ARCHIVES OF THE TURKISH SOCIETY OF CARDIOLOGY

Official journal of the TURKISH SOCIETY OF CARDIOLOGY

Comparison of CHA_2DS_2-VASc, C_2HEST, HAT_2CH_2, SYNTAX, GRACE, and SYNTAX II Scores for Predicting New-Onset Atrial Fibrillation Complicating Acute Myocardial Infarction

Akut Miyokard Enfarktüs Seyrinde Yeni Gelişen Atriyal Fibrilasyonu Öngörmede CHA_2DS_2-VASc, C_2HEST, HAT_2CH_2, SYNTAX, GRACE VE SYNTAX 2 Skorlarının Karşılaştırılması

ABSTRACT

Objective: This study evaluated the most effective scoring system for predicting new-onset atrial fibrillation (NOAF) during acute myocardial infarction (AMI). Identifying the best predictive tool may help clinicians select the most appropriate personalized treatment based on individual risk scores to prevent NOAF complicating AMI.

Method: A total of 2,206 patients diagnosed with AMI between June 2021 and January 2023 were included in this study. After excluding cases with missing data, univariable and multivariable analyses were conducted on 1,672 patients to assess the association between baseline characteristics and the development of atrial fibrillation. The CHA_2DS_2-VASC (Congestive heart failure, Hypertension, Age ≥ 75 years, Diabetes mellitus, Stroke/TIA/thromboembolism, Vascular disease, Age 65-74 years, Sex category), C_2HEST (Coronary artery disease, Chronic obstructive pulmonary disease, Hypertension, Elderly [age ≥ 75], Systolic heart failure, Thyroid disease), HAT_2CH_2 (Hypertension, Age > 75, Stroke/TIA, Chronic obstructive pulmonary disease, Heart failure), SYNTAX (Synergy Between PCI with Taxus and Cardiac Surgery), GRACE 2.0 (Global Registry of Acute Coronary Events), and SYNTAX II scores were calculated for each patient.

Results: Receiver operating characteristic (ROC) analysis showed that the SYNTAX score (SxS) had the highest predictive value for NOAF during AMI, with an area under the curve (AUC) of 0.785 (95% confidence interval [CI]: 0.767-0.802, P < 0.001), followed by the SYNTAX II score (SxSII) with an AUC of 0.747 (95% CI: 0.728-0.765, P < 0.001), and the GRACE 2.0 risk score (RS) with an AUC of 0.740 (95% CI: 0.721-0.758, P < 0.001). It was shown that the modified scores (created by incorporating hemoglobin A1c [HbA1c] levels), the primary independent predictive parameter in this study, into the existing risk models demonstrated higher predictive value for NOAF (C-statistic: 0.784-0.794).

Conclusion: Combining HbA1c levels with SxS yielded the highest diagnostic performance for predicting NOAF during AMI. In this study, while SxS outperformed other risk models, the GRACE 2.0 and SxSII scores also demonstrated relatively strong predictive value and were superior to the CHA_2DS_2-VASC, C_2HEST, and HAT_2CH_2 scores for predicting NOAF in the setting of AMI.

Keywords: Acute myocardial infarction, atrial fibrillation, hemoglobin A1c, SYNTAX score

ÖZET

Amaç: Bu çalışma, akut miyokard enfarktüsü (AMI) sırasında ortaya çıkan yeni başlangıçlı atriyal fibrilasyonu (NOAF) öngörmede en etkili skorlama yöntemini belirlemeyi amaçlamıştır. Bu sayede, AMI'ye eşlik eden NOAF'nin önlenmesi için, öngörülen risk skorlarına göre hekimlerin en uygun kişiselleştirilmiş tedaviyi seçmesine rehberlik edilebilir.

Yöntem: Haziran 2021 ile Ocak 2023 arasında toplam 2206 AMI hastası bu çalışmaya dâhil edilmiştir. Eksik veri nedeniyle, 1672 hasta için başlangıç faktörleri ile atriyal fibrilasyon gelişimi arasındaki ilişkileri değerlendirmek üzere tek değişkenli ve çok değişkenli analizler kullanılmıştır. Her bir hasta için CHA_2D_2-VASC, C_2HEST, HAT_2CH_2, SYNTAX, GRACE 2.0 ve SYNTAX II skorları hesaplanmıştır.

Bulgular: AMI sürecinde NOAF'ı öngörmek amacıyla yapılan ROC analizinde, SYNTAX skoru (SxS) için eğri altında kalan alan 0.785 (GA %95 0.767–0.802, P < 0.001) olarak bulunmuş;

ORIGINAL ARTICLE
KLİNİK ÇALIŞMA

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Received: February 19, 2025
Accepted: July 09, 2025

Cite this article as: Bilgin Doğan N, Dolu AK, Ekinci S, Şimşek EÇ. Comparison of CHA_2DS_2-VASc, C_2HEST, HAT_2CH_2, SYNTAX, GRACE, and SYNTAX II Scores for Predicting New-Onset Atrial Fibrillation Complicating Acute Myocardial Infarction. *Turk Kardiyol Dern Ars.* 2025;53(0):000-000.

DOI: 10.5543/tkda.2025.38852

bunu sırasıyla SYNTAX II skoru (SxSII) 0.747 (GA %95 0.728–0.765, P < 0.001) ve GRACE 2.0 risk skoru (RS) 0.740 (GA %95 0.721–0.758, P < 0.001) izlemiştir. Çalışmanın bağımsız en güçlü öngörücü parametresi olan HbA1c düzeyinin, bu risk skorlarına bir puanlama parametresi olarak eklenmesiyle oluşturulan "modifiye" skorların NOAF'ı öngörmedeki değerinin daha yüksek olduğu gösterilmiştir (C istatistiği, 0.784–0.794).

Sonuç: HbA1c düzeyinin SxS ile birleştirilmesi, AMI sırasında NOAF tahmini açısından en iyi tanısal performansı sağlamıştır. Bu çalışmada, SxS diğer risk skorlarından daha iyi performans gösterirken, GRACE 2.0 risk skoru ile SxSII skorunun da görece yüksek bir öngörü değeri olduğu ve NOAF tahmini açısından CHA_2D_2-VASC, C_2HEST ve HAT_2CH_2 skorlarından daha başarılı olduğu saptanmıştır.

