Article

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Clinical Risk Factors and Characteristics of Coronary Artery Lesions in Premature Acute Myocardial Infarction Patients

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Background: The incidence of atherosclerotic cardiovascular disease (ASCVD) is increasing, with individuals experiencing acute myocardial infarction (AMI) at a younger age. Premature AMI is a serious condition with high rates of morbidity and mortality. This study aimed to identify clinical characteristics and risk factors associated with premature AMI and to evaluate the diagnostic value of those risk factors.

Methods: The study collected data from first-time AMI patients who underwent coronary angiography at the hospital between January 2022 and April 2023. They were divided into two groups by age: premature AMI (men <55 years, women <65 years) and non-premature AMI. A control group of similar-aged patients without coronary artery disease was also included.

Results: Out of 388 patients with first-time AMI, 313 were male, and 249 had ST-segment elevation myocardial infarction (STEMI). Among 73 control patients, 31 were male. Those with premature AMI had more risk factors like smoking, overweight, obesity, family history of coronary artery disease, and STEMI. They also had shorter hospital stays and higher diastolic blood pressure and faster heart rates. Single-vessel lesions were more frequent in premature AMI patients. After adjusting for confounding factors, smoking status (Odds ratio (OR) 4.454, 95% confidence interval (CI): 1.836–10.806, $p = 0.001$), glycated hemoglobin (HbA1c) level (OR 2.261, 95% CI: 1.219–4.193, $p = 0.010$), the non-high-density lipoprotein cholesterol (non-HDL-C)/HDL-C ratio (OR 4.394, 95% CI: 1.204–16.031, $p = 0.025$), and the monocyte-to-high-density lipoprotein ratio (MHR) (OR 6.164, 95% CI: 1.386–27.417, $p = 0.017$) were identified as independent risk factors for premature AMI development. The combination of these risk factors provided the greatest predictive value for premature AMI (area under the curve (AUC) = 0.874, 95% CI: 0.826–0.922, $p < 0.001$, sensitivity = 0.843, specificity = 0.795).

Conclusions: Premature AMI is often characterized by STEMI, single-vessel lesions, and a low occurrence of left main coronary artery involvement. Smoking status, HbA1c levels, the non-HDL-C/HDL-C ratio, and the MHR are significantly associated with premature AMI.

Keywords: acute myocardial infarction; premature; coronary angiography; non-HDL-C/HDL-C

Introduction

Research data indicate that cardiovascular diseases are the leading cause of death among noncommunicable diseases, followed by neoplasms and chronic respiratory diseases [1]. The number of deaths from ischemic heart disease is increasing yearly. Acute myocardial infarction (AMI) is a serious medical emergency that requires immediate intervention to prevent sudden cardiac arrest. Premature coronary artery disease (CAD) and premature AMI are typically defined using age cut-offs, with most studies using 55 years for men and 65 years for women [2–5]. However, these cutoff values can vary between 40 and 65 years according to different studies [6–8]. The aforementioned studies tend to define the age limit for premature AMI as men under 55 years old and women under 65 years old, and we adopt this definition. In addition to common risk factors such as smoking, obesity, and hyperlipidemia [9], patients with premature AMI often exhibit plaque erosion and fewer vulnerable plaque features [10]. Treatment for these patients involves aggressive management of risk factors and complete revascularization.

Non-high-density lipoprotein cholesterol (non-HDL-C) is considered a key target for preventing atherosclerotic cardiovascular disease (ASCVD) according to international lipid guidelines [11]. The American Association of Clinical Endocrinologists (AACE) recommends a stringent non-HDL-C target of <80 mg/dL for patients with premature CAD due to their high cardiovascular risk [12]. Furthermore, the ratio of non-HDL-C to HDL-C [13–15] and the monocyte-to-high-density lipoprotein ratio (MHR) [16,17] have been identified as predictors of atherosclerosis, coro-

