Clinics Cardiology
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2688-5646

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The Value of Establishing a Predictive Model for Assessing Coronary Artery Lesions Based on Clinical Indicators

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Article Info:
Open Access | Research Article |
6(3), 2026 | PP - 01- 14
Reference Number: CC-v6-1053

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Received: Sept 24, 2026
Accepted: Nov 23, 2026
Published: Nov 24, 2026

Citation: Wang X, Yu H, Song B, Han W, The Value of Establishing a Predictive Model for Assessing Coronary Artery Lesions Based on Clinical Indicators, Clin Cardiol. 2026; 6(3): 53.

ABSTRACT

Background: Given the rising incidence of coronary artery disease, the rate of emergency coronary intervention has significantly increased, making noninvasive and rapid preoperative risk stratification using clinical characteristics particularly important. Therefore, this study aimed to develop and validate a prediction model based on clinical characteristics to forecast the likelihood of ≥70% coronary artery stenosis in patients.

Methods: Clinical data and coronary outcomes from 200 patients were retrospectively collected between 2022 and 2025.Data were randomly divided into two cohorts: the training set (n = 141) and the validation set (n = 59). A nomogram was constructed based on the results of multivariate logistic regression analysis in the training set. The predictive accuracy and discriminatory ability of the nomogram were then evaluated in both the training and validation sets using receiver operating characteristic (ROC) curves, calibration curves, and decision curve analysis (DCA) to demonstrate its clinical utility.

Results: A total of 200 patients were ultimately included, comprising 141 cases (70%) in the training set and 59 cases (30%) in the validation set. Among these, 115 patients (57.5%) exhibited coronary artery stenosis ≥70%, including 77 cases (54.61%) in the training set and 38 cases (64.41%) in the validation set. Six predictors were identified: CAVI > 9 m/s (OR: 0.022, 95% CI: 1.49–165.13, P = 0.022), CAR between 0.1–0.2 (OR: 4.06, 95% CI: 1.49–11.03, P 0.006), CAR > 0.2 (OR: 5.32, 95% CI: 1.19–23.76, P 0.029), BPV1 between 10–20 mmHg (OR: 3.16, 95% CI: 1.09–9.17, P 0.034), BPV1 > 20 mmHg (OR: 5.98, 95% CI: 1.54–23.13,P 0.01), Lpa > 300 mg/L (OR: 5.17, 95% CI: 1.95–13.75, P < 0.001), oral CCB (OR: 0.39, 95% CI: 0.15–0.99, P 0.047),TTR > 75% (OR: 6.98, 95% CI: 1.59–30.67, P < 0.01). The nomogram demonstrated good discriminatory ability with a concordance index (C-index) of 0.89 (95% CI: 0.84–0.94) during internal validation and showed good calibration. In the validation cohort, the model maintained good discriminatory ability (AUC = 0.87; 95% CI 0.70–1.00) and calibration. Decision curve analysis confirmed the clinical utility of the nomogram.

Conclusion: The nomogram incorporating clinical indicators such as LPA, CAVI, BPV, TTR, CAR, and CCB demonstrated excellent discriminatory

ability. This nomogram represents a promising tool for predicting the severity of coronary artery stenosis and guiding personalized treatment strategies.

2. Keywords: Coronary Artery Stenosis; Nomogram; Clinical Prediction Model; Risk Stratification; Percutaneous Coronary Intervention

Introduction

Coronary artery disease (CAD), one of the leading causes of death and disability globally, imposes a significant health and economic burden [1]. Based on data from NHANES 2017 to 2020, approximately 20.5 million Americans have CAD, while the PCI treatment rate decreased from 260.2 to 232.8 per 100,000 (−10.5%; P<0.001). During the same period, the outpatient PCI rate increased from 33.8 to 66.7 per 100,000 (+97.1%; P_{trend}<0.001), while the inpatient PCI rate decreased from 226.4 to 166.2 per 100,000 (−26.6%; P(trend) <0.001) [2,3]. As incidence and prevalence increased, the rate of inpatient PCI declined while the rate of outpatient and emergency PCI rose, indirectly highlighting the importance of clinical indicators in assessing coronary risk prior to angiography. Although the ISCHEMIA trial found no association between ischemic severity and increased risk of adverse CAD outcomes, more severe CAD was associated with increased all-cause mortality risk [4]. Therefore, predicting the severity of coronary stenosis not only heightens awareness of coronary lesions but also enables timely and appropriate vascular intervention, thereby reducing mortality and myocardial infarction rates while improving patient symptoms, quality of life, and long-term survival [3,4]. Currently, troponin is considered the most sensitive and specific biomarker [5]. However, elevated levels of these biomarkers are not unique to coronary artery disease, and an increasing number of clinical indicators for predicting coronary artery disease have emerged. Therefore, identifying more sensitive and specific biomarkers and optimizing treatment strategies remain key challenges in current clinical practice.