Anahtar Kelimeler: Akut miyokard enfarktüsü, atriyal fibrilasyon, hemoglobin A1c, SYNTAX skoru

Patients with myocardial infarction (MI)[1] frequently develop new-onset atrial fibrillation (NOAF), a condition strongly associated with increased mortality and adverse in-hospital outcomes, including prolonged length of stay, higher complication rates, and an increased need for intensive care or readmission.[2,3] Several clinical risk scores have been developed to assess atrial fibrillation (AF) risk in the general population, such as the Framingham Heart Study (FHS) score, the Atherosclerosis Risk in Communities (ARIC) score, and the Cohorts for Heart and Aging Research in Genomic Epidemiology Atrial Fibrillation (CHARGE-AF) score. Although advanced age, pre-existing heart failure, and extensive myocardial infarction have long been recognized as risk factors for atrial fibrillation during acute myocardial infarction (AMI), particularly in studies from the fibrinolytic therapy era,[4] there is a paucity of research on risk assessment modeling for NOAF in patients undergoing invasive revascularization. Identifying the most effective risk stratification model for NOAF is clinically important, particularly given the wide range of clinical, laboratory, and electrocardiographic parameters available at admission, and the fact that NOAF management in AMI patients remains both controversial and not well understood. Despite the availability of several general AF risk scores, there remains a significant gap in tools specifically designed or validated to predict NOAF in the setting of AMI. Most existing models lack integration of angiographic complexity and contemporary biomarker data, which may limit their clinical applicability in this high-risk population. This study aimed to conduct a comparative validation of the GRACE 2.0 Global Registry of Acute Coronary Events), CHA_2DS_2-VASc (Congestive heart failure, Hypertension, Age ≥75 years, Diabetes mellitus, Stroke/TIA/thromboembolism, Vascular disease, Age 65–74 years, Sex category), C_2HEST (Coronary artery disease, Chronic obstructive pulmonary disease, Hypertension, Elderly \[age ≥75], Systolic heart failure, Thyroid disease), HAT_2CH_2 (Hypertension, Age >75, Stroke/TIA, Chronic obstructive pulmonary disease, Heart failure), SYNTAX (Synergy Between PCI with Taxus and Cardiac Surgery), and SYNTAX II risk scores in predicting the likelihood of NOAF during AMI in patients undergoing invasive treatment.

ABBREVIATIONS

ACC/AHA	American College of Cardiology/ American Heart Association
ACS	Acute coronary syndrome
AMI	Acute myocardial infarction
ARIC	Atherosclerosis Risk in Communities
ASA	Acetylsalicylic acid
AUC	Area under the curve
CABG	Coronary artery bypass grafting
CHARGE-AF	Cohorts for Heart and Aging Research in Genomic Epidemiology Atrial Fibrillation
COPD	Chronic obstructive pulmonary disease
COVID-19	Coronavirus Disease 2019
CULPRIT-SHOCK trial	Culprit Lesion Only PCI versus Multivessel PCI in Cardiogenic Shock
DAPA-HF	Dapagliflozin and Prevention of Adverse Outcomes in Heart Failure
DECLARE-TIMI 58	Dapagliflozin Effect on Cardiovascular Events -Thrombolysis in Myocardial Infarction 58
ECG	Electrocardiogram
ESC	European Society of Cardiology
FHS	Framingham Heart Study
ICCU	Intensive cardiac care unit
LASSO	Least Absolute Shrinkage and Selection Operator
LVEF	Left ventricular ejection fraction
MI	Myocardial infarction
MR	Mitral regurgitation
NOAF	New-onset atrial fibrillation
OAD	Oral antidiabetic drugs
PCI	Percutaneous coronary intervention
ROC	Receiver operating characteristic
SPAP	Systolic pulmonary artery pressure
SYNTAX	Synergy Between PCI with Taxus and Cardiac Surgery
TIA	Transient ischemic attack

Materials and Methods

This retrospective observational cohort study was conducted at two high-volume tertiary invasive cardiology centers in İzmir, Türkiye. All patients diagnosed with AMI and admitted between June 2021 (coinciding with the Turkish Ministry of Health's announcement to normalize lifestyle habits due to low Coronavirus Disease 2019 (COVID-19) case numbers) and January 2023 were included. The diagnosis of AMI was based on clinical evidence of myocardial injury, including necrosis and elevated cardiac troponin levels exceeding the 99th percentile reference limit. Atrial fibrillation was diagnosed by a physician based on electrocardiographic (ECG) findings, in accordance with

established guidelines.[5] All patients were either continuously monitored for at least 24 hours during their stay in the intensive cardiac care unit (ICCU), or received daily 12-lead ECGs (or ECGs in response to new symptoms) throughout their hospitalization in the cardiac ward. Patients were excluded if they met any of the following criteria:

- History of known AF;
- Diagnosis other than acute coronary syndrome (ACS);
- Development of atrial fibrillation while being followed-up in the ICCU for a non-ACS condition;
- Known congenital heart disease;
- Organic mitral regurgitation (MR) (defined as MR due to structural deformities or injury to the leaflets, chordae, and/or papillary muscles leading to incomplete leaflet closure during systole);
- Stenosis (e.g., rheumatic mitral valve disease);
- History of mitral valve endocarditis;
- History of cardiac surgery other than coronary artery bypass grafting (CABG);
- Active infection such as pneumonia;
- Death during catheterization or within 24 hours of ICCU admission;
- Requirement for inotropic support during catheterization or ICCU stay (Figure 1).

New-onset atrial fibrillation was defined as atrial fibrillation detected for the first time during the index hospitalization in patients without a previously documented history of AF, in accordance with the 2020 European Society of Cardiology (ESC) Guidelines for the management of atrial fibrillation.[5] Among the 64 patients diagnosed with NOAF during hospitalization, 18 had atrial fibrillation recorded at the time of admission. In these cases, NOAF was considered to be associated with the acute myocardial infarction, based on the absence of prior AF documentation in national electronic health records and patient charts. Additionally, outpatient ECGs and hospital discharge reports from the preceding 12 months were reviewed to confirm the absence of pre-existing atrial fibrillation. Only patients with clear documentation of sinus rhythm prior to admission—or without any prior AF-related findings—were classified as true new-onset cases. The diagnosis of NOAF was based on 12-lead electrocardiograms recorded at admission, during catheterization, or in the intensive cardiac care unit, provided the episode was long enough to be documented and confirmed by a cardiologist. Patients were continuously monitored for arrhythmias in the ICCU or underwent daily ECGs or symptom-triggered ECGs in the general ward. ST-segment deviation was defined as the presence of ≥ 1 mm (0.1 mV) of ST-segment elevation or depression, measured 60 ms after the J point, observed in at least two contiguous leads on the 12-lead ECG recorded at admission. This assessment was performed manually by two independent cardiologists who were blinded to clinical outcomes. This definition aligns with standard criteria recommended by the American College of Cardiology/American Heart Association

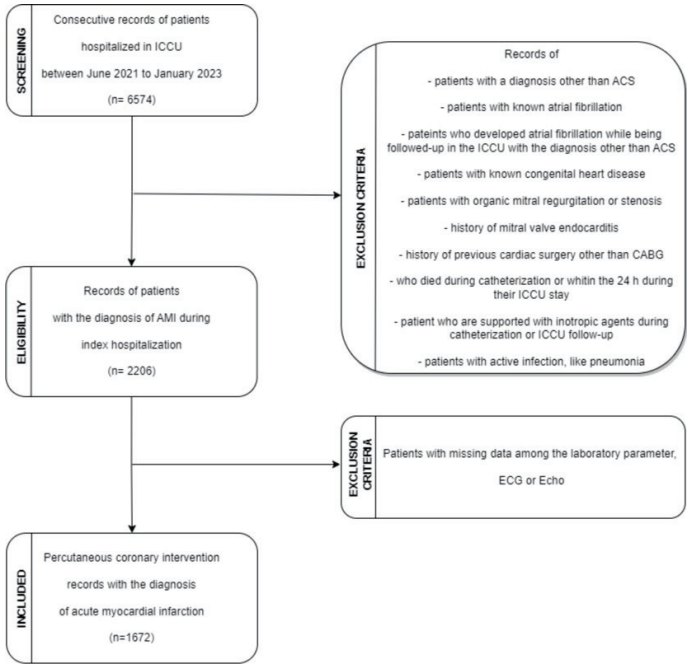

Figure 1. Patient identification flowchart.