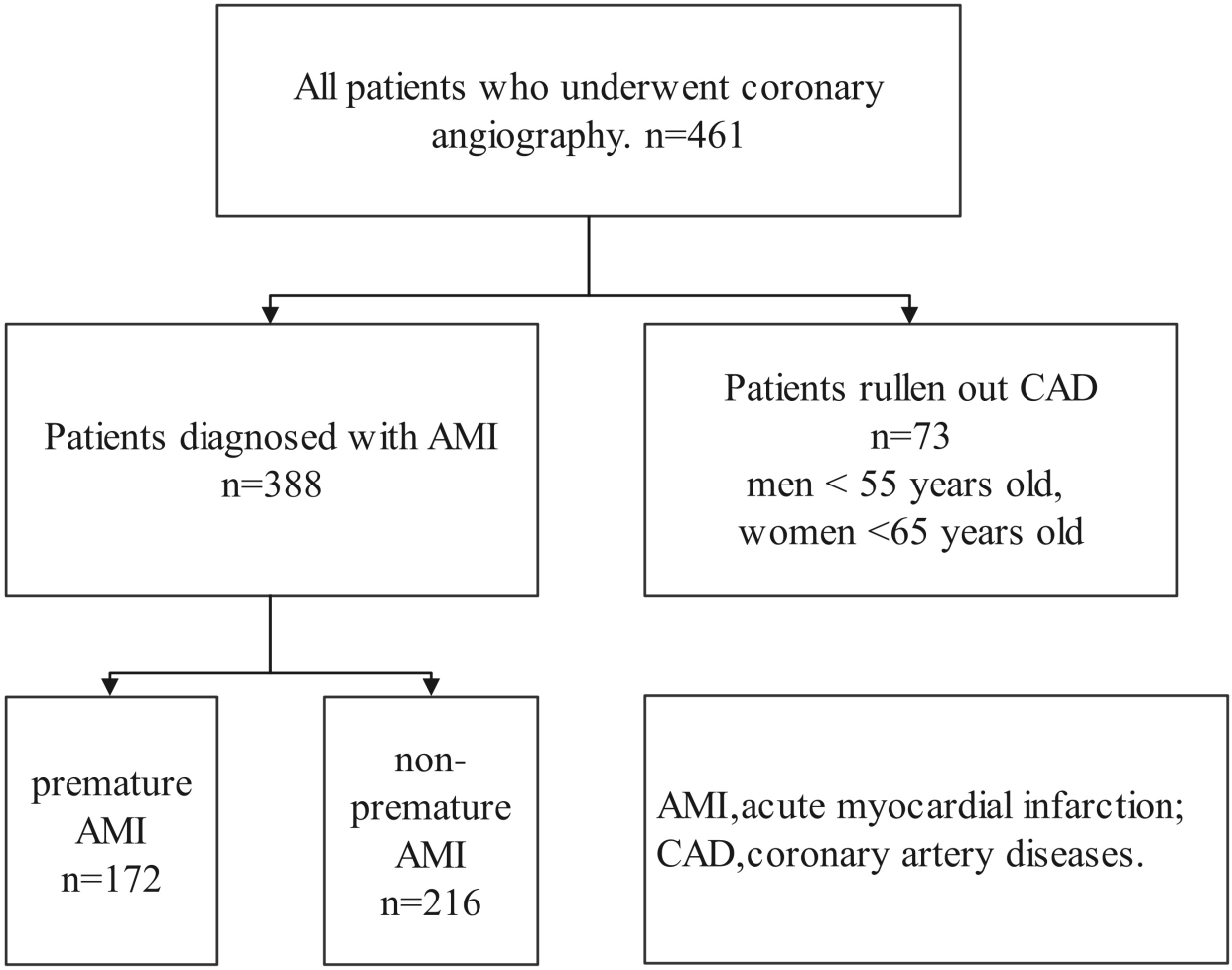

Fig. 1. Flowchart showing the distribution of patients. AMI, acute myocardial infarction; CAD, coronary artery disease.

nary heart disease progression, and CAD severity. However, the majority of above-mentioned studies focused on middle-aged and older populations with fewer data on patients with premature AMI; in addition, there is limited research on the relationship between premature AMI and non-HDL-C, the non-HDL-C/HDL-C ratio, and the MHR. This study aimed to investigate the clinical characteristics, coronary lesion features, and predictive value of the non-HDL-C/HDL-C ratio and MHR in patients with premature AMI. The results emphasize the need for future research to establish the clinical utility of these factors in routine practice and underscore the importance of studying the non-HDL-C/HDL-C ratio and MHR as potential tools for enhancing the ability to prevent, diagnose, and treat premature AMI.

Materials and Methods

Study Population

This study was a retrospective observational analysis conducted at a single medical center. As illustrated in the flowchart in Fig. 1, a total of 388 patients who were diagnosed with AMI at the Department of Cardiovascular Medicine of the First Affiliated Hospital of Chongqing Medical University between January 2022 and April 2023 were included in this study. The sample size calculations were appropriate given actual resource constraints and ethical considerations. The patients were divided into two groups based on age: 172 patients, under 55 years of age for men and under 65 years of age for women, were classified into the premature AMI group, whereas the remaining 216 patients were categorized into the nonpremature AMI group. Additionally, 73 patients (men under 55 years old and women under 65 years old) without CAD were selected as the control group. The inclusion criteria for the study were as follows: (1) met the Fourth Universal Definition of Myocardial Infarction criteria; (2) underwent coronary angiography; and (3) had complete case information. The exclusion criteria were as follows: (1) a history of previous myocardial infarction (MI), coronary artery bypass grafting, or percutaneous coronary intervention; (2) other heart conditions, such as atrial septal defect, ventricular septal defect, or dilated heart disease; (3) heart transplant; (4) pacemaker; or (5) a diagnosis of malignant tumors, severe liver or kidney failure, or myocarditis (as shown in Table 1).

Table 1. The inclusion criteria and exclusion criteria for the study.

Inclusion criteria	Exclusion criteria
Met the Fourth Universal Definition of MI criteria	A history of previous MI/CABG/PCI
Underwent CAG	Other heart conditions: atrial septal defect/ventricular septal defect/dilated heart disease
Had complete case information	Heart transplant
-	Pacemaker
-	A diagnosis of malignant tumors/severe liver/kidney failure/myocarditis

MI, myocardial infarction; CAG, coronary angiography; CABG, coronary artery bypass grafting; PCI, percutaneous coronary intervention.

The study was approved by the ethics committee of the First Affiliated Hospital of Chongqing Medical University (No. K2023-274). We confirmed that all procedures were conducted in compliance with the relevant guidelines and regulations. Simultaneously, informed consent was obtained from patients for all materials and data used.

Clinical Data and Laboratory Tests

The demographic data, clinical characteristics, laboratory test results, and coronary angiography findings of the patients included in our study were collected. The demographic data included age, sex, body mass index (BMI), the presence of hypertension, diabetes status, smoking history, and family history of CAD. Clinical characteristics included the type of MI, Killip classification, blood pressure measurements (systolic [SBP] and diastolic [DBP]), heart rate (HR), length of hospital stay, and use of an intra-aortic balloon pump (IABP). Laboratory test results included glycated hemoglobin (HbA1c), uric acid (UA), creatinine (Cr), urea nitrogen (urea), lipid levels, liver function, routine blood test results, fibrinogen (FIB), D-dimer, and N-terminal pro-B-type natriuretic peptide (NT-proBNP). Cardiac ultrasound and coronary angiography results were also documented. As mentioned above, we comprehensively considered previous research, biological plausibility, hypothesized associations with the outcome variable, and the availability of data in determining the variables to include in the analysis.