Current research on coronary artery disease risk factors predicting coronary artery stenosis involves diabetes, uric acid, CAVI, CRP/ALB, CAC, etc· [6-11]. Related studies found that during a median follow-up of 6.32 years, urate-lowering therapy was associated with a significant reduction in all-cause mortality (hazard ratio [HR]: 0.915; 95% confidence interval [CI]: 0.645–0.998) and major adverse cardiovascular events (MACE) (HR: 0.887; 95% CI: 0.661–0.990). The Cardioankle Vascular Index (CAVI) is a key indicator for assessing arterial stiffness (AS). CAVI in the CAD group (9.21 ± 0.79) was significantly higher than in the non-CAD group (8.48 ± 0.62) (p < 0.001). Elevated CAVI correlated with higher SYNTAX scores (mild, moderate, and severe: 9.09 ± 0.65, 9.80 ± 1.05, and 9.45 ± 0.46, respectively; p < 0.001) and Gensini scores (mild, moderate, and severe: 8.90 ± 0.65, 9.37 ± 0.95, and 9.43 ± 0.59, respectively; p < 0.001). Additionally, chronic low-grade inflammation associated with obesity, aging, and metabolic syndrome may further amplify inflammatory signals by stimulating inflammasomes, NF-κB, and B-cell signaling, thereby inducing coronary endothelial injury. Clinical studies have revealed that patients with severe CAD exhibit higher Gensini scores and higher C-reactive protein to albumin ratios (CAR) compared to those with non-seB, thereby amplifying inflammatory signals and inducing coronary endothelial injury. Clinical studies have revealed significant statistical differences in Gensini scores and the CRP-to-albumin ratio (CAR) between patients with severe CAD and those with non-severe CAD. Regression model analysis confirmed that CAR is a significant determinant of CAD severity, with a 95% confidence interval (CI) of 1.75–4.96 (p < 0.001). Numerous clinical studies have examined coronary artery stenosis, including the calculation of fractional flow reserve (FFR-CT) from coronary CTA, combined with coronary artery stenosis resistance (SR) [12], to assess coronary stenosis. Coronary stenosis generates turbulent blood flow patterns, manifesting as intravascular murmurs, which can be identified through acoustic techniques combined with modern analytical methods [13]. Myocardial perfusion imaging H_2O positron emission tomography (PET) to predict coronary artery stenosis in patients without a history of ischemic heart diseaseetc [14]. Considering that existing studies partially lack feasibility and employ single indicators, this research aims to develop and validate a prediction model based on clinical characteristics to forecast the likelihood of ≥70% coronary artery stenosis in patients.

Materials and Methods

Study population

This retrospective study collected data from all patients undergoing coronary angiography at Beijing Hospital of Integrated Traditional Chinese and Western Medicine, affiliated with Beijing University of Chinese Medicine, from April 2022 to April 2025. All methods complied with relevant guidelines and regulations. Inclusion criteria: 1. Age >18 years; 2. Complete clinical auxiliary tests and coronary angiography; 3. Informed consent and voluntary participation. Exclusion criteria: 1. Patients with prior interventions (stents, balloon angioplasty); 2. Incomplete information; 3. Severe heart failure (e.g., NYHA Class IV); 4. Uncontrolled severe arrhythmias (e.g., persistent atrial fibrillation, ventricular premature beats); 5. Severe hepatic impairment or renal insufficiency; 6. History of acute stroke or cerebrovascular disease;

7. Pregnant or lactating women; 8. Patients allergic to contrast agents; 9. Subjects with poor compliance unable to cooperate with testing. Study Endpoint: Braunwald's Heart Disease (12th Edition, 2022) states that coronary artery stenosis ≥70% is generally considered "hemodynamically significant" and may lead to myocardial ischemia. Therefore, the primary endpoint of this study is ≥70% coronary artery stenosis [14] (Figure 1). Participant Selection and Evaluation Process Flowchart.Ethics Approval and Consent to Participate

This study was approved by the Medical Ethics Review Committee of Beijing Hospital of Integrated Traditional Chinese and Western Medicine (Approval No. 202508280022) and conducted in accordance with the ethical principles outlined in the 1964 Declaration of Helsinki and its subsequent amendments, or other equivalent ethical standards. To ensure patient confidentiality, the identities of individuals included in this study were anonymized using computer-generated ID numbers. Upon admission, all patients signed written informed consent authorizing the analysis and publication of their anonymized medical data for research purposes.

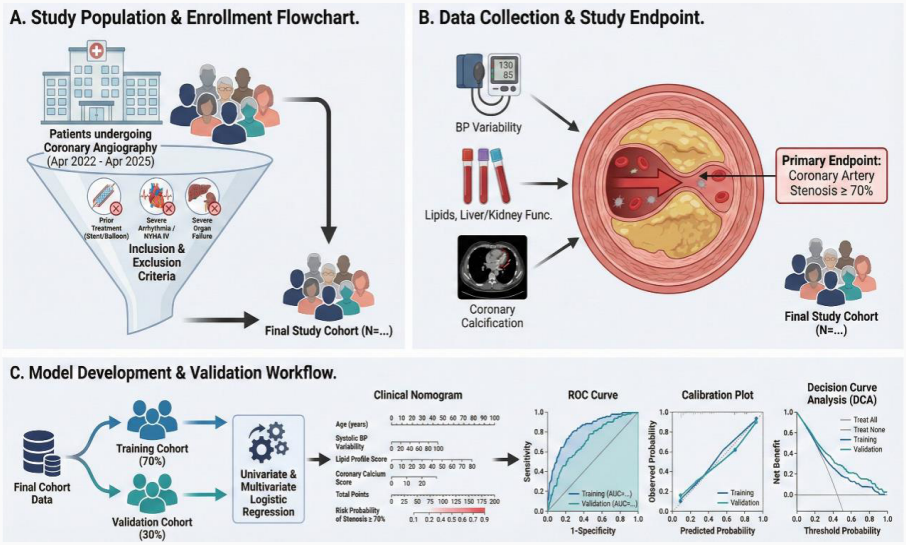


Figure 1: Overview of study design and workflow.

A. Patient enrollment and screening process: This displays all patients undergoing coronary angiography who underwent initial screening between April 2022 and April 2025. Funnel screening excluded patients with prior stent/balloon interventions, severe heart failure (NYHA Class IV), severe arrhythmias, and severe hepatic or renal impairment, ultimately defining the study cohort. B. Data Collection and Study Endpoint: Detailed extraction of clinical variables including blood pressure variability, lipid profiles, hepatic/renal function, and coronary artery calcification. The clinical endpoint was defined as ≥70% luminal stenosis demonstrated by coronary angiography.C. Model Development and Validation: The final cohort was divided into training and validation sets at a 7:3 ratio. Predictors were selected through univariate and multivariate logistic regression analyses, and a visual nomogram was constructed. Model predictive performance and clinical utility were evaluated across both cohorts using ROC curves, calibration plots, and decision curve analysis (DCA).