(ACC/AHA) guidelines and is consistent with large-scale studies such as GUSTO-I (the Global Utilization of Streptokinase and Tissue Plasminogen Activator for Occluded Coronary Arteries I) and FAST-MI (the French Registry of Acute ST-Elevation or Non-ST-Elevation Myocardial Infarction) studies.[4-6] Baseline demographic, clinical, echocardiographic (performed during the index hospitalization), and angiographic data were collected and analyzed from medical records, procedural reports, and angiographic studies. Patients with ACS were categorized into two groups based on the presence of AF: the NOAF group and the non-NOAF group. Risk scores used in daily clinical practice for assessing in-hospital and long-term morbidity (CHA_2DS_2-VASC, C_2HEST, HAT_2CH_2, SYNTAX, SYNTAX II, and GRACE 2.0) were calculated using available data. The study was conducted in accordance with the principles of the Declaration of Helsinki. Informed consent was obtained from all patients. No artificial intelligence (AI)-assisted technologies, including large language models (LLMs), chatbots, or image generators, were used in the production of this manuscript.

The GRACE 2.0 ACS Risk Calculator (available via the MDCalc Medical Calculator app) was used to compute the GRACE risk score (GRACE RS). This score incorporates eight prognostic variables: ST-segment deviation, age, heart rate, systolic blood pressure, creatinine level, Killip classification, cardiac arrest at presentation, and elevated necrosis biomarkers.[6] Notably, in accordance with our inclusion criteria, ST-segment deviation and elevated troponin levels were considered present ("true") for all patients.

The SYNTAX score (SxS) and SYNTAX score II (SxSII) were calculated using the SYNTAX 2020 application. SxS is a widely recognized scoring algorithm used to assess the degree of complexity of coronary artery disease (CAD). It serves as a comprehensive angiographic grading tool that assists in objective

decision-making between CABG and percutaneous coronary intervention (PCI). The SxSII expands upon the original score by incorporating seven additional clinical variables to guide individualized treatment decisions based on mortality risk: age, creatinine clearance, left ventricular ejection fraction (LVEF), presence of unprotected left main coronary artery disease, peripheral vascular disease, female sex, and chronic obstructive pulmonary disease (COPD).[7]

The CHA_2DS_2-VASC score (which assigns 1 point for congestive heart failure, hypertension, age 65–74 years, diabetes mellitus, vascular disease, and female sex, and 2 points for age 75 and prior stroke or transient ischemic attack [TIA]) has a maximum total of 9 points. It is an effective tool for assessing ischemic stroke risk in patients with AF.[8] Moreover, several previous studies[9,10] have investigated its clinical utility in predicting the development of AF specifically.

For the purpose of predicting atrial fibrillation in the general population, the C_2HEST score, a composite of six parameters, was utilized in a large-scale study conducted in Asia. These parameters include: CAD or chronic obstructive pulmonary disease [1 point each], hypertension [1 point], elderly (age ≥ 75 years) [2 points], systolic heart failure [2 points], and thyroid disease (hyperthyroidism) [1 point].[11]

The HAT_2CH_2 score, developed in 2010, is based on patient age (≥ 75 years) [1 point], hypertension [1 point], stroke or transient ischemic attack [2 points], chronic obstructive pulmonary disease [1 point], and heart failure [2 points]. It was designed to help identify patients at risk of developing persistent AF.[12] In this study, we evaluated all of these risk scores, which are simple tools that can be calculated at the bedside using smartphones or paper charts, for their ability to predict NOAF in patients with AMI. The study was approved by Ethics Committee of Health Sciences University Tepecik Training and Research Hospital Non-Interventional Research Ethics Committee (Approval Number: 2022/04-41, Date: 15.04.2022).

Statistical Analysis

Continuous variables are presented as medians (25^{th}–75^{th} percentiles), while categorical variables are expressed as numbers (n) and percentages (%). Non-parametric tests were chosen for statistical analysis, as the Shapiro-Wilk test indicated that most parameters were not normally distributed, even after logarithmic transformation. The Mann-Whitney U test was used to compare continuous variables between groups. For categorical variables, comparisons were made using Pearson's chi-square test, Yates's chi-square test, or Fisher's exact test. To assess the direction and strength of correlations between non-normally variables, Spearman's correlation coefficients were calculated. The area under the receiver operating characteristic (ROC) curve (AUC) was used to evaluate the predictive performance of the established variables for identifying NOAF. Optimal cut-off values were selected based on the best combination of sensitivity and specificity. To identify variables most strongly associated with NOAF, univariate logistic regression analyses were performed using pre-specified cut-off values. Continuous variables were categorized according to ROC-derived thresholds, while categorical variables significantly associated with NOAF (p-value of 0.05 or less) univariate analysis were included in the multivariate analysis. Variables included in the final model were determined using a backward elimination approach, starting with all statistically significant predictors. Given the heterogeneity in group sizes between NOAF and non-NOAF patients, variable selection was also performed using the Least Absolute Shrinkage and Selection Operator (LASSO) regression to mitigate potential bias and prevent model due to class imbalance. Since the dependent variable in the study is binary, variable selection was based on logistic regression using the LASSO method. Analyses were conducted in R (version 4.4.2) using the "glmnet" package. The LASSO model was optimized for the penalty parameter (lambda) through 5-fold cross-validation, and model fit was evaluated based on the deviance criterion. The optimal model was selected using the "lambda.min" parameter, which corresponds to the lambda value yielding the minimum cross-validated deviance. The regression coefficients from this model were then examined, and variables with non-zero coefficients were considered statistically relevant. All analyses were conducted using IBM SPSS Statistics version 21.0 (IBM Corp., 2012, Armonk, NY), with $P < 0.05$ set as the threshold for statistical significance. A post hoc power analysis was performed based on the difference in hemoglobin A1c (HbA1c) levels between the NOAF (+) and NOAF (–) groups. Assuming a medium effect size (Cohen's d = 0.5), with group sizes of 1,608 and 64, and an alpha level of 0.05, the calculated statistical power was 0.975. This indicates that the sample size was sufficient to detect clinically meaningful differences in HbA1c between the groups.

Results

A total of 2,206 AMI patients were enrolled in the study. Of these, 64 (2.9%) were diagnosed with NOAF during hospitalization. Among the 64 NOAF patients, 18 (28.1%) had arrhythmia at the time of admission, 20 (31.3%) during catheterization in the lab, and 26 (40.6%) during their stay in the ICCU. Due to missing data, variables from 1,672 patents were evaluated in the study.

Regarding patient demographics and comorbidities, those who developed NOAF were significantly older, with a mean age of 63.64 ± 12.93 years, compared to 52.94 ± 10.15 years in the non-NOAF group ($P < 0.001$). The NOAF group also had a higher prevalence of hypertension (53.13% vs. 27.80%, $P < 0.001$), prior coronary artery disease (37.5% vs. 21.95%, $P = 0.013$), and heart failure (23.4% vs. 13.06%, $P = 0.025$), as detailed in Table 1.