Definitions

(1) Coronary artery lesions were assessed by multiple cardiovascular interventional physicians after reviewing the angiography images, and a stenosis $\geq$50% of the coronary artery diameter was considered as a lesion. (2) A single stenotic lesion with a length of $\geq$20 mm was classified as a diffuse lesion. (3) Thrombus burden was evaluated based on the presence of thrombus shadows in coronary angiography images. (4) Intracoronary thrombolysis was defined as the administration of Recombinant Tissue Plasminogen Activator (rt-PA), its analogs, or tirofiban during coronary angiography. (5) Overweight was defined as a BMI of 25–30 kg/m^2, while obesity was defined as a BMI $\geq$30 kg/m^2. (6) Non-HDL-C was calculated as total cholesterol minus HDL-C. (7) The MHR was defined as the ratio of monocytes to high-density lipoprotein (HDL) cholesterol.

Gensini Score

The scoring of the degree of stenosis and the lesion site of the coronary artery was as follows [18]. The basic score was first determined according to the degree of coronary stenosis, with 1 point for stenosis diameter $<$25%, 2 points for $\geq$25% to $<$50%, 4 points for $\geq$50% to $<$75%, 8 points for $\geq$75% to $<$90%, l6 points for $\geq$90% to $<$99%, and 32 points for 99% to 100%, and the corresponding coefficients were determined according to the different coronary branches, as follows: left main coronary artery (LM) lesions $\times$ 5; left anterior descending (LAD) lesions: proximal segment $\times$ 2.5, middle segment $\times$ 1.5, and distal segment $\times$ 1.5, respectively. The coefficients were as follows: LM lesion $\times$ 5; LAD lesion: proximal $\times$ 2.5, middle $\times$ 1.5, distal $\times$ 1; diagonal branch lesion: D1 $\times$ 1, D2 $\times$ 0.5; left circumflex branch (LCX) lesion: proximal $\times$ 2.5, obtuse rim $\times$ 1, distal $\times$ 1, posterior descending branch $\times$ 1, posterior lateral branch $\times$ 0.5; right coronary artery (RCA) lesion: proximal, middle, distal, and posterior descending branches $\times$ 1. The basic stenosis score of each coronary artery was multiplied by the coefficient of the lesion site, yielding a score for that vessel. The sum of the scores of each lesion vessel was the total score of the degree of stenosis of the patient's coronary artery lesion. The Gensini score was determined by two different physicians.

Statistical Analysis

Statistical analysis was conducted using IBM SPSS Statistics 25.0 (SPSS Inc., Armonk, NY, USA). Normally distributed quantitative variables were presented as the mean and standard deviation (SD) and group differences were assessed using independent sample t tests. Non-normally distributed variables were expressed as medians and ranges and group differences were analyzed using the Mann–Whitney–Wilcoxon test. Categorical variables were expressed as number and percentage and were compared using Pearson's chi-square test or Fisher's exact test. Binary logistic regression models were used for univariate analyses to identify risk factors associated with premature AMI. All variables with $p \geq 0.05$ were included in the multiple logistic regression model. Odds ratios (ORs) and 95% confidence intervals (CIs) were calculated. p value < 0.05 was considered statistically significant for all analyses.

Table 2. Sociodemographic, clinical, and biochemical characteristics of the enrolled AMI patients.