Data acquisition, Nomogram development and validation

We retrospectively analyzed basic information, including age, medical history, blood pressure variability, time spent within blood pressure targets, lipid profile, liver and kidney function, thyroid function, coronary artery calcification, and medication status. First, the entire cohort was divided into training and validation sets in a 7:3 ratio. Univariate logistic regression models were applied to the training set to assess the predictive ability of clinical factors for coronary artery lesions. Variables showing significant differences (P<0.05) in univariate analysis were included in multivariate analysis. Variables remaining significant (P<0.05) in multivariate analysis were visualized to establish a predictive model. The predictive capability of the nomogram was then evaluated using receiver operating characteristic (ROC) curves, calibration curves, and decision curve analysis (DCA) in both the training and validation cohorts to demonstrate its clinical utilityStatistical Analysis

All data analysis and graphing were performed using R software version 4.2.3. Continuous variables were compared using independent samples t-tests and rank-sum tests. Categorical variables were compared using chi-square tests and Fisher's exact tests. Univariate binary logistic regression analysis was performed with coronary artery stenosis ≥70% as the binary dependent variable and all clinical indicators as independent variables to identify statistically significant variables (P<0.05).These were incorporated into a multivariate binary logistic regression analysis (forward stepwise method, Forward: LR) to adjust for confounding effects. Variables were included at P < 0.05 and excluded at P > 0.10 to identify independent predictors of significant coronary artery stenosis.

Odds ratios (OR), 95% confidence intervals (CI), and two-tailed P values were calculated for each variable. All tests were two-tailed, with P < 0.05 indicating statistical significance.

Results

Baseline Characteristics of Patients

This study included 200 patients, comprising 141 in the training set and 59 in the validation set. Based on coronary angiography results, patients were categorized into three groups: stenosis ≥70% group (significant stenosis), and <70% group (non-significant stenosis). The significant stenosis group included 115 patients, with a mean age of 65.47 ± 11.38 years (Table 1). Among coronary lesions identified, patients in the significant stenosis group were older. Additionally, no significant differences existed between groups in gender, BMI, personal history of smoking or alcohol consumption, diabetes, or hypertension (p > 0.05). Regarding laboratory indicators, patients in the significant stenosis group exhibited higher CAR, Lpa, CAVI, and BPV compared to the non-significant stenosis group (p < 0.01).Screening for Independent Predictive Factors

Univariate analysis revealed that patients with ≥70% coronary artery stenosis exhibited the following characteristics: Specifically: Diastolic blood pressure >90 mmHg (OR: 0.33, 95% CI: 0.12–0.93, P=0.036) Short-term systolic blood pressure fluctuations of 10-20 mmHg (OR: 4.56, 95% CI: 2.09–9.95, P < 0.01) Short-term systolic blood pressure fluctuations > 20 mmHg (OR: 8.63, 95% CI: 2.81–26.48, P < 0.01) short-term diastolic blood pressure variability 10-20 mmHg (OR: 4.23, 95% CI: 1.06–16.82, P=0.041), short-term diastolic blood pressure variability >20 mmHg (OR: 5.98, 95% CI: 1.53–23.36, P<0.01), Blood pressure variability between 25-75% (OR: 2.27, 95% CI: 1.07–4.79, P = 0.032) CAVI between 8-9 m/s (OR: 4.80, 95% CI: 2.17–10.61, P < .001) CAVI > 9 m/s (OR: 18.10, 95% CI: 2.23–146.95, P = 0.007) , CAR at 0.1–0.2 (OR: 4.01, 95% CI: 1.88–8.58, P < 0.001),CAR > 0.2 (OR: 4.90, 95% CI: 1.57–15.28, P = 0.006), coronary artery calcification visible on lung CT (OR: 4.84, 95% CI: 2.05–11.42, P < 0.001), Lpa > 300 mg/L (OR: 6.71, 95% CI: 3.20–14.10, P < 0.001), APoA1 > 1 (OR: 0.29, 95% CI: 0.14–0.59, P < 0.001), Tg > 1.7 (OR: 2.17, 95% CI:1.10–4.30, P < 0.026), and multiple oral CCBs (OR: 0.47, 95% CI: 0.24–0.92, P < 0.028)(Table 2).

Table 1: Baseline Characteristics of Patients

Variables	Total (n = 200)	Training cohort (n = 141)	Validation cohort (n = 59)	Statistic	*P*
Age, n(%)				χ^2=0.83	0.364
<60	62 (31.00)	41 (29.08)	21 (35.59)		
>60	138 (69.00)	100 (70.92)	38 (64.41)		
BMI, n(%)				χ^2=0.29	0.592
<23.9	70 (35.00)	51 (36.17)	19 (32.20)		
>23.9	130 (65.00)	90 (63.83)	40 (67.80)		
SBP, n(%)				χ^2=1.61	0.205
<140	98 (49.00)	65 (46.10)	33 (55.93)		
>140	102 (51.00)	76 (53.90)	26 (44.07)		
DBP, n(%)				χ^2=0.40	0.525
<90	171 (85.50)	122 (86.52)	49 (83.05)		
>90	29 (14.50)	19 (13.48)	10 (16.95)		
EF, n(%)				χ^2=0.22	0.642
<50%	20 (10.00)	15 (10.64)	5 (8.47)		
>50%	180 (90.00)	126 (89.36)	54 (91.53)		
Lpa, n(%)				χ^2=0.01	0.917
<300	87 (43.50)	61 (43.26)	26 (44.07)		
>300	113 (56.50)	80 (56.74)	33 (55.93)		
CREA, n(%)				χ^2=0.06	0.807
<90	171 (85.50)	120 (85.11)	51 (86.44)		
>90	29 (14.50)	21 (14.89)	8 (13.56)		
BUN, n(%)				χ^2=1.41	0.235
<7	189 (94.50)	131 (92.91)	58 (98.31)		
>7	11 (5.50)	10 (7.09)	1 (1.69)		
eGFR, n (%)				χ^2=3.32	0.068
<90%	108 (54.00)	82 (58.16)	26 (44.07)		
>90%	92 (46.00)	59 (41.84)	33 (55.93)		
ALT, n(%)				χ^2=0.00	1.000
<50	186 (93.00)	131 (92.91)	55 (93.22)		
>50	14 (7.00)	10 (7.09)	4 (6.78)		
AST, n(%)				χ^2=0.09	0.770
<40	175 (87.50)	124 (87.94)	51 (86.44)		
>40	25 (12.50)	17 (12.06)	8 (13.56)		
GGT, n(%)				χ^2=0.54	0.461
<60	181 (90.50)	129 (91.49)	52 (88.14)		
>60	19 (9.50)	12 (8.51)	7 (11.86)		
HDLC, n(%)				χ^2=0.53	0.467
<1	123 (61.50)	89 (63.12)	34 (57.63)		
>1	77 (38.50)	52 (36.88)	25 (42.37)		
LDLC, n(%)				χ^2=0.50	0.481
<2.4	94 (47.00)	64 (45.39)	30 (50.85)		
>2.4	106 (53.00)	77 (54.61)	29 (49.15)		
ApoA1, n(%)				χ^2=0.02	0.879
<1	117 (58.50)	82 (58.16)	35 (59.32)		
>1	83 (41.50)	59 (41.84)	24 (40.68)		
ApoB, n(%)				χ^2=2.66	0.103
<0.6	37 (18.50)	22 (15.60)	15 (25.42)		
>0.6	163 (81.50)	119 (84.40)	44 (74.58)		
Tg, n(%)				χ^2=0.93	0.335
<1.7	115 (57.50)	78 (55.32)	37 (62.71)		
>1.7	85 (42.50)	63 (44.68)	22 (37.29)		
Tc, n(%)				χ^2=0.42	0.519