The risk of NOAF was significantly higher in patients who were taking acetylsalicylic acid prior to admission, with 33.33% of NOAF patients using aspirin compared to 16.67% in the non-NOAF group (odds ratio [OR]: 2.50, $P = 0.001$). Similarly, statin use before admission was more frequent among NOAF patients (18.8% vs. 5.53%, OR: 4.34, $P < 0.001$). Additionally, the absence of a smoking history was more common in the NOAF group, with 33.33% being non-smokers and 14.04% ex-smokers, compared to 11.73% and 5.86%, respectively, in the non-NOAF group ($P < 0.001$), as shown in Table 1.

The presence of diabetes mellitus did not significantly affect the development of NOAF during AMI (40.6% vs. 33.0%, $P = 0.258$). However, patients who developed NOAF had significantly higher blood glucose levels at admission (181.83 ± 85.23 mg/

Table 1. Baseline clinical characteristics

	Non-NOAF (-) (n = 1608)	NOAF (+) (n = 64)	P
BMI (kg/m²)	26.74 ± 3.74 27.12 (23.74–28.41)	27.43 ± 3.98 27.04 (24.29–29.41)	0.212[MW]
Age (years)	52.94 ± 10.15 51.00 (47.00–59.00)	63.64 ± 12.93 64.50 (56.00–73.00)	< 0.001[MW]
Gender			0.351***
Male, n (%)	1295 (80.53%)	48 (75.00%)	
Female, n (%)	313 (19.47%)	16 (25.00%)	
Previous stroke/TIA, n (%)	90 (5.60%)	5 (7.81%)	0.634****
Hypertension, n (%)	447 (27.80%)	34 (53.13%)	<0.001***
Prior coronary artery disease (CABG/PCI), n (%)	353 (21.95%)	24 (37.5%)	0.013*
Peripheral artery disease (carotid/peripheral arteries), n (%)	90 (5.60%)	1 (1.56%)	0.265****
Heart failure, n (%)	210 (13.06%)	15 (23.4%)	0.025***
Chronic renal disease, n (%)	122 (7.59%)	9 (14.06%)	0.098***
Smoking status			< 0.001**
Current smoker, n (%)	1251 (82.41%)	30 (52.63%)	
Ex-smoker, n (%)	89 (5.86%)	8 (14.04%)	
Non-smoker, n (%)	178 (11.73%)	19 (33.33%)	
Thyroid disease			0.543**
Hypothyroidism	85 (5.3%)	2 (3.1%)	
Hyperthyroidism	21 (1.3%)	0 (0.0%)	
None	1502 (93.4%)	62 (96.9%)	
Chronic obstructive pulmonary disease, n (%)	123 (7.65%)	8 (12.90%)	0.204****
On-admission treatment			
Acetylsalicylic acid, n (%)	268 (16.67%)	21 (33.33%)	0.001****
B-blocker, n (%)	179 (11.13%)	12 (18.75%)	0.056****
ACE-I/ARB, n (%)	536 (33.33%)	24 (37.5%)	0.302***
Statin, n (%)	89 (5.53%)	12 (18.8%)	< 0.001****

* Pearson Chi-Square; ** Pearson Exact Chi-Square; *** Yates's Chi-Square; **** Fisher's Exact Test; MW: Mann-Whitney U Test. ACE-I, Angiotensin-converting enzyme inhibitor; ARB, Angiotensin receptor blocker; BMI, Body max index; CABG, Coronary artery bypass grafting; PCI, Percutaneous coronary intervention; TIA, Transient ischemic attack.

dL vs. 131.89 ± 52.53 mg/dL, P < 0.001) and higher HbA1c levels (6.98 ± 1.81% vs. 6.16 ± 1.19%, P = 0.007), indicating poorer glycemic control. In terms of lipid and renal parameters, NOAF patients had lower high-density lipoprotein (HDL) levels (39.4 ± 7.34 mg/dL vs. 44.6 ± 7.36 mg/dL, P < 0.001), lower creatinine clearance (75.1 ± 30.6 mL/min vs. 99.7 ± 32.84 mL/min, P < 0.001), and lower total cholesterol levels (186.0 ± 43.38 mg/dL vs. 223.4 ± 49.76 mg/dL, P < 0.001), as summarized in Table 2. Detailed clinical data at admission are presented in Table 2. Patients who developed NOAF had a significantly higher heart rate on admission (93.2 ± 28.8 bpm vs. 82.44 ± 11.34 bpm, P = 0.007) and lower systolic blood pressure (122.6 ± 21.4 mmHg vs. 131.41 ± 21.88 mmHg, P = 0.016) compared to those without NOAF. However, the presence of cardiac arrest on admission was not significantly different between the groups (6.3% in NOAF vs. 14.7% in non-NOAF, P = 0.088). Similarly, Killip class distribution showed no significant difference (Killip class I: 81.3% vs. 83.5%; class II: 17.2% vs. 15.0%; class III: 1.6% in both groups; P = 0.890).

Electrocardiographic, echocardiographic, and angiographic findings are summarized in Table 3. Among the echocardiographic parameters, patients who developed NOAF had a significantly larger left atrial diameter (38.7 ± 4.84 mm vs. 36.32 ± 2.39 mm, P < 0.001), increased left ventricular end-systolic diameter (LVESD) (35.18 ± 7.96 mm vs. 30.51 ± 3.79 mm, P < 0.001), and increased left ventricular end-diastolic diameter (LVEDD) (49.33 ± 6.03 mm vs. 45.39 ± 2.87 mm, P < 0.001). Systolic pulmonary artery pressure (SPAP) was also significantly higher in the NOAF group (37.9 ± 10.2 mmHg vs. 30.0 ± 2.12 mmHg, P < 0.001). Mitral regurgitation, even when mild, was more common among NOAF patients, with 53.1% having any degree of MR compared to 14.8% in the non-NOAF group (P < 0.001). Additionally, patients with NOAF had lower left ventricular ejection fraction (43.11 ± 10.2% vs. 48.43 ± 6.55%, P < 0.001).

The effects of the variables included in the study on NOAF development were assessed using univariate and multivariate analyses. Stepwise logistic regression was performed with variables found to be significant in univariate logistic regression.