	Premature	Nonpremature	$\chi^2/t/Z$	p value
	AMI (n = 172)	AMI (n = 216)		
Age (years)	48.17 ± 7.83	69.32 ± 7.93	–26.245	<0.001
Male (n, %)	145 (84.3%)	168 (77.8%)	2.614	0.106
BMI (kg/m^2)	25.86 ± 3.25	24.18 ± 3.47	4.887	<0.001
Overweight (n, %)	87 (50.6%)	83 (38.4%)	5.747	0.017
Obesity (n, %)	17 (9.9%)	7 (3.2%)	7.282	0.007
Hypertension (n, %)	71 (41.3%)	137 (63.4%)	18.884	<0.001
Diabetes (n, %)	42 (24.4%)	81 (37.5%)	7.568	0.006
Smoking (n, %)	116 (67.4%)	83 (38.4%)	32.268	<0.001
Family history of CAD (n, %)	23 (13.4%)	13 (6.0%)	6.151	0.013
Clinical features	-	-	-	-
STEMI (n, %)	123 (71.5%)	126 (58.3%)	7.233	0.007
NSTEMI (n, %)	49 (28.5%)	90 (41.7%)	-	-
Killip class	-	-	-	-
I (n, %)	141 (82.0%)	162 (75.0%)	2.724	0.099
II (n, %)	19 (11.0%)	31 (14.4%)	-	-
III (n, %)	5 (2.9%)	9 (4.2%)	-	-
IV (n, %)	7 (4.1%)	14 (6.5%)	-	-
Length of hospitalization (days)	7 (6, 10)	8 (7, 10.75)	–2.54	0.011
SBP (mmHg)	128.18 ± 22.64	132.60 ± 22.87	–1.898	0.058
DBP (mmHg)	83.06 ± 17.02	79.62 ± 15.10	2.109	<0.001
HR (bpm)	84.5 (74.25, 95)	80 (71, 89)	–2.12	0.034
IABP (n, %)	4 (2.3%)	3 (1.4%)	0.093	0.761
TC (mmol/L)	4.76 (3.89, 5.55)	4.42 (3.7, 5.32)	–1.831	0.067
TG (mmol/L)	1.76 (1.30, 2.83)	1.47 (1.07, 2.03)	–3.745	<0.001
HDL-C (mmol/L)	0.96 (0.82, 1.1)	1.04 (0.87, 1.23)	–3.776	<0.001
LDL-C (mmol/L)	3.08 (2.25, 3.72)	2.745 (2.18, 3.62)	–1.553	0.12
ApoA1 (g/L)	1.18 ± 0.22	1.23 ± 0.23	–2.353	0.019
ApoB (g/L)	1.08 ± 0.36	1.00 ± 0.31	2.469	0.014
Lp(a) (mg/L)	108 (50.25, 234.75)	155.5 (58.5, 437)	–2.349	0.019
Non-HDL-C (mmol/L)	3.795 (2.81, 4.635)	3.44 (2.6, 4.23)	–2.617	0.009
Non-HDL-C/HDL-C	3.83 (2.88, 5.27)	3.1 (2.39, 4.23)	–4.393	<0.001
WBC (10^9/L)	11.01 (8.58, 14.1)	9.20 (7.30, 11.59)	–4.354	<0.001
Hemoglobin (g/L)	146.09 ± 16.01	134.60 ± 18.83	6.374	<0.001
Platelets (10^9/L)	246.80 ± 83.82	220.20 ± 84.15	3.105	0.002
MPV (fl)	10.77 ± 1.33	11.15 ± 1.23	–2.927	0.004
Neutrophils (10^9/L)	8.28 (5.86, 11.325)	6.94 (5.37, 9.25)	–3.171	0.002
Lymphocytes (10^9/L)	1.59 (1.20, 2.17)	1.34 (0.96, 1.84)	–3.808	<0.001
Monocytes (10^9/L)	0.6 (0.42, 0.80)	0.575 (0.45, 0.82)	–0.477	0.633
MHR	0.63 (0.48, 0.93)	0.58 (0.37, 0.8)	–2.425	0.015
ALT (U/L)	37 (27, 48.75)	28 (19.0, 49.0)	–3.775	<0.001
AST (U/L)	76.5 (37, 200.5)	58.0 (32.25, 135.25)	–1.842	0.066
LDH (U/L)	283.5 (210.5, 472)	269 (198.5, 416.75)	–0.774	0.439
Urea (mmol/L)	5.2 (4.3, 6.28)	6.25 (5.1, 7.6)	–5.42	<0.001
Cr (μmol/L)	73 (61, 86)	76 (64.25, 93.0)	–1.8	0.072
UA (μmol/L)	377.19 ± 113.84	344.98 ± 98.16	2.986	0.003
FIB (g/L)	3.2 (2.66, 4.2)	3.485 (2.82, 4.52)	–2.05	0.04
D-dimer (ng/mL)	288 (209, 492.75)	523 (358, 907.25)	–7.685	<0.001
NT-proBNP (pg/mL)	406 (126, 961)	911.5 (286.75, 2500)	–5.168	<0.001
HbA1c (%)	6.0 (5.7, 6.78)	6.2 (5.8, 7.58)	–2.282	0.022
LVEF (%)	53.95 ± 9.41	52.52 ± 10.39	1.396	0.164
FS (%)	29 (24, 33)	29 (22, 32)	–1.168	0.243
Mural thrombus (n, %)	5 (2.9%)	2 (0.9%)	2.121	0.145
Ventricular aneurysm (n, %)	15 (8.7%)	19 (8.8%)	0.001	0.979

BMI, body mass index; STEMI, ST-segment elevation myocardial infarction; NSTEMI, non-ST-segment elevation myocardial infarction; SBP, systolic blood pressure; DBP, diastolic blood pressure; HR, heart rate; IABP, intra-aortic balloon pump; TC, total cholesterol; TG, triglyceride; HDL-C, high-density lipoprotein cholesterol; LDL-C, low-density lipoprotein cholesterol; ApoA1, apolipoprotein A1; ApoB, apolipoprotein B; Lp(a), lipoprotein(a); WBC, white blood cell count; MPV, mean platelet volume; MHR, monocyte-to-high-density lipoprotein ratio; ALT, alanine aminotransferase; AST, aspartate aminotransferase; LDH, lactate dehydrogenase; urea, urea nitrogen; Cr, creatinine; UA, uric acid; FIB, fibrinogen; NT-proBNP, N-terminal pro-B-type natriuretic peptide; HbA1c, glycated hemoglobin; LVEF, left ventricular ejection fraction; FS, fractional shortening.

Results

Baseline Characteristics and Clinical Features

The study included 461 patients, 74.6% of whom were men, and the mean age was 58.71 ± 12.72 years. Among them, 388 patients had AMI, and 73 were controls. Characteristics of AMI patients are shown in Table 2. Of the patients with AMI, 172 patients were classified as having premature AMI (84.3% men), with a mean age of 48.17 ± 7.83 years, while 216 patients were classified as having nonpremature AMI (77.8% men), with a mean age of 69.32 ± 7.93 years. The mean BMI in the premature AMI group was 25.86 ± 3.25 kg/m^2, whereas it was 24.18 ± 3.47 kg/m^2 in the nonpremature AMI group ($p < 0.001$). Smoking ($p < 0.001$), family history of CAD ($p = 0.013$), overweight ($p = 0.017$), and obesity ($p = 0.007$) were more prevalent in the premature AMI group than in the nonpremature AMI group. In contrast, hypertension ($p < 0.001$) and diabetes ($p = 0.006$) were more common in the nonpremature AMI group.

In terms of clinical characteristics, the majority of premature AMI patients had ST-elevation myocardial infarction (STEMI) ($p = 0.007$). Upon admission, patients with premature AMI had higher diastolic blood pressure ($p < 0.001$) and heart rate ($p = 0.034$). The average length of hospital stay was shorter for patients with premature AMI, averaging 7 days, compared to 8 days for the control group ($p = 0.011$). Additionally, there were no significant differences between the two groups regarding Killip classification or IABP use.