<6	175 (87.50)	122 (86.52)	53 (89.83)		
>6	25 (12.50)	19 (13.48)	6 (10.17)		
MYO, n(%)				χ^2=0.00	0.980
<58	195 (97.50)	138 (97.87)	57 (96.61)		
>58	5 (2.50)	3 (2.13)	2 (3.39)		
CKMB, n(%)				χ^2=0.00	1.000
<5	189 (94.50)	133 (94.33)	56 (94.92)		
>5	11 (5.50)	8 (5.67)	3 (5.08)		
TNI, n(%)				-	0.701
<0.1	12 (6.00)	10 (7.09)	2 (3.39)		
>0.1	170 (85.00)	117 (82.98)	53 (89.83)		
<0.5	1 (0.50)	1 (0.71)	0 (0.00)		
>0.1	17 (8.50)	13 (9.22)	4 (6.78)		
NT-proBNP, n(%)				χ^2=0.13	0.714
<900	156 (78.00)	109 (77.30)	47 (79.66)		
>900	44 (22.00)	32 (22.70)	12 (20.34)		
Sex, n(%)				χ^2 = 0.17	0.682
male	123 (61.50)	88 (62.41)	35 (59.32)		
female	77 (38.50)	53 (37.59)	24 (40.68)		
Smoke, n(%)				-	0.371
no	87 (43.50)	63 (43.97)	24 (40.68)		
yes	113 (56.50)	79 (56.03)	34 (57.63)		
Drink, n(%)				χ^2=0.00	0.974
no	119 (59.50)	84 (59.57)	35 (59.32)		
yes	81 (40.50)	57 (40.43)	24 (40.68)		
HTN, n(%)				χ^2=3.30	0.069
no	84 (42.00)	65 (46.10)	19 (32.20)		
yes	116 (58.00)	76 (53.90)	40 (67.80)		
DM, n(%)				χ^2=0.67	0.415
no	110 (55.28)	80 (57.14)	30 (50.85)		
yes	89 (44.72)	60 (42.86)	29 (49.15)		
Result, n(%)				χ^2=1.63	0.201
<70%	85 (42.50)	64 (45.39)	21 (35.59)		
≥70%	115 (57.50)	77 (54.61)	38 (64.41)		
CAVI, n(%)				χ^2=5.43	0.066
<8 m/s	108 (54.00)	82 (58.16)	26 (44.07)		
8-9 m/s	69 (34.50)	47 (33.33)	22 (37.29)		
<9 m/s	23 (11.50)	12 (8.51)	11 (18.64)		
CAR, n(%)				χ^2=1.58	0.454
<0.1	97 (48.50)	66 (46.81)	31 (52.54)		
0.1–0.2	74 (37.00)	56 (39.72)	18 (30.51)		
>0.2	29 (14.50)	19 (13.48)	10 (16.95)		
BPV1, n(%)				χ^2=0.10	0.949
<10 mmHg	86 (43.00)	60 (42.55)	26 (44.07)		
10–20 mmHg	78 (39.00)	56 (39.72)	22 (37.29)		
>20 mmHg	36 (18.00)	25 (17.73)	11 (18.64)		
CAC, n(%)				χ^2=0.00	0.954
no	48 (24.00)	34 (24.11)	14 (23.73)		
yes	152 (76.00)	107 (75.89)	45 (76.27)		
CCB, n(%)				χ^2=3.70	0.054
no	99 (49.50)	76 (53.90)	23 (38.98)		
yes	101 (50.50)	65 (46.10)	36 (61.02)		
TTR, n(%)				χ^2=2.06	0.358
<25%	99 (49.50)	74 (52.48)	25 (42.37)		
25-75%	72 (36.00)	49 (34.75)	23 (38.98)		
>75%	29 (14.50)	18 (12.77)	11 (18.64)		
Gensini, n(%)				χ^2=1.87	0.394
<20	110 (55.00)	80 (56.74)	30 (50.85)		
20-40	54 (27.00)	39 (27.66)	15 (25.42)		
>40	36 (18.00)	22 (15.60)	14 (23.73)		
BPV2, n(%)				χ^2=0.40	0.819
<10 mmHg	21 (10.50)	14 (9.93)	7 (11.86)		
10–20 mmHg	81 (40.50)	56 (39.72)	25 (42.37)		
>20 mmHg	98 (49.00)	71 (50.35)	27 (45.76)		
FT3, n(%)				-	0.655
<3.1 pmol/L	12 (6.00)	8 (5.67)	4 (6.78)		
3.1–6.8 pmol/L	186 (93.00)	132 (93.62)	54 (91.53)		
>6.8 pmol/L	2 (1.00)	1 (0.71)	1 (1.69)		
FT4, n(%)				-	1.000
<12 pmol/L	12 (6.00)	9 (6.38)	3 (5.08)		
12–22 pmol/L	185 (92.50)	130 (92.20)	55 (93.22)		
>22 pmol/L	3 (1.50)	2 (1.42)	1 (1.69)		
TSH, n(%)				χ^2=0.77	0.681
<0.27 mIU/L	11 (5.50)	8 (5.67)	3 (5.08)		
0.27–4.2 mIU/L	155 (77.50)	107 (75.89)	48 (81.36)		
>4.2 mIU/L	34 (17.00)	26 (18.44)	8 (13.56)		
RAAS, n(%)				χ^2=0.02	0.888
no	90 (45.00)	63 (44.68)	27 (45.76)		
yes	110 (55.00)	78 (55.32)	32 (54.24)		
BB, n(%)				χ^2=0.39	0.532
no	64 (32.00)	47 (33.33)	17 (28.81)		
yes	136 (68.00)	94 (66.67)	42 (71.19)		