Table 2. Laboratory parameters and clinical data at admission

	Non-NOAF (-) (n = 1608)	NOAF (+) (n = 64)	P
Diagnosis			0.206**
NSTEMI	537 (33.40%)	16 (25.0%)	
STEMI	1071 (66.60%)	48 (75.0%)	
Laboratory parameters at admission			
Blood glucose (mg/dL)	131.89 ± 52.53 120.50 (106.00-136.00)	181.83 ± 85.23 154.00 (127.00-226.00)	< 0.001^{MW}
CrC (mL/min)	99.69 ± 32.84 102.50 (65.38-118.94)	75.1 ± 30.6 74.0 (50.0-92.0)	< 0.001^{MW}
TSH (mIU/L)	1.30 ± 0.44 1.19 (0.91-1.50)	1.64 ± 1.35 1.10 (0.71-2.26)	0.922^{MW}
HB (g/dL)	13.98 ± 1.18 14.00 (13.00-14.70)	15.75 ± 17.93 13.60 (11.73-15.35)	0.173^{MW}
HDL (mg/dL)	44.60 ± 7.36 43.00 (40.00-51.00)	39.4 ± 7.34 39.50 (34.30-43.00)	< 0.001^{MW}
Total cholesterol (mg/dL)	223.44 ± 49.76 212.00 (187.00-243.00)	186.00 ± 43.38 183.50 (162.50-217.25)	< 0.001^{MW}
HbA1c (%)	6.16 ± 1.19 6.00 (5.40-6.60)	6.98 ± 1.81 6.20 (5.70-8.10)	0.007^{MW}
Clinical status at admission			
Heart rate (bpm)	82.44 ± 11.34 80.0 (77.0-90.0)	93.2 ± 28.8 85.0 (78.0-120)	0.007^{MW}
Systolic blood pressure (mmHg)	131.41 ± 21.88 130.00 (115.00-150.00)	122.6 ± 21.4 120.00 (107.00-135.00)	0.016^{MW}
Modified killip class			0.890*
Class I	1342 (83.5%)	52 (81.3%)	
Class II	241 (15%)	11 (17.2%)	
Class III	25 (1.6%)	1 (1.6%)	
Cardiac arrest			0.088**
Yes	236 (14.68%)	4 (6.3%)	
No	1372 (85.32%)	60 (93.8%)	

* Pearson Exact Chi-Square; ** Yates's Chi-Square; MW: Mann-Whitney U Test. CrC, Creatinine clearance; bpm, Beats per minute; HB, Hemoglobin; HDL, High-density lipoprotein; NSTEMI, Non-ST-elevation myocardial infarction; STEMI, ST-elevation myocardial infarction; TSH, Thyroid-stimulating hormone.

Additionally, to address the class imbalance between NOAF and non-NOAF groups and to prevent model overfitting, LASSO regression was used for variable selection. After including all candidate variables in the model, a LASSO regression analysis was performed. Variables with coefficients shrunk to zero by the penalty parameter (lambda) were excluded from the final model, and only those with non-zero coefficients were retained. The variables that remained in the model following LASSO penalization, indicating their relative contribution to the model, are listed below: Age (coefficient: 0.27005); time from symptom onset to reperfusion (4–12 hours) (0.19003); > 12 hours (0.27992); blood glucose level (0.1800); HbA1C (0.349906); systolic blood pressure (-0.220029); number of ischemic ST-segment elevation leads (0.3099); left ventricular end-diastolic diameter (0.21009); and presence of mitral regurgitation (reference: none) (0.28997). These variables, identified by having non-zero coefficients, were considered the most informative predictors retained in the final LASSO-selected model. The major predictors of NOAF were determined accordingly, as shown in Table 4. In the final multivariate logistic regression analysis, several variables were identified as independent predictors of NOAF during acute myocardial infarction: Age ≥ 60 years was associated with a significantly increased risk (OR: 2.103; 95% confidence interval [CI]: 1.378–3.134; P < 0.001). Compared to patients who received reperfusion within 4 hours, those treated between 4–12 hours had a moderately increased risk (OR: 1.912; 95% CI: 1.134–3.221; P = 0.015), while those treated after 12 hours had a substantially higher risk (OR: 2.708; 95% CI: 1.619–4.382; P < 0.001). Elevated blood glucose levels (≥ 127 mg/dL; OR: 1.593; 95% CI: 1.087–2.408; P = 0.015) and high HbA1c levels (≥5.6%; OR: 2.841; 95% CI: 1.923–4.195; P < 0.001) were also significant predictors of NOAF. Low systolic blood pressure (≤125 mmHg) was found to be a predictive factor (OR: 0.693; 95% CI: 0.509–0.944; P = 0.028). Additional independent risk factors included a higher number of ischemic ST-segment derivations (≥ 5 leads) (OR: 2.482; 95% CI: 1.517–4.062; P < 0.001), increased left ventricular end-diastolic diameter (≥ 48 mm) (OR: 2.011; 95% CI: 1.211–3.197; P = 0.006), and the presence of any grade of mitral regurgitation (OR: 1.864; 95% CI: 1.090–3.126; P = 0.012).

Table 3. Electrocardiographic, echocardiographic, and coronary angiographic findings during hospitalization

	Non-NOAF (-) (n = 1608)	NOAF (+) (n = 64)	P
Electrocardiographic parameters at admission			
Ischemic ST derivation lead number	4.17 ± 2.36 4.00 (2.00-6.00)	4.91 ± 2.25 6.00 (3.00-6.00)	0.012[MW]
Echocardiographic parameters			
Left atrial size (mm)	36.32 ± 2.39 36.0 (35.0-37.0)	38.7 ± 4.84 38.0 (36.0-42.0)	< 0.001[MW]
LVEF (%)	48.43 ± 6.55 50.0 (41.25-50.0)	43.11 ± 10.2 45.0 (39.0-50.0)	< 0.001[MW]
LVESD (mm)	30.51 ± 3.79 31.0 (28.0-33.0)	35.18 ± 7.96 35.0 (30.0-40.0)	< 0.001[MW]
LVEDD (mm)	45.39 ± 2.87 45.0 (44.0-47.0)	49.33 ± 6.03 48.0 (46.0-53.0)	< 0.001[MW]
Mitral regurgitation			< 0.001*
None	1332 (82.8%)	26 (40.6%)	
Mild	238 (14.8%)	34 (53.1%)	
Moderate	38 (2.4%)	4 (6.3%)	
SPAP (mmHg)	30.00 ± 2.12 30.0 (29.0-32.50)	37.9 ± 10.2 36.0 (35.0-42.3)	< 0.001[MW]
Coronary angiographic parameters			
SYNTAX score	11.47 ± 6.53 8.00 (8.00-14.5)	21.25 ± 10.1 22.0 (14.0-26.0)	< 0.001[MW]
No-reflow phenomenon	43 (3.0%)	3 (4.7%)	0.692**
Time from symptom onset to reperfusion			< 0.001**
<4 hours	1430 (88.93%)	31 (48.4%)	
4–12 hours	178 (11.07%)	14 (21.9%)	
>12 hours	0 (0.0%)	19 (29.7%)	

* Pearson Chi-Square; ** Fisher's Exact Chi-Square; MW: Mann-Whitney U Test. LVEDD, Left ventricular end-diastolic diameter; LVEF, Left ventricular ejection fraction; LVESD, Left ventricular end-systolic diameter; SPAP, Systolic pulmonary arterial pressure.

In this study, we found that each risk score can be used to predict NOAF complicating AMI (Table 5). Patients with the following values had an increased risk of developing NOAF during AMI: SxS RS ≥ 16.1, SxSII RS ≥ 26.2, GRACE 2.0 ≥ 122, CHA_2DS_2-VASC RS ≥ 3, C_2HEST RS ≥ 3, or HAT_2CH_2 RS ≥ 1. ROC analysis showed that the AUC of the SxS for predicting NOAF in the setting of AMI was 0.785 (95% CI: 0.767–0.802, P < 0.001), followed by SxSII (AUC: 0.747; 95% CI: 0.728–0.765, P < 0.001), and the GRACE 2.0 risk score (AUC: 0.740; 95% CI: 0.721–0.758, P < 0.001) (Table 6). Based on these results, SxS was identified as the most predictive RS for NOAF complicating AMI (Figure 2).