Laboratory Test Variables

In terms of lipid levels, individuals with premature AMI had higher levels of triglycerides ($p < 0.001$), apolipoprotein B ($p = 0.014$), and non-HDL-C ($p = 0.009$) and a higher non-HDL-C/HDL-C ratio ($p < 0.001$) than those with nonpremature AMI. Conversely, the nonpremature AMI group had higher levels of HDL cholesterol ($p < 0.001$), apolipoprotein A1 ($p = 0.019$), and lipoprotein (a) ($p = 0.019$). No significant differences were identified in total cholesterol or low-density lipoprotein cholesterol levels between the two groups. Additionally, patients with premature AMI exhibited elevated white blood cell counts, hemoglobin levels, platelet counts, neutrophil counts, lymphocyte counts, uric acid levels, and MHR. In contrast, the nonpremature AMI group had greater mean platelet volume and alanine aminotransferase, urea nitrogen, fibrinogen, D-dimer, NT-proBNP, and glycated hemoglobin (HbA1c) levels (all p values < 0.05). Cardiac ultrasound revealed no significant differences in the left ventricular ejection fraction (LVEF), fractional shortening (FS), mural thrombus, or ventricular aneurysm between the premature and nonpremature AMI groups (Table 2).

Coronary Angiography Results

The Gensini score is a useful tool for assessing the severity of coronary artery lesions. Analysis revealed no significant difference in the Gensini score between patients with premature AMI and those with nonpremature AMI ($p = 0.384$) (Table 3). However, there were differences in lesion vessel sites, with greater proportions of left main coronary artery lesions ($p = 0.003$), left circumflex branch lesions ($p = 0.012$), and right coronary artery lesions ($p = 0.036$) in the nonpremature AMI group. Patients with premature AMI were more likely to have single-vessel lesions ($p < 0.001$). No statistically significant variation was observed in the application of coronary stenting between the premature AMI group (83.7%) and the nonpremature AMI group (86.6%, $p = 0.43$). However, patients with premature AMI had less stents implanted ($p = 0.013$) than those with nonpremature AMI. Furthermore, no significant differences were identified between the two groups concerning drug-coated balloons, intracoronary thrombolysis, or transcatheter thrombus aspiration.

Logistic Regression Analysis of Premature AMI

As shown in Table 4, binary logistic regression analysis was performed to compare the premature AMI group and the control group (control group = 0, premature AMI group = 1). Variables with $p < 0.05$ in the single binary logistic regression analysis were included in the multiple binary logistic regression model. The final results indicated that smoking (adj. Odds ratio (OR) 4.454, 95% confidence interval (CI): 1.836–10.806, $p = 0.001$), HbA1c level (adj. OR 2.261, 95% CI: 1.219–4.193, $p = 0.01$), non-HDL-C/HDL-C (adj. OR 4.394, 95% CI: 1.204–16.031, $p = 0.025$), and MHR (adj. OR 6.164, 95% CI: 1.386–27.417, $p = 0.017$) were significantly associated with premature AMI after adjusting for confounders, including age, sex, diabetes, BMI, triglyceride (TG), HDL-C, non-HDL-C, and UA.

ROC Prediction Model for Premature AMI

This study revealed that HbA1c, the non-HDL-C/HDL-C ratio, the MHR, and smoking were significantly associated with premature AMI. Receiver operating characteristic curve analysis was conducted to determine the area under the curve (AUC) and cutoff values. HbA1c had an AUC of 0.691 (95% CI: 0.621–0.76, $p < 0.001$), the non-HDL-C/HDL-C ratio had an AUC of 0.739 (95% CI: 0.677–0.801, $p < 0.001$), the MHR had an AUC of 0.801 (95% CI: 0.74–0.862, $p < 0.001$), and smoking had an AUC of 0.728 (95% CI: 0.659–796, $p < 0.001$) (Fig. 2A,B). The cutoff values were 5.95% for HbA1c (sensitivity = 0.535, specificity = 0.795), 3.423 for the non-HDL-C/HDL-C ratio (sensitivity = 0.628, specificity = 0.781), and 0.498 for the MHR (sensitivity = 0.727, specificity = 0.795). The AUC was 0.856 (95% CI: 0.805–0.908, $p < 0.001$, sensitivity = 0.872, specificity = 0.712) for the combination of HbA1c, non-HDL-C/HDL-C ratio, and MHR (Fig. 2C), and

Table 3. Comparison of coronary angiography results between the two groups.

	Premature	Nonpremature	$\chi^2/t/Z$	*p* value
	AMI (n = 172)	AMI (n = 216)		
Gensini score	54 (34, 82)	56 (38, 82)	–0.871	0.384
Lesioned vessels	-	-	-	-
LM (n, %)	7 (4.1%)	21 (9.7%)	4.569	**0.033**
LAD (n, %)	136 (79.1%)	186 (86.1%)	3.363	0.067
LCX (n, %)	75 (43.6%)	122 (56.5%)	6.352	**0.012**
RCA (n, %)	86 (50.0%)	131 (60.6%)	4.405	**0.036**
Single-vessel lesion (n, %)	81 (47.1%)	58 (26.9%)	17.064	**<0.001**
Coronary stenting (n, %)	144 (83.7%)	187 (86.6%)	0.622	0.43
Number of stents	1 (1, 2)	2 (1, 2)	–2.489	**0.013**
Length of stent (mm)	23 (18, 28)	(18.75, 28.88)	–1.156	0.248
Stent diameter (mm)	2.85 (2.5, 3.23)	2.75 (2.5, 3.0)	–1.465	0.143
Diffuse lesion (n, %)	31 (18%)	64 (29.6%)	6.976	**0.008**
Vascular occlusion (n, %)	97 (56.4%)	103 (47.7%)	2.909	0.088
DCB (n, %)	11 (6.4%)	15 (6.9%)	0.046	0.83
Thrombus burden (n, %)	30 (17.4%)	30 (13.9%)	0.925	0.336
Intracoronary thrombolysis (n, %)	22 (12.8%)	21 (9.7%)	0.915	0.339
Transcatheter thrombus aspiration (n, %)	7 (4.1%)	3 (1.4%)	1.777	0.183
MB (n, %)	6 (3.5%)	10 (4.6%)	0.315	0.574
Operation time (minutes)	60 (43, 88.75)	71 (48, 100)	–2.819	**0.005**

LM, left main coronary artery; LAD, left anterior descending; LCX, left circumflex branch; RCA, right coronary artery; DCB, drug-coated balloon; MB, myocardial bridging.
Significant *p* values are indicated in bold.