Age, BMI, SBP, DBP, EF, Lp(a), CREA, BUN, eGFR, ALT, AST, GGT, HDL-C, LDL-C, Apo A1: apolipoprotein A1, Apo B: apolipoprotein B, TG: triglycerides, TC: total cholesterol, MYO: myoglobin, CK-MB: creatine kinase MB isoenzyme, TNI: troponin I, NT-proBNP N-terminal pro-B-type natriuretic peptide precursor, HTN: hypertension, DM: Diabetes Mellitus, CAVI: Ankle-Brachial Index, CAR: CRP/Albumin Ratio, BPV1: Systolic Blood Pressure Variability, BPV2: Diastolic Blood Pressure Variability, CAC: Coronary Artery Calcification (detected by lung CT), CCB: Oral Calcium Channel Blocker, TTR: Time in Target Range, Gensini is a clinically used quantitative assessment system for coronary artery stenosis severity, FT3: Free Triiodothyronine, FT4: Free Tetraiodothyronine, TSH: Thyroid-Stimulating Hormone, RAAS: Renin-Angiotensin-Aldosterone System Inhibitor, BB: Beta-Adrenergic Receptor Blocker.

Table 2: Univariate Analysis

Variables	β	S.E	Z	P	OR (95% CI)
Age					
<60					1.00 (Reference)
>60	0.33	0.37	0.89	0.374	1.39 (0.67 ~ 2.89)
Sex					
male					1.00 (Reference)
female	0.50	0.35	1.41	0.158	1.65 (0.82 ~ 3.31)
BMI					
<23.9					1.00 (Reference)
>23.9	-0.52	0.36	-1.46	0.146	0.59 (0.29 ~ 1.20)
Smoke					
no					1.00 (Reference)
yes	-0.13	0.34	-0.39	0.697	0.88 (0.45 ~ 1.71)
Drink					
no					1.00 (Reference)
yes	0.10	0.35	0.30	0.764	1.11 (0.56 ~ 2.18)
HTN					
no					1.00 (Reference)
yes	0.17	0.34	0.51	0.612	1.19 (0.61 ~ 2.31)
DM					
no					1.00 (Reference)
yes	-0.42	0.34	-1.22	0.222	0.66 (0.33 ~ 1.29)
SBP (mmHg)					
<140					1.00 (Reference)
>140	0.17	0.34	0.51	0.612	1.19 (0.61 ~ 2.31)
DBP (mmHg)					
<90					1.00 (Reference)
>90	-1.10	0.53	-2.10	0.036	0.33 (0.12 ~ 0.93)
BPV1 (mmHg)					
<10					1.00 (Reference)
10–20	1.52	0.40	3.80	<.001	4.56 (2.09 ~ 9.95)
>20	2.16	0.57	3.77	<.001	8.63 (2.81 ~ 26.48)
BPV2 (mmHg)					
<10					1.00 (Reference)
10–20	1.44	0.70	2.05	0.041	4.23 (1.06 ~ 16.82)
>20	1.79	0.70	2.57	0.010	5.98 (1.53 ~ 23.36)
TTR(%)					
<25					1.00 (Reference)
25-75	0.82	0.38	2.14	0.032	2.27 (1.07 ~ 4.79)
>75	0.99	0.55	1.81	0.070	2.69 (0.92 ~ 7.85)
CAVI (m/s)					
<8					1.00 (Reference)
8–9	1.57	0.40	3.88	<.001	4.80 (2.17 ~ 10.61)
<9	2.90	1.07	2.71	0.007	18.10 (2.23 ~ 146.95)
EF (%)					
<50					1.00 (Reference)
>50	-0.92	0.61	-1.50	0.133	0.40 (0.12 ~ 1.32)
CAR					
<0.1					1.00 (Reference)
0.1–0.2	1.39	0.39	3.59	<.001	4.01 (1.88 ~ 8.58)
>0.2	1.59	0.58	2.74	0.006	4.90 (1.57 ~ 15.28)
CAC					
no					1.00 (Reference)
yes	1.58	0.44	3.61	<.001	4.84 (2.05 ~ 11.42)
FT3					
<3.1 pmol/L					1.00 (Reference)
3.1–6.8 pmol/L	-0.98	0.83	-1.17	0.242	0.38 (0.07 ~ 1.93)
>6.8 pmol/L	13.47	882.74	0.02	0.988	706059.99 (0.00 ~ Inf)
FT4					
<12 pmol/L					1.00 (Reference)
12–22 pmol/L	-2.02	1.08	1.88	0.061	0.13 (0.02 ~ 1.09)
>22 pmol/L	13.49	1029.12	0.01	0.990	719726.61 (0.00 ~ Inf)
TSH					
<0.27 mIU/L					1.00 (Reference)
0.27–4.2 mIU/L	0.13	0.73	0.18	0.858	1.14 (0.27 ~ 4.80)
>4.2 mIU/L	0.47	0.81	0.58	0.564	1.60 (0.32 ~ 7.89)
CREA (μmol/L)					
<90					1.00 (Reference)
>90	-0.11	0.47	0.22	0.824	0.90 (0.36 ~ 2.28)
BUN (μmol/L)					
<7					1.00 (Reference)
>7	-1.11	0.71	1.56	0.120	0.33 (0.08 ~ 1.33)
eGFR (%)					
<90%					1.00 (Reference)
>90%	0.45	0.35	1.29	0.196	1.57 (0.79 ~ 3.09)
ALT (U/L)					
<50					1.00 (Reference)
>50	0.24	0.67	0.35	0.723	1.27 (0.34 ~ 4.70)
AST (U/L)					
<40					1.00 (Reference)
>40	0.20	0.52	0.37	0.710	1.22 (0.43 ~ 3.40)