A new scoring model was developed by combining HbA1c level, identified as the most predictive risk factor for NOAF, with each of the risk scores included in the study. This combined approach was found to be superior in predicting NOAF in the context of AMI. ROC analysis demonstrated the following AUC values for the new scoring models in predicting NOAF in the context of AMI: 0.794 (95% CI: 0.764–0.808, P < 0.001) for SxS, 0.790 (95% CI: 0.734–0.812, P < 0.001) for SxSII, 0.784 (95% CI: 0.750–0.795, P < 0.001) for the GRACE 2.0 risk score, 0.705 (95% CI: 0.657–0.707, P < 0.001) for the CHA_2DS_2-VASc score, 0.673 (95% CI: 0.674–0.723, P < 0.001) for the C_2HEST score, and 0.650 (95% CI: 0.627–0.678, P < 0.001) for the HAT_2CH_2 score.

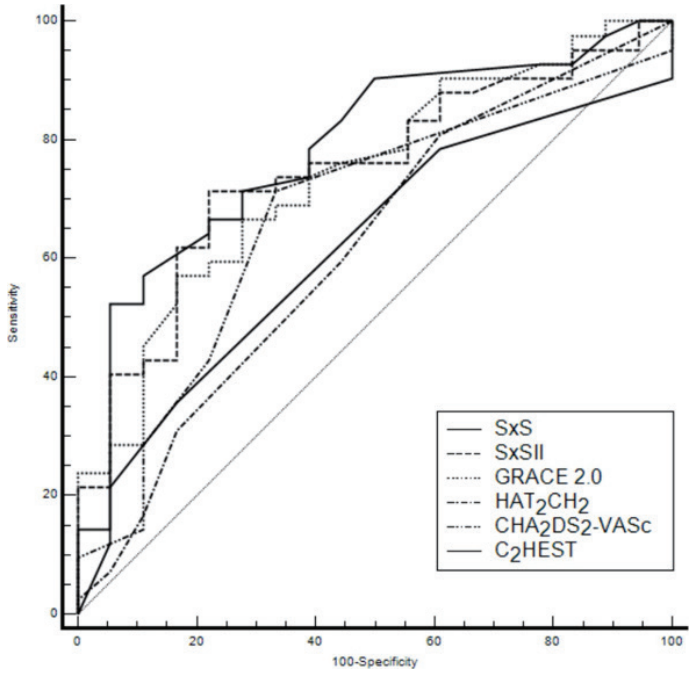

Figure 2. Receiver operating characteristic (ROC) curve analysis of risk scores for predicting new-onset atrial fibrillation (NOAF) during acute myocardial infarction (AMI).

Table 4. Univariate and multivariable logistic regression analyses of independent predictors of new-onset atrial fibrillation (NOAF)

Variable	Univariate analysis		Multivariable analysis	
	OR (95% CI)	P	OR (95% CI)	P
Age	1.09 (1.06–1.11)	< 0.001	**2.103 (1.378–3.134)**	**< 0.001**
HT (Ref: None)	2.946 (1.787–4.858)	< 0.001		
CAD (Ref: None)	3.000 (1.786–5.039)	< 0.001		
HF (Ref: None)	5.204 (2.835–9.550)	< 0.001		
Time from symptom onset to reperfusion (Ref: <4 hours)	4–12 hours: > 3.310 (1.666–6.5742) >12 hours: > Inf.	< 0.001 0.972	**4–12 hours: >1.912 (1.134–3.221)** **>12 hours: >2.708 (1.619–4.382)**	**0.015** **< 0.001**
Blood glucose level	1.009 (1.006–1.012)	< 0.001	**1.593 (1.087–2.408)**	**0.015**
Creatinine clearance	0.979 (0.971–0.986)	< 0.001		
HDL	0.892 (0.848–0.937)	< 0.001		
Total cholesterol	0.976 (0.966–0.986)	< 0.001		
HbA1c	1.484 (1.220–1.807)	< 0.001	**2.841 (1.923–4.195)**	**< 0.001**
Heart rate	1.050 (1.040–1.071)	< 0.001		
Systolic blood pressure	0.980 (0.965–0.994)	0.005	**0.693 (0.509–0.944)**	**0.028**
Ischemic ST derivation lead count	1.140 (1.026–1.267)	0.015	**2.482 (1.517–4.062)**	**< 0.001**
Left atrial size	1.360 (1.240–1.480)	< 0.001		
LVEF	0.885 (0.850–0.921)	< 0.001		
LVESD	1.300 (1.210–1.390)	< 0.001		
LVEDD	1.440 (1.320–1.580)	< 0.001	**2.011 (1.211–3.197)**	**0.006**
Mitral regurgitation (Ref: None)	9.692 (5.748–16.341)	< 0.001	**1.864 (1.091–3.126)**	**0.012**
SPAP	1.580 (1.250–2.00)	< 0.001		
Acetylsalicylic acid use	2.500 (1.462–4.272)	< 0.001		
Statin use (Ref: None)	4.340 (2.242–8.400)	< 0.001		

CAD, Coronary artery disease; HDL, High-density lipoprotein; HF, Heart failure; HT, Hypertension; LVEDD, Left ventricular end-diastolic diameter; LVEF, Left ventricular ejection fraction; LVESD, Left ventricular end-systolic diameter; SPAP, Systolic pulmonary artery pressure.

Table 5. Risk scores

	Non-NOAF (-) (n = 1608)	NOAF (+) (n = 64)	P
SYNTAX score	11.48 ± 6.53 8.00 (8.00-14.50)	21.25 ± 10.1 22.0 (14.0-26.0)	< 0.001^{MW}
CHA_2DS_2-VASc	2.44 ± 1.57 2.00 (1.00-3.00)	3.41 ± 1.76 3.00 (2.00-4.25)	< 0.001^{MW}
C_2HEST	1.89 ± 0.99 2.00 (1.00-2.00)	2.38 ± 1.36 2.00 (2.00-3.00)	0.002^{MW}
HAT_2CH_2	1.39 ± 1.46 1.00 (0.00-2.00)	2.02 ± 1.49 2.00 (1.00-3.00)	< 0.001^{MW}
SYNTAX II score	22.59 ± 9.36 22.80 (14.20-26.10)	35.42 ± 13.27 34.0 (26.2-46.0)	< 0.001^{MW}
GRACE 2.0	102.44 ± 23.54 102.00 (85.00-120.00)	129.78 ± 26.01 134 (110-146)	< 0.001^{MW}

MW, Mann-Whitney U Test.