Table 4. Logistic regression analysis of premature acute myocardial infarction.

Variables	Univariate analysis		Multivariate analysis	
	OR (95% CI)	*p* value	Adjusted OR (95% CI)	*p* value
Sex (male)	0.137 (0.074–0.255)	<0.001	1.493 (0.474–4.705)	0.494
Age	0.93 (0.892–0.969)	<0.001	0.981 (0.922–1.043)	0.532
Hypertension	1.435 (0.332–1.757)	0.218	-	-
Diabetes	4.394 (1.661–11.620)	0.003	1.735 (0.438–6.864)	0.432
Smoking	7.379 (3.892–13.991)	<0.001	4.454 (1.836–10.806)	**0.001**
Family history of CAD	1.254 (0.533–2.951)	0.604	-	-
BMI	1.094 (1.005–1.191)	0.038	0.923 (0.811–1.049)	0.220
TC	1.067 (0.869–1.310)	0.536	-	-
TG	1.64 (0.511–1.105)	0.001	0.744 (0.507–1.093)	0.132
HDL-C	0.02 (0.006–0.68)	<0.001	2.159 (0.09–51.69)	0.635
LDL-C	1.107 (0.871–1.407)	0.406	-	-
HbA1c	2.266 (1.439–3.569)	<0.001	2.261 (1.219–4.193)	**0.010**
Non-HDL-C	1.308 (1.038–1.649)	0.023	0.359 (0.114–1.127)	0.079
Non-HDL-C/HDL-C	1.968 (1.53–2.532)	<0.001	4.394 (1.204–16.031)	**0.025**
MHR	45.368 (11.92–172.678)	<0.001	6.164 (1.386–27.417)	**0.017**
UA	1.006 (1.003–1.009)	<0.001	1.003 (0.998–1.007)	0.203

Odds ratios (ORs) refer to a 1-SD increase in continuous variables. CI, confidence interval; OR, Odds ratio.
Significant *p* values are indicated in bold.

when smoking was included, the AUC was 0.874 (95% CI: 0.826–0.922, $p < 0.001$, sensitivity = 0.843, specificity = 0.795) (Fig. 2D). All models had good predictive value.

Discussion

In this retrospective study, smoking, a high BMI, a family history of CAD, triglyceridemia, and low HDL-C levels were more prevalent in patients who experienced premature AMI. Conversely, the prevalence of diabetes and