GGT (U/L)					
<60					1.00 (Reference)
>60	-0.20	0.60	0.33	0.738	0.82 (0.25 ~ 2.67)
HDLC (mmol/L)					
<1					1.00 (Reference)
>1	0.20	0.35	0.56	0.574	1.22 (0.61 ~ 2.43)
LDLC (mmol/L)					
<2.4					1.00 (Reference)
>2.4	0.34	0.34	1.00	0.317	1.41 (0.72 ~ 2.74)
Lpa (mg/L)					
<300					1.00 (Reference)
>300	1.90	0.38	5.03	<.001	6.71 (3.20 ~ 14.10)
ApoA1 (g/L)					
<1					1.00 (Reference)
>1	-1.23	0.36	3.45	<.001	0.29 (0.14 ~ 0.59)
ApoB (g/L)					
<0.6					1.00 (Reference)
>0.6	-0.22	0.47	0.46	0.646	0.81 (0.32 ~ 2.03)
Tg (mmol/L)					
<1.7					1.00 (Reference)
>1.7	0.78	0.35	2.23	0.026	2.17 (1.10 ~ 4.30)
Tc (mmol/L)					
<6					1.00 (Reference)
>6	0.15	0.50	0.31	0.757	1.17 (0.44 ~ 3.10)
MYO (ng/mL)					
<58					1.00 (Reference)
>58	0.52	1.24	0.42	0.675	1.68 (0.15 ~ 18.96)
CKMB (ng/mL)					
<5					1.00 (Reference)
>5	0.96	0.83	1.15	0.249	2.62 (0.51 ~ 13.45)
CCB					
no					1.00 (Reference)
yes	-0.76	0.34	2.19	0.028	0.47 (0.24 ~ 0.92)
TNI (ng/mL)					
<0.1	0.05	0.66	0.08	0.938	1.05 (0.29 ~ 3.83)
>0.1	1.70	1.00	1.71	0.087	5.50 (0.78 ~ 38.70)
NT-proBNP (ng/mL)					
<900					1.00 (Reference)
>900	0.42	0.41	1.02	0.310	1.52 (0.68 ~ 3.41)
RAAS					
no					1.00 (Reference)
yes	0.63	0.34	1.83	0.067	1.88 (0.96 ~ 3.68)
BB					
no					1.00 (Reference)
yes	0.34	0.36	0.96	0.339	1.41 (0.70 ~ 2.85)

Subsequently, variables showing significant differences in univariate analysis were included in multivariate analysis. Multivariate analysis revealed that CAVI > 9 m/s (OR: 0.022, 95% CI: 1.49–165.13, P = 0.022),CAR between 0.1 and 0.2 (OR: 4.06, 95% CI: 1.49–11.03, P < 0.006), and CAR > 0.2 (OR: 5.32, 95% CI: 1.19–23.76, P 0.029), short-term systolic blood pressure variability 10–20 mmHg (OR: 3.16, 95% CI: 1.09–9.17, P 0.034), short-term systolic blood pressure variability >20 mmHg (OR: 5.98, 95% CI: 1.54–23.13,P 0.01), Lpa > 300 mg/L (OR: 5.17, 95% CI: 1.95–13.75, P < 0.001), oral CCB (OR: 0.39, 95% CI: 0.15–0.99,P 0.047), and blood pressure variability >75% (OR: 6.98, 95% CI: 1.59–30.67, P 0.01) were independent predictors of significant coronary artery stenosis (Table 3)

Model Construction and Validation

For variables showing residual variation in the multivariate analysis (defined as independent predictors), we visualized them to construct a nomogram. Subsequently, we validated the model's predictive performance on both the training and validation sets, yielding area under the ROC curve (AUC) values of 0.88 (95% CI: 0.83–0.94) and 0.77 (95% CI: 0.66–0.89), respectively. Both calibration and decision curves indicate the model's strong predictive performance (Figure 2).

Discussion

With the rising incidence and prevalence of coronary heart disease, it is imperative to address how to reduce the misdiagnosis rate among patients with atypical symptoms, enhance diagnostic accuracy for early intervention in adverse cardiovascular events, and more precisely identify high-risk patients for stratification and personalized treatment. This approach aims to reduce healthcare costs for low-risk patients and improve the overall patient experience [15-17]. Clinical evidence predicting coronary artery stenosis severity remains limited. Therefore, we innovatively constructed a visual clinical model for predicting significant coronary stenosis. Its nomogram validation demonstrated excellent discrimination and calibration capabilities. Previous studies have identified correlations between Lpa, TTR, CAVI, BPV, CAR, and coronary artery stenosis [18], which aligns with our findings. We identified Lpa, TTR, CAVI, BPV, CAR, and oral CCB as independent predictors. Based on the standard deviation of the nomogram, CAVI emerged as the most significant risk factor, followed by