Discussion

Atrial fibrillation, the most common clinical arrhythmia, frequently occurs as a complication of AMI and serves as an independent predictor of adverse outcomes.[3,13,14] In this study, the SYNTAX score demonstrated the best diagnostic performance for predicting NOAF in the context of AMI, followed by the GRACE 2.0 RS and SYNTAX II scores. These three scores outperformed CHA_2D_2-VASc, C_2HEST, and HAT_2CH_2 scores. Notably, the predictive performance of the SYNTAX score was further enhanced by incorporating HbA1c levels, resulting in a modified model that combines both anatomical and metabolic risk factors. This combined SYNTAX

Table 6. Pairwise comparison of receiver operating characteristic (ROC) curves

	SYNTAX	C_2HEST	CHA_2DS_2-VASc	HAT_2CH_2	GRACE 2.0	SYNTAX II
SYNTAX score	AUC: 0.785	–	–	–	–	–
C_2HEST	P < 0.001	AUC: 0.618	–	–	–	–
CHA_2DS_2-VASc	P = 0.018	P = 0.021	AUC: 0.672	–	–	–
HAT_2CH_2	P < 0.001	P = 0.976	P = 0.016	AUC: 0.617	–	–
GRACE 2.0	P = 0.262	P = 0.001	P = 0.045	P < 0.001	AUC: 0.740	–
SYNTAX II score	P = 0.317	P < 0.001	P = 0.018	P < 0.001	P = 0.810	AUC: 0.747

+ HbA1c model achieved a higher AUC (0.794) compared to the SYNTAX score alone (0.785), suggesting improved discrimination for NOAF prediction. This improvement can be attributed to the complementary nature of the included parameters: while the SYNTAX score reflects the complexity of coronary artery disease, HbA1c represents chronic metabolic stress, which contributes to atrial structural remodeling and arrhythmogenesis. Therefore, integrating these parameters may offer a more holistic risk stratification tool in the setting of acute MI. Additionally, several clinical and echocardiographic parameters identified in our study as significant predictors of NOAF during AMI have also been reported in previous research. For instance, left atrial enlargement and increased left ventricular end-diastolic diameter have consistently been associated with a higher risk of NOAF, likely due to elevated atrial pressure and stretch.[15] Similarly, mitral regurgitation, even when mild, has been found to contribute to NOAF development by increasing left atrial volume and promoting electrical remodeling.[16] In addition, prolonged time from symptom onset to reperfusion (> 4 hours), which was significant in our analysis, has been shown in earlier reports to increase ischemic burden and sympathetic activation, both of which predispose patients to atrial fibrillation.[17] ST-segment deviation on admission electrocardiogram (ECG), another predictor in our model, is also supported by prior research as a marker of widespread ischemia and atrial irritability.[18] These consistent findings across studies support the robustness of our model and highlight the multifactorial nature of NOAF during AMI. In our study, the CHA_2DS_2-VASc, C_2HEST, and HAT_2CH_2 scores demonstrated relatively poor predictive performance for NOAF in the setting of AMI. These scores were originally developed for general AF risk assessment in broader outpatient or community-based populations, not for acute ischemic settings. One possible reason for their limited utility is that they do not incorporate acute-phase variables such as infarct size, ischemic burden, or angiographic complexity, all of which may play a significant role in NOAF development during AMI. Additionally, these scores lack integration of acute metabolic and hemodynamic parameters (e.g., blood glucose, HbA1c, troponin, or ST-segment changes), which have been shown to influence arrhythmogenesis in acute coronary syndromes. Therefore, the application of these scores in this high-risk inpatient population may not reflect the true burden of NOAF risk.

In our study, the use of acetylsalicylic acid (ASA) and statins was more common among patients who developed NOAF. Interestingly, regression analysis revealed that ASA and statin users had a 2.5-fold (P = 0.001) and 4.34-fold (P < 0.001) increased risk of NOAF, respectively (Table 1). However, this finding is likely influenced by confounding factors, as these medications are more frequently prescribed to patients with a higher burden of coronary artery disease and comorbidities. Therefore, this observed association should be interpreted with caution and not assumed to be causal.

When HbA1c, the main independent predictor identified in our cohort, was added to the risk scores as an additional parameter, the predictive accuracy for NOAF during AMI improved noticeably. Atrial fibrillation complicating AMI has been reported to have a wide incidence range, from 2.3% and 21%.[1,19,20] In our study, the incidence of NOAF was 2.92%, which is consistent with previous reports, particularly those focusing on NOAF occurring during hospitalization in the modern revascularization era.[1,21,22]

Various studies have explored the predictors of NOAF in the context of AMI, identifying numerous significant and independent factors.[4,23] Various studies have demonstrated that the onset of NOAF during ACS involves multiple mechanisms. Although the precise cause remains uncertain, one potential mechanism is inflammation, a shared feature in both NOAF and CAD. The effects of inflammation on coronary arteries depends on multiple factors, one of which is elevated blood glucose levels due to uncontrolled or undiagnosed diabetes. Interestingly, a history of diabetes was not a predictor of clinical outcomes in the current study. Regardless of diabetes status, a high blood glucose level or elevated HbA1c on admission was associated with an increased risk of NOAF complicating AMI in our cohort. The literature reports varying HbA1c cut-off values associated with atherosclerosis, demonstrating increased CAD risk even among non-diabetic individuals.[24] According to the American Diabetes Association, the prediabetic range is defined as an HbA1c of 5.7–6.4.[25] In our study, an HbA1c threshold of ≥ 5.6 emerged as the most influential independent predictor of NOAF among AMI patients. This finding may be explained by the hypothesis that diabetes-related end-organ damage—reflected by elevated HbA1c levels in patients who developed NOAF and detected via coronary anatomy and calcification scores—provides a more accurate measure of risk in this population than metabolic markers alone. Several mechanisms may underlie the relationship between elevated HbA1c and NOAF development in the setting of AMI. Chronic hyperglycemia contributes to left atrial structural remodeling through increased oxidative stress, inflammation, and interstitial fibrosis, all of which can alter atrial electrophysiology and promote arrhythmogenesis. Moreover, elevated HbA1c levels are indicative of poor glycemic control, insulin resistance,

and metabolic dysregulation, all of which are independently associated with atrial fibrillation in both diabetic and non-diabetic populations. These pathophysiological changes may explain why HbA1c emerged as the strongest independent predictor of NOAF in our study. Although the exact number of patients receiving sodium-glucose cotransporter 2 (SGLT2) inhibitor therapy in our cohort is unknown—due to the classification of antidiabetic treatment into broader categories (i.e., oral antidiabetic drugs [OAD], insulin, or a combination of both)—emerging evidence suggests that these agents may reduce the incidence of atrial fibrillation. This benefit is believed to occur through mechanisms such as favorable cardiac remodeling, reduction of oxidative stress, and improvement in metabolic profiles. Several large trials and meta-analyses (e.g., DECLARE-TIMI 58 [Dapagliflozin Effect on Cardiovascular Events-Thrombolysis in Myocardial Infarction 58], DAPA-HF [Dapagliflozin and Prevention of Adverse Outcomes in Heart Failure]) have reported a reduced risk of AF in patients treated with SGLT2 inhibitors. While our dataset does not allow for a direct evaluation of this association, the strong link between elevated HbA1c and NOAF supports the hypothesis that targeting glycemic control—potentially through SGLT2 inhibitors—could serve as a preventive strategy against NOAF in the post-MI setting. Future studies are warranted to explore this potential therapeutic benefit. An angiographic tool for assessing CAD complexity, the SYNTAX score is commonly used in clinical practice.[26] Drawing on data from the SYNTAX trial, SxS was originally developed to predict clinical outcomes in stable patients presenting with three-vessel and/or left main disease who underwent PCI or CABG.[27,28] Subsequently, the SxS was applied across a broader range of patient populations with various clinical scenarios, including those presenting with ACS and undergoing primary PCI.[29,30] Patients with a higher SYNTAX score are known to have more jeopardized myocardium under ischemia, and this was reflected in our study, where widespread ST-segment deviation on admission ECG emerged as an independent predictor of NOAF. To complement angiographic data with clinical variables, the SYNTAX score II was developed. In our study, SxSII was found to be as helpful as the SxS in predicting NOAF complicating AMI, but not superior.