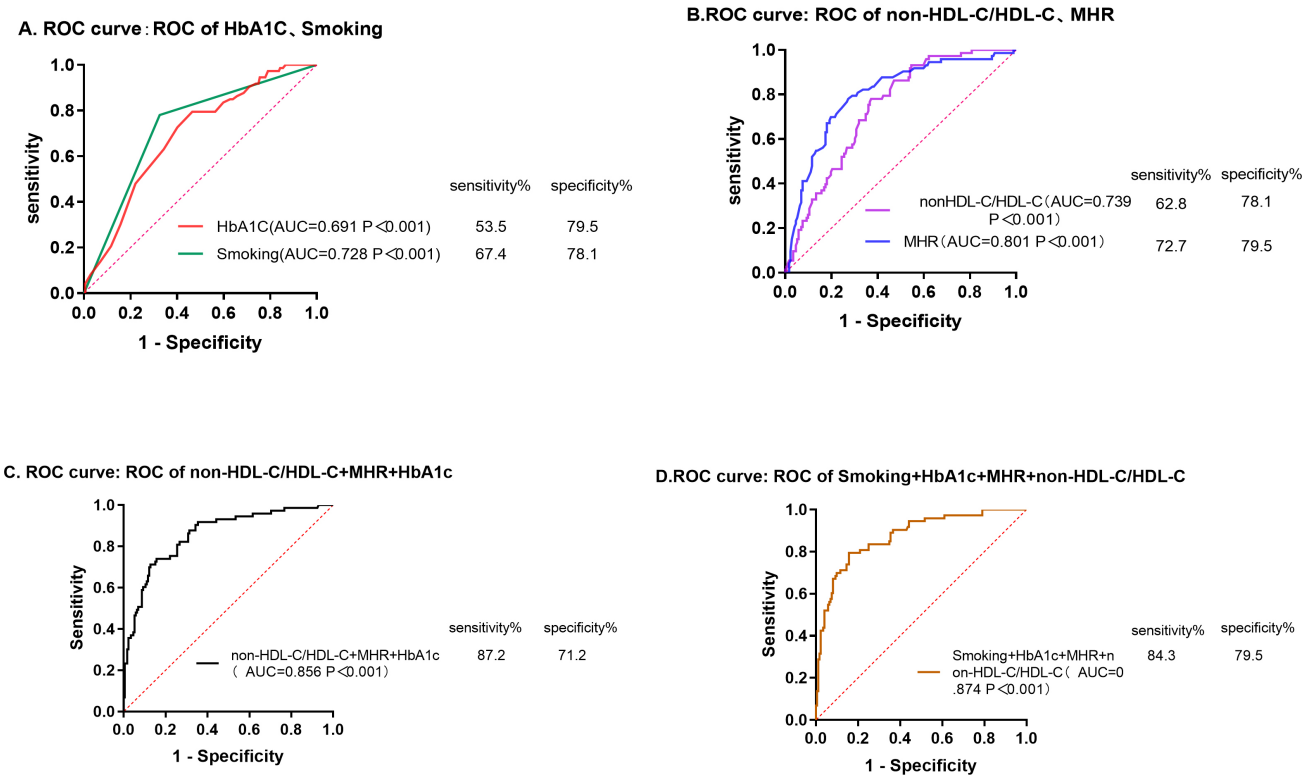

Fig. 2. ROC curve analysis of the predictive values of smoking status, HbA1c, non-HDL-C/HDL-C, and MHR. The cutoff points for HbA1c, non-HDL-C/HDL-C, and MHR were 5.95, 3.423, and 0.5, respectively. (A) HbA1c (AUC = 0.691, 95% CI 0.621–0.761, $p < 0.001$); smoking (AUC = 0.728, 95% CI 0.659–0.796, $p < 0.001$). (B) Non-HDL-C/HDL-C ratio (AUC = 0.739, 95% CI 0.677–0.801, $p < 0.001$); MHR (AUC = 0.801, 95% CI 0.740–0.862, $p < 0.001$). (C) Combined HbA1c, non-HDL-C/HDL-C ratio, and MHR (AUC = 0.856, 95% CI 0.805–0.908, $p < 0.001$, sensitivity 0.872, specificity 0.712). (D) Combined HbA1c, non-HDL-C/HDL-C ratio, MHR, and smoking (AUC = 0.874, 95% CI 0.826–0.922, $p < 0.001$, sensitivity 0.843, specificity 0.795). ROC, receiver operating characteristic; AUC, area under the curve.

hypertension was lower in the premature AMI group compared to the nonpremature AMI group, which is consistent with previous studies [2,3,19]. The majority of patients in both age groups were male. There was a predominance of ST-segment elevation myocardial infarction (STEMI) among patients with premature AMI. According to a study by Deora *et al.* [20], STEMI accounts for the majority of patients with premature acute coronary syndrome. Similarly, Mahendiran *et al.* [21] investigated 7070 patients aged <50 years with acute coronary syndrome and reported that 63.8%, 33%, and 3.2% of them exhibited STEMI, non-STEMI, and unstable angina, respectively. This phenomenon may be due to a lack of anastomoses and collaterals in younger individuals, which can lead to more severe myocardial damage in the event of total occlusion and result in higher rates of STEMI in this patient population [9].

In patients with coronary artery disease, the LAD branch is commonly affected, while left main coronary artery lesions are rare. Patients with premature AMI tend to have a greater proportion of single-branch vascular lesions than those with nonpremature AMI. Research by Wittlinger *et al.* [22] involving 5854 MI patients revealed that the LAD was the most frequently affected branch in both younger and older patients, with approximately two-thirds of younger patients having one-vessel disease. Older patients are more likely to have multiple-vessel disease due to the increased incidence of atherosclerosis with age. In our study, there was no significant difference in the Gensini score between the two groups, but there were differences in the number of stents, diffuse lesion presence, and operation time, indicating more severe coronary artery disease in patients with nonpremature AMI. Percutaneous coronary intervention (PCI), such as percutaneous transluminal coronary angioplasty (PTCA), coronary stenting, rotational atherectomy (RA), and intracoronary thrombus aspiration, is commonly used in clinical practice for the treatment of AMI. With advancements in technology and techniques, PCI is becoming sophisticated and crucial for the long-term prognosis of patients. The high percentage of primary coronary stenting in both age groups (83.7% vs. 86.6%) indicates that this first-line treatment is well established; this result is consistent with those of previous studies. However, the use of drug-coated balloons (DCBs) was not as prevalent in patients with a first AMI (6.4% vs. 6.9%). Al-

fonso noted that DCBs are suitable for selected de novo coronary lesions (small vessels, diffuse disease, and side branches) and demonstrated that DCBs are safe and effective for coronary in-stent restenosis (ISR) [23]. In terms of coronary clinical experience, DCBs are used most in ISR, and the observations of Giacoppo *et al.* [24] further confirmed that DCB angioplasty and repeat stenting with drug-eluting stents are equally effective and safe for the treatment of bare metal stent ISR.

Multiple binary logistic regression analysis revealed that smoking, HbA1c, the non-HDL-C/HDL-C ratio, and the MHR were independent predictors of premature AMI. Interestingly, when smoking, HbA1c, non-HDL-C/HDL-C, and MHR were considered together, their predictive value was greater. Notably, the non-HDL-C/HDL-C ratio was found to have a stronger association with premature AMI, with an odds ratio of 1.968 in univariate analysis and 4.394 after adjusting for confounders, suggesting that it may be a stronger predictor of premature AMI.

A prospective study with a follow-up period of 9.1 years revealed that a significant percentage of patients (58.6%) with premature AMI continued smoking after their initial heart attack. Continued smoking was identified as the most significant long-term predictor for recurrent major adverse cardiovascular events (MACEs) in young survivors of premature AMI [25]. Scarpone *et al.* [26] reported that smoking was significantly related to STEMI (OR 1.45; 95% CI: 1.32–1.59) compared with non-STEMI, and smokers experienced myocardial infarction (either STEMI or non-STEMI) much earlier than nonsmokers among both women and men. Bujak *et al.* [27] reported that tobacco use was an independent risk factor for 36-month mortality in patients with STEMI treated with primary percutaneous coronary intervention [26]. These findings highlight the importance of implementing effective smoking cessation policies to improve the long-term prognosis of young coronary patients. According to Mallah *et al.* [28], smoking can cause changes in lipids, inflammation, and vascular dysregulation, leading to the occurrence and progression of atherosclerosis. Therefore, it is important to reduce cigarette smoke exposure and improve air pollution environment.