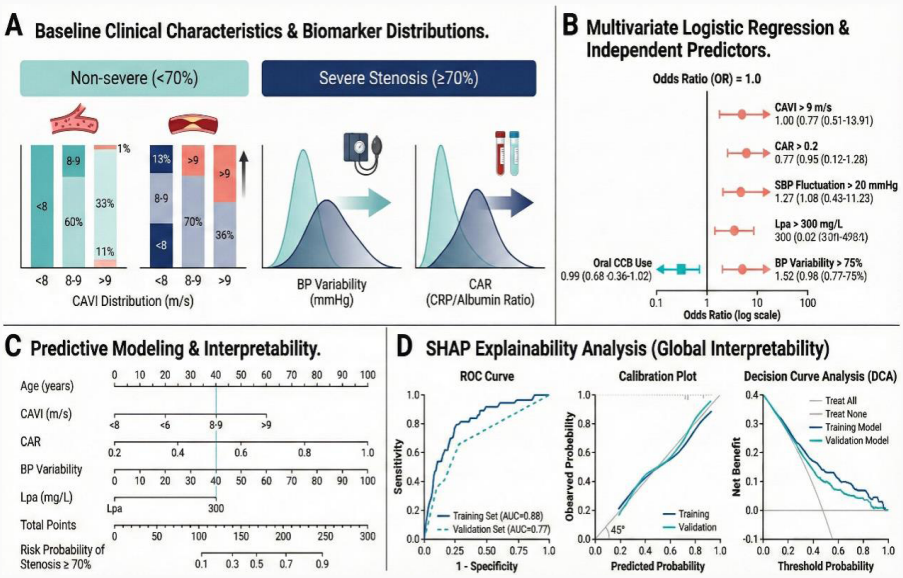


Figure 2: Performance evaluation of the predictive model.

A. Distribution of clinical baseline characteristics and biomarkers: Demonstrates significant differences in key indicators between the significantly stenotic group (≥70%) and the non-significantly stenotic group. The prevalence of significant stenosis shows a marked upward trend with increasing levels of CAVI (carotid artery stiffness index), BPV (blood pressure variability), and CAR (C-reactive protein/albumin ratio) (dark blue indicates high-risk group). B. Multivariate Logistic Regression Analysis: Forest plot displays independent predictors of significant stenosis. CAVI > 9 m/s, CAR > 0.2, short-term systolic blood pressure variability > 20 mmHg, and Lpa > 300 mg/L are significant risk factors (OR > 1); oral CCB (calcium channel blocker) is the sole significant protective factor (OR < 1). C. Clinical Nomogram: A visual prediction tool constructed based on multivariate regression results. By accumulating scores for age, CAVI, CAR, BPV, and Lpa, it enables intuitive estimation of an individual's probability of developing significant coronary artery stenosis. D. Model Validation: ROC Curve: Training set (AUC = 0.88) and validation set (AUC = 0.77) demonstrate good discriminatory ability. Calibration Plot: Predicted probabilities align closely with observed probabilities, indicating high model accuracy. Decision Curve Analysis (DCA): The model delivers significant clinical net benefit across a broad range of threshold probabilities.

Table 3: Multivariate Analysis

Variables	Univariate						Multivariate				
	β	S.E	Z	*P*	OR (95% CI)		β	S.E	Z	*P*	OR (95% CI)
CAVI (m/s)											
<8					1.00 (Reference)						1.00 (Reference)
8-9	1.57	0.40	3.88	<.001	4.80 (2.17 ~ 10.61)		0.70	0.51	1.35	0.177	2.00 (0.73 ~ 5.50)
<9	2.90	1.07	2.71	0.007	18.10 (2.23 ~ 146.95)		2.75	1.20	2.29	0.022	15.68 (1.49 ~ 165.13)
CAR											
<0.1					1.00 (Reference)						1.00 (Reference)
0.1–0.2	1.39	0.39	3.59	<.001	4.01 (1.88 ~ 8.58)		1.40	0.51	2.74	0.006	4.06 (1.49 ~ 11.03)

>0.2	1.59	0.58	2.74	0.006	4.90 (1.57 ~ 15.28)		1.67	0.76	2.19	0.029	5.32 (1.19 ~ 23.76)
BPV1 (mmHg)											
<10					1.00 (Reference)						1.00 (Reference)
10–20	1.52	0.40	3.80	<.001	4.56 (2.09 ~ 9.95)		1.15	0.54	2.12	0.034	3.16 (1.09 ~ 9.17)
>20	2.16	0.57	3.77	<.001	8.63 (2.81 ~ 26.48)		1.79	0.69	2.59	0.010	5.98 (1.54 ~ 23.13)
Lpa (mg/L)											
<300					1.00 (Reference)						1.00 (Reference)
>300	1.90	0.38	5.03	<.001	6.71 (3.20 ~ 14.10)		1.64	0.50	3.30	<.001	5.17 (1.95 to 13.75)
CCB											
no					1.00 (Reference)						1.00 (Reference)
yes	-0.76	0.34	-2.19	0.028	0.47 (0.24 ~ 0.92)			0.48	-1.99	0.047	0.39 (0.15 ~ 0.99)
TTR(%)											
<25					1.00 (Reference)						1.00 (Reference)
25-75	0.82	0.38	2.14	0.032	2.27 (1.07 ~ 4.79)		0.86	0.53	1.63	0.104	2.37 (0.84 ~ 6.74)
>75	0.99	0.55	1.81	0.070	2.69 (0.92 ~ 7.85)		1.94	0.76	2.57	0.010	6.98 (1.59 ~ 30.67)

TTR, BPV, CAR, and Lpa.

CAVI (Coronary Artery Compliance Index) is a composite index derived by assessing arterial pulse wave velocity (PWV) and adjusting for factors such as blood pressure [19]. Our study found that CAVI values in the non-significant stenosis group were predominantly within the normal range, indicating good vascular compliance and diastolic function in these patients. Conversely, CAVI values in the significant stenosis group were often elevated, reflecting more severe arterial stiffness. As is well known, while the aorta transmits pulse waves to peripheral arteries, they also reflect at the bifurcation of the common iliac arteries. The retrograde reflected pulse wave amplifies arterial blood pressure upon returning to the ascending aorta, a phenomenon termed the Windkessel effect. Increased arterial stiffness weakens this Windkessel effect, elevating afterload and myocardial oxygen demand. Long-term hemodynamic alterations reduce coronary perfusion pressure [15,16]. This may promote atherosclerotic plaque formation, increasing the risk of coronary artery occlusion and myocardial ischemia.