Although low HDL-cholesterol and statin use have been previously associated with atrial fibrillation in various studies, these variables did not remain independent predictors of NOAF in our multivariate model.[31] Nevertheless, their established roles in modulating systemic inflammation and atherosclerotic burden may still contribute indirectly to arrhythmic risk, particularly in patients with chronic dyslipidemia.[32]

Among the echocardiographic parameters evaluated, only mitral regurgitation was identified as an independent predictor of NOAF in our model (OR: 1.864; 95% CI: 1.090–3.126; P = 0.012). While variables such as left ventricular end-systolic diameter, left atrial diameter, and systolic pulmonary artery pressure (SPAP) have been associated with atrial pressure and structural remodeling in previous studies, they did not retain statistical significance in our multivariate analysis.[33,34] This suggests that volume overload, as reflected by MR, may play a more dominant role in the development of atrial fibrillation during AMI in this patient cohort.

Other independent predictors of NOAF in the setting of AMI included age, heart rate, systolic blood pressure, and creatinine level, all of which are among the eight prognostic variables included in the GRACE 2.0 RS. Initially developed from the GRACE registry, the GRACE risk score (2.0) was later validated in the French FAST-MI 2005 registry for both acute ST-elevation and non-ST-elevation MI.[6] This updated risk assessment model is important for its simplicity and compatibility with handheld electronic devices and smartphones. It predicts mortality at 6 months, 1 year, and 3 years in patients with ACS. Notably, the occurrence of NOAF during AMI has consistently been associated with worse clinical outcomes, including higher rates of in-hospital mortality, ischemic stroke, and long-term mortality.[4,35,36] Therefore, it is not surprising that the GRACE 2.0 RS proved valuable in predicting NOAF in patients with AMI. In our multivariate model, two key components of the GRACE score—age and systolic blood pressure—were independently associated with the development of NOAF, further supporting the relevance of this risk score in this clinical context. According to current guidelines, moderate- or high-risk GRACE scores in ACS patients are associated with worse clinical outcomes,[6] which may also reflect a higher risk for NOAF during AMI. In our study, a GRACE (2.0) risk score above 122 defined this high-risk subgroup. The GRACE 2.0 score, which is calculated using clinical data independent of coronary angiographic findings, was shown to be nearly as effective as the SxSII score in predicting NOAF complicating AMI, supporting its practical utility.

Etiologies of AF during AMI, aside from inflammation, include excessive sympathetic stimulation, pressure overload of the left or right ventricle, and hypoxia.[1,2,37] All of these factors are commonly seen in patients with heart failure. Elevated heart rates and reduced systolic blood pressure likely indicate hemodynamic compromise, a relationship further supported by their association with heart failure and markers of more extensive MI, such as a lower ejection fraction.[38,39] A sub-analysis of the CULPRIT-SHOCK trial (Culprit Lesion Only PCI versus Multivessel PCI in Cardiogenic Shock) found that 52 of 142 patients (37%) with cardiogenic shock complicating AMI developed new-onset AF during their initial hospital stay.[40] However, in our study, the two GRACE RS components (cardiac arrest at admission and Killip class (signs/symptoms)) did not affect the occurrence of NOAF during AMI, likely due to non-homogeneous sample sizes in these subgroups. However, this finding is not clinically significant and represents one of the limitations of the study.

Although the CHA_2DS_2-VASc score is widely used to assess ischemic stroke risk in patients with AF,[41] its role in predicting the onset of AF has been evaluated in several studies.[9,42] In an ACS cohort, Mitchell et al.[43] demonstrated that neither the $CHADS_2$ nor CHA_2DS_2-VASc scores were effective in predicting incident AF. Similarly, in our study, even after modifying the CHA_2DS_2-VASc score by incorporating HbA1c levels, its diagnostic performance remained relatively poor, with C-statistics of 0.705 and 0.672, respectively. The C_2HEST score, which has been widely studied in Asian populations, has shown superior predictive performance for incident AF compared to the $CHADS_2$, CHA_2DS_2-VASc, and HATCH scores in the general population.[11]

Additionally, studies have explored the use of the HAT_2CH_2 score to predict AF in various patient populations, such as those with cancer[44] or patients presenting to the emergency department.[45] Despite the poor predictive performance of the CHA_2DS_2-VASc score, both the HAT_2CH_2 and C_2HEST scores performed even worse in our study. This discrepancy may be explained by the low prevalence of COPD—a key component of both the HAT_2CH_2 and C_2HEST scores—within our study population. Furthermore, the study's primary outcome may be influenced by the fact that the research sample consisted exclusively of AMI patients, a clinical setting in which risk scores such as SYNTAX, GRACE 2.0, and SYNTAX II are more likely to provide predictive value in assessing disease severity.

Limitations

The relatively small sample size of patients with NOAF may limit the strength of independent predictors identified through multivariate analysis, potentially affecting the comprehensiveness of our conclusions. It is also possible that some asymptomatic paroxysmal AF cases in the non-NOAF group went undetected due to minimal diagnostic monitoring in the cardiology department—where only one daily 12-lead ECG was performed. Additionally, individuals with asymptomatic AF prior to the index AMI may have been misclassified as NOAF, despite our exclusion of patients with documented AF. Although the study included patients with AMI, the majority were ST-elevation myocardial infarction (STEMI) cases, as both participating centers functioned as primary PCI hubs for İzmir Province.

Conclusion

In this study, we demonstrated that the SYNTAX RS has clinically relevant superiority over other risk scores in predicting NOAF among patients with AMI. Additionally, HbA1c emerged as an important biomarker for NOAF, independent of the patient's diabetes status. A modified SxS created by adding HbA1C to the original SxS, was shown to have better predictive value for NOAF in the setting of AMI.

Ethics Committee Approval: Ethics committee approval was obtained from Ethics Committee of Health Sciences University Tepecik Training and Research Hospital Non-Interventional Research Ethics Committee (Approval Number: 2022/04-41, Date: 15.04.2022).

Informed Consent: Informed consent was obtained from all patients.

Conflict of Interest: The authors have no conflicts of interest to declare.

Funding: The authors declared that this study received no financial support.

Use of AI for Writing Assistance: No artificial intelligence (AI)-assisted technologies, including large language models (LLMs), chatbots, or image generators, were used in the production of this manuscript.

Author Contributions: Concept – N.B.D., A.K.D., S.E., E.Ç.Ş.; Design – N.B.D., E.Ç.Ş.; Supervision – N.B.D., E.Ç.Ş.; Resource – N.B.D., A.K.D., S.E.; Materials – N.B.D., A.K.D., S.E.; Data Collection and/or Processing – N.B.D., A.K.D.; Analysis and/or Interpretation – N.B.D.; Literature Review – N.B.D., E.Ç.Ş.; Writing – N.B.D.; Critical Review – E.Ç.Ş.

Acknowledgments: Assistant Professor Dr. Muzaffer Bilgin is kindly acknowledged for contributing to the statistical analysis.

Peer-review: Externally peer-reviewed.

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