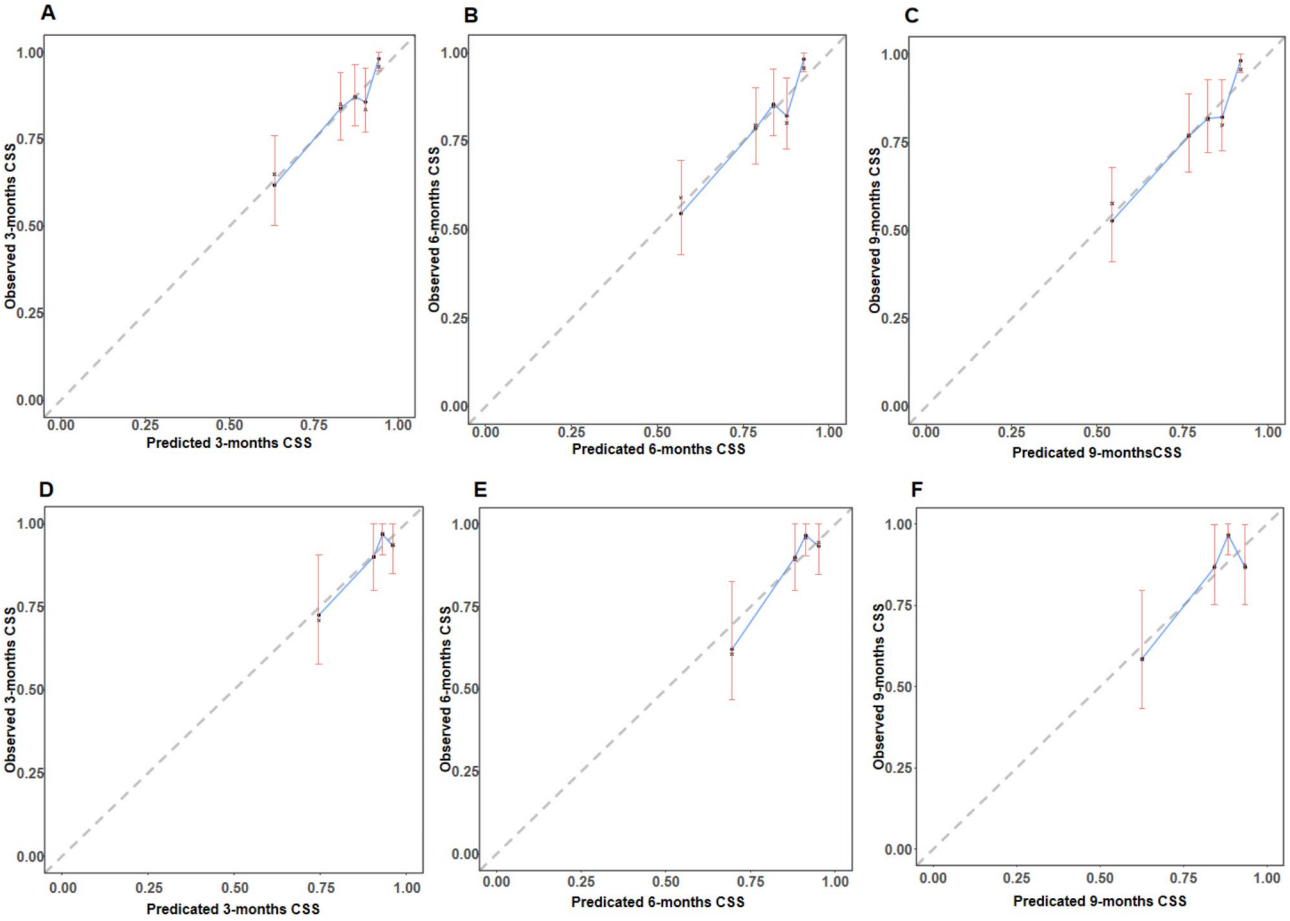

Fig. 5 (**A**–**C**) Calibration curves for the training set at 3, 6, and 9 months, respectively, illustrating close agreement between the predicted probabilities of disease progression and the observed outcomes. (**D**–**F**) Calibration curves for the validation set at 3, 6, and 9 months, respectively, confirming that the nomogram's predictive accuracy and reliability are maintained in an independent cohort

activation [35]. A prior MI often coexists with impaired EF and extensive coronary lesions [36]. In diabetic patients, these interactions are further intensified by microvascular disease, metabolic dysregulation, and chronic inflammation. Therefore, integrating these four parameters enables a comprehensive assessment of structural, functional, and systemic risk, offering robust predictive value for disease progression in this high-risk population.