HbA1c is a reliable indicator of average blood glucose levels over a period of 2 to 3 months and is considered the gold standard for assessing glycemic control and predicting diabetic complications (an HbA1c level $\geq$6.5% is used in the diagnosis of diabetes mellitus, reflecting an individual's glycemic control over the past few months). Research has shown that HbA1c levels are associated with the severity of CAD in both diabetic and nondiabetic patients [29]. Che *et al.* [30] speculated that patients without diabetes mellitus who had an HbA1c >5.9% were at increased risk of having a higher Killip class and multivessel CAD and that the HbA1c level was positively related to CAD progression in diabetes mellitus patients. We obtained a cutoff value of 5.95% for HbA1c, which can serve as a clinical reference for secondary prevention of AMI. The HbA1c level was shown to be associated with the severity and complexity of coronary lesions, and the HbA1c level, rather than glucose, was shown to be a significant predictor of MACEs after AMI in nondiabetic patients [31–33]. Elevated HbA1c indicates poor glycemic control in the past, which can lead to damage to vascular endothelial cells, impaired vascular function, increased extracellular matrix formation, and abnormal cell proliferation.

In recent years, there has been a growing focus on the non-HDL-C to HDL-C ratio (non-HDL-C/HDL-C), which has been closely linked to various health conditions, such as metabolic syndrome, nonalcoholic steatohepatitis [34], diabetes mellitus [35], and atherosclerosis [36]. Research has shown that this ratio plays a significant role in the risk and progression of CAD and is an independent predictor of the severity of CAD and MACEs [14,15,37]. Winter's [38] study showed that non-HDL and remnant cholesterol were strongly associated with adverse outcomes and that patients with premature MI had a lipid phenotype with a predominance of elevated triglyceride-rich lipoproteins, which was considered high risk [38]. Interestingly, the present study also revealed that the MHR was significantly associated with premature AMI and that its AUC showed good predictive value. The MHR, a newly identified indicator of inflammation and oxidative stress, has been widely explored in the field of cardiovascular disorders, including AMI [39,40]. Therefore, using a combination of the non-HDL-C/HDL-C ratio and the MHR may offer a more comprehensive and accurate assessment of the risk of early AMI than relying solely on individual lipid measurements such as LDL-C and HDL-C.

Identifying modifiable risk factors for premature AMI, including smoking, HbA1c, the non-HDL-C/HDL-C ratio, and the MHR, provides an opportunity to optimize clinical strategies and research. The smoking-AMI link indicates the need for smoking cessation programs in routine care, with emphasis on high-risk individuals. HbA1c's prognostic significance in diabetes necessitates tighter glycemic targets, particularly for those with cardiovascular family history. The non-HDL-C/HDL-C ratio's stronger correlation with premature AMI suggests its incorporation into cardiovascular risk assessments, surpassing traditional lipid profiling. The MHR's predictive value highlights the role of inflammation in cardiovascular disease, advocating for enhanced clinical focus on inflammatory markers. Further research should elucidate health disparities influencing premature AMI and identify novel biomarkers to refine risk prediction. Targeted interventions and research on these predictors could reduce premature AMI incidence and improve cardiovascular outcomes.

Several limitations of this study should be highlighted. First, this study was conducted in a single center and relied on retrospective data, which makes it less reliable than a prospective design and may have introduced selection bias.

Further research is needed to understand the long-term impact of these risk factors on cardiovascular health, which can provide insights into the progression from risk factor exposure to clinical events. Second, since this is an observational study, the data cannot be randomly assigned, which means that potential confounding factors may affect the outcomes. With the predictive value of these factors established, intervention studies could be designed to evaluate the effectiveness of targeted lifestyle modifications and pharmacological interventions. Third, the sample size was limited and may not be fully representative of the general population. Finally, the control group included patients who were admitted to the hospital with suspected symptoms and who underwent coronary angiography to rule out coronary artery disease, suggesting that these enrolled patients may have risk factors for coronary artery disease. Therefore, this group of patients was not a complete blank control.

Conclusions

The increasing prevalence of MI among younger individuals is a pressing concern that has been the focus of much discussion in recent years. Our study suggests that adopting healthy habits, including refraining from smoking and tobacco consumption, is essential for preventing premature AMI. It is imperative for patients to quit smoking without delay. Furthermore, HbA1c, the non-HDL-C/HDL-C ratio, and the MHR have been identified as key influencing factors for premature AMI. These factors could be crucial in predicting and targeting treatment for young patients.

Availability of Data and Materials

The data and analyses conducted in this study are accessible upon request to the principal investigator, contingent upon the stipulation of reasonable grounds for the inquiry. Due to the confidential and sensitive aspects of the patient data, the primary data and associated materials are not intended for public dissemination.

Author Contributions

RW and SQT contributed to the acquisition, analysis, and drafting of the manuscript. Both authors have made equally significant contributions to the work. MZT contributed to the interpretation of data and the preparation of graphs. LYG contributed to conception and design of the article, acquisition of data, supervision and reviewed the manuscript. All authors have read and approved the final manuscript. All authors contributed significantly to editorial changes of important content. All authors have participated sufficiently in the work and agreed to be accountable for all aspects of the work.

Ethics Approval and Consent to Participate

The study was approved by the ethics committee of the First Affiliated Hospital of Chongqing Medical University (No. K2023-274). Simultaneously, informed consent was obtained from patients for all materials and data used.

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Conflict of Interest

The authors declare no conflict of interest.

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