



Ann Med Res

Current issue list available at [AnnMedRes](http://AnnMedRes)

Annals of Medical Research

journal page: [www.annalsmedres.org](http://www.annalsmedres.org)

# CHA<sub>2</sub>DS<sub>2</sub>-VASc score as a predictor of new onset atrial fibrillation in patients with non-ST segment elevation myocardial infarction who underwent percutaneous coronary intervention

✉ Mehdi Karasu<sup>a,\*</sup>, ✉ Erkan Yildirim<sup>b</sup>, ✉ Yucel Karaca<sup>a</sup>, ✉ Mehmet Ali Kobat<sup>b</sup>

<sup>a</sup>Elazığ Fethi Sekin City Hospital, Department of Cardiology, Elazığ, Türkiye

<sup>b</sup>Fırat University, Faculty of Medicine, Department of Cardiology, Elazığ, Türkiye

## ARTICLE INFO

### Keywords:

NSTEMI

Atrial fibrillation

CHA<sub>2</sub>DS<sub>2</sub>-VASc

Received: Aug 22, 2023

Accepted: Oct 09, 2023

Available Online: 25.10.2023

DOI:

[10.5455/annalsmedres.2023.08.212](https://doi.org/10.5455/annalsmedres.2023.08.212)

## Abstract

**Aim:** New occurrence of atrial fibrillation (NOAF) frequently accompanies acute coronary syndromes (ACS), leading to unfavorable short- and long-term outcomes. Nonetheless, the existing risk stratification model for forecasting NOAF in cases of non-ST-segment elevation myocardial infarction (NSTEMI) necessitates further elucidation. Certain variables within the CHA<sub>2</sub>DS<sub>2</sub>-VASc score exhibit close associations with atrial fibrillation (AF) onset.

**Materials and Methods:** This retrospective inquiry encompassed 670 successive NSTEMI individuals seeking treatment at our cardiovascular center from June 2020 to June 2022, all of whom received percutaneous coronary intervention (PCI).

**Results:** Incidences of NOAF emerged during inpatient care for 55 individuals (12.5%). NOAF cases were characterized by advanced age, elevated high-sensitivity C-reactive protein (hs-CRP) levels, augmented left atrial volume index, Post-PCI thrombolysis in myocardial infarction (TIMI) grades below 3, heightened CHA<sub>2</sub>DS<sub>2</sub>-VASc scores, maximal troponin I peak (ng/ml), and SYNTAX scores (SS). Subsequent to univariate logistic regression analysis targeting predictors of NOAF incidence, CHA<sub>2</sub>DS<sub>2</sub>-VASc score, sub-3 Post-PCI TIMI grade, hemoglobin levels, hsCRP levels, and SS emerged as predictors; however, multivariate analysis identified CHA<sub>2</sub>DS<sub>2</sub>-VASc score, sub-3 Post-PCI TIMI grade, and hemoglobin levels as pivotal determinants.

**Conclusion:** The potential of the CHA<sub>2</sub>DS<sub>2</sub>-VASc score as a predictive tool for anticipating NOAF subsequent to PCI in NSTEMI cases is apparent. With the exception of components inherent to the CHA<sub>2</sub>DS<sub>2</sub>-VASc score, post-PCI TIMI grades below 3 and reduced hemoglobin levels stand as autonomous risk influencers for NSTEMI-NOAF.



Copyright © 2023 The author(s) - Available online at [www.annalsmedres.org](http://www.annalsmedres.org). This is an Open Access article distributed under the terms of Creative Commons Attribution-NonCommercial-NoDerivatives 4.0 International License.

## Introduction

Various forms of cardiac arrhythmias can manifest during coronary interventions and hospital stays in individuals experiencing acute coronary syndromes (ACS). Among these, atrial fibrillation (AF) stands out, presenting a prevalent occurrence and correlating with unfavorable prognostic implications [1]. AF is seen with a frequency of 6%-21% in ACS patients [2]. Arrhythmias, which alone have a great effect on mortality and morbidity, accompany diseases with high mortality such as NSTEMIs, further increasing the mortality rate.

Numerous investigations have elucidated an elevated mortality risk among individuals experiencing acute coronary

syndromes (ACS) concomitant with atrial fibrillation (AF) [3]. Particularly, patients encountering new-onset atrial fibrillation (NOAF) subsequent to ACS, who had not previously received an AF diagnosis, demonstrated elevated mortality rates in comparison to ACS patients with pre-existing AF records [4]. The advent of NOAF frequently complicates ACS occurrences, ushering in detrimental short- and long-term consequences. This arrhythmia's potential to induce swift ventricular rhythms, oxygen deprivation, reduced blood pressure, impaired atrial contraction, and atrioventricular discordance further exacerbates acute ischemic events and heart failure. Hospitalization and subsequent 6-month periods manifest augmented mortality rates, along with increased incidents