We evaluated model performance at extended time points (12, 18, and 24 months, as presented in Supplementary Fig. 1), and while the Area Under the Curve (AUC) values remained acceptable, our analysis showed that the highest predictive performance was observed at 3, 6, and 9 months (Fig. 4). These shorter-term intervals demonstrated stronger discriminative power, suggesting that early events in diabetic patients with CAD are more reliably predictable. Furthermore, early disease progression is clinically meaningful in this population. Coronary microvascular disease (CMD)—a condition that often precedes overt coronary artery disease—is known to play a central role in early cardiovascular deterioration and has been associated with worse long-term outcomes. As such, the 3-, 6-, and 9-month windows were intentionally chosen to capture these critical early stages, where timely intervention may have the greatest impact. While longer follow-up periods may provide additional insights, our aim was to identify early predictive markers that can support proactive clinical decision-making. We plan to expand the follow-up period in future prospective studies to assess long-term applicability.

Despite its strengths, our study has limitations. First, this was a retrospective, single-center analysis, which may limit the generalizability of our findings. Third, global longitudinal strain (GLS), a sensitive marker of subclinical myocardial dysfunction in diabetic patients, was not included due to limited data availability, which may have affected the model's ability to detect early cardiac changes. Fourth, the nomogram's predictive performance was evaluated only at 3, 6, and 9 months; extending predictions to longer time horizons would provide additional clinical utility. In addition, the model was not compared against existing risk scores such as SYNTAX, GRACE, or TIMI, which may limit the context for interpreting its relative predictive value. Finally, the retrospective design may introduce potential biases, and prospective studies are needed to validate the nomogram's real-world applicability.

Conclusion

We developed and validated a multimodal nomogram integrating clinical, imaging, and biochemical data to predict disease progression in diabetic patients with CAD. The model demonstrated good discrimination and calibration, providing a practical tool for individualized risk stratification. Our findings underscore the value of multimodal data in personalized cardiovascular risk assessment and patient management.

Supplementary Information

The online version contains supplementary material available at https://doi.org/10.1186/s12872-025-04737-1.

Supplementary Material 1: Fig. 1 (A) Receiver operating characteristic (ROC) curves for the training set. The model showed a decreasing trend in predictive performance with longer follow-up periods: AUC = 0.702 (95% CI: 0.627–0.777) at 12 months, 0.683 (95% CI: 0.621–0.764) at 18 months, and 0.689 (95% CI: 0.618–0.759) at 24 months. (B) ROC curves for the validation set showing a similar trend: AUC = 0.743 (95% CI: 0.594–0.892) at 12 months, 0.703 (95% CI: 0.57–0.836) at 18 months, and 0.687 (95% CI: 0.557–0.817) at 24 months.

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Author contributions

Jing Chen and Ling Yue contributed equally to this work. Ruonan Wang, Sunjing Shu, Liu Jin, and Mingmin Yan contributed to data collection and analysis. Changkong Ye and Liu Shuang contributed to the study design, data interpretation, and manuscript preparation. Changkong Ye and Liu Shuang are corresponding authors.

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Data availability

No datasets were generated or analysed during the current study.

Declarations

Ethics approval and consent to participate

The study was approved by the Ethics Committee of the Fourth Affiliated Hospital of China Medical University (Ethics ID: EC-2024-KS-117), and informed consent was obtained from all enrolled participants. All procedures were conducted in accordance with the principles outlined in the Declaration of Helsinki.

Consent for publication

All participants involved in this study have provided informed consent for the publication of the study results.

Competing interests

The authors declare no competing interests.

Clinical trial number

Not applicable.

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