Studies indicate that greater short-term systolic blood pressure variability in hypertensive patients correlates with increased coronary risk. In each TIA

cohort, SBP variability during follow-up emerged as a strong independent predictor of coronary events, surpassing the predictive value of mean SBP. Maximum SBP demonstrated greater predictive utility than mean SBP, and stable hypertension yielded better prognosis than episodic hypertension. Our study found that, relative to diastolic pressure, greater short-term systolic pressure variability positively correlates with the severity of coronary artery disease. Similarly, in this prospective, single-center cohort, a 3-year follow-up revealed [20], systolic BPV was strongly associated with risk stratification after myocardial infarction. A meta-analysis incorporating 41 studies further confirmed that long-term BPV independently predicts cardiovascular events and all-cause mortality, holding significant importance in long-term cardiovascular risk stratification. This may be related to mechanical damage caused by prolonged high-pressure blood flow impacting the vascular endothelium [21]. This includes adaptive responses by endothelial cells, manifested as cell alignment in the direction of blood flow, upregulation of antioxidant and anti-inflammatory gene expression, imbalance in the production of procoagulant substances, weakened vasodilatory-vasoconstrictive responses, increased expression of adhesion molecules and chemokines, differentiation of monocytes into macrophages, uptake of oxidized LDL (oxLDL), and foam cell formation, which subsequently promotes the progression of coronary atherosclerosis.

Lipoprotein(a) [Lp(a)] is an LDL-like molecule characterized by an additional glycoprotein—apolipoprotein(a) (apo(a))—linked to apoB-100 via a single disulfide bond. The pathophysiological function of Lp(a) is primarily attributed to the presence of its apo(a) subunit. Lp(a) promotes platelet activation and aggregation by attenuating collagen- and ADP-driven platelet aggregation via apo(a). High Lp(a) levels result in excessive OxPL accumulation in the arterial wall, thereby inducing inflammation, promoting endoplasmic reticulum stress and macrophage apoptosis, and altering fibrin network structure to reduce permeability and enhance resistance to lysis, contributing to the development of a more vulnerable plaque phenotype [13,14]. Our study found that patients with Lp(a) > 300 mg/dL may exhibit more severe coronary artery stenosis than those with Lp(a) < 300 mg/dL. A study involving 408,687 individuals from the UK Biobank [22], an observational study found that low Lp(a) levels were associated with reduced cardiovascular disease risk. A retrospective analysis of 272 patients suspected of coronary heart disease [23], coronary plaque status was assessed using coronary computed tomography angiography (CTCA). Logistic regression analysis revealed that Lp(a) levels were an independent predictor of high-risk plaque (HRP), defined as the presence of positive remodeling and/or low-attenuation plaques. Elevated Lp(a) levels correlated with the severity of coronary atherosclerosis.

Recent studies have demonstrated a close relationship between inflammation and atherosclerosis [24,25]. Elevated C-reactive protein (CRP) and reduced albumin levels correlate with coronary thrombotic burden. The cutoff value for high thrombotic burden was 0.15, with a sensitivity of 88.4% and specificity of 43.6%.and a specificity of 43.6%. In a study of 100 myocardial infarction patients, the CRP/ALB ratio showed a significant positive correlation with the severity of coronary artery disease, and elevated serum CRP/ALB levels increased the risk of poor prognosis. Compared to single markers, CAR integrates and quantitatively assesses the overall imbalance between inflammatory activation and nutritional status [26]. Our study similarly confirms this imbalance is independently associated with the severity of coronary atherosclerotic lesions. This may relate to serum albumin's multiple physiological functions, including anti-inflammatory, antioxidant, anticoagulant, and antiplatelet aggregation activities. CRP actively participates in activating the complement system, inducing apoptosis, vascular cell activation, leukocyte recruitment, lipid accumulation, platelet aggregation, and ultimately thrombus formation [27].

Our study found that patients taking oral calcium channel blockers (CCBs) were more likely to have severe coronary stenosis. As is well known [28], in short-term studies, CCBs increase coronary blood flow and improve myocardial oxygenation by reducing total peripheral resistance and lowering arterial pressure. In fact, earlier studies such as INTACT, MHIT, and PREVENT (Prospective Randomized Evaluation of Novascidic Acid's Effects on Vascular Function) have reported [29-31], that dihydropyridine calcium channel blockers like nifedipine, nicardipine, and amlodipine are ineffective against existing coronary artery lesions. Similarly, the long-acting nifedipine did not slow vascular progression in coronary artery disease. This may be because, owing to its antianginal effects, the drug reduces the current need for coronary revascularization. Therefore, in clinical practice, we should pay closer attention to the coronary artery status of patients receiving long-term oral CCBs.

The findings of this study are largely exploratory and suggestive, providing direction and preliminary evidence for subsequent large-scale prospective research. However, this study has several limitations. 1. First, due to the inherent limitations of retrospective studies, we cannot avoid potential research bias. Therefore, large-scale clinical trials are still needed to further validate our results. 2. Second, the small sample size may result in insufficient statistical power, sensitivity to chance, difficulty in reflecting the diversity of the target population, and potential distortion of true trends by individual outliers. 3. Finally, this study employed internal validation. Although it achieved a favorable AUC value, external validation is still required to enhance the reliability of the findings.

Conclusion

In summary, compared to single risk factors or predicting the severity of coronary artery lesions, our nomogram demonstrates higher accuracy, good clinical utility, and relatively more precise individualized treatment prediction. Therefore, our nomogram may be used to predict the degree of coronary artery stenosis.

Declarations

Ethics Approval and Consent to Participate

This study was approved by the Medical Ethics Review Committee of Beijing Hospital of Integrated Traditional Chinese and Western Medicine (Approval No. 202508280022) and was conducted in accordance with the ethical standards of the 1964 Declaration of Helsinki and its later amendments. All participants provided written informed consent prior to enrollment, authorizing the use of their anonymized clinical data for research purposes.

Availability of Data and Materials

The datasets generated and analyzed during the current study are not publicly available due to patient confidentiality but are available from the corresponding author on reasonable request.

Funding

This research did not receive any specific grant from funding agencies in the public, commercial, or not-for-profit sectors.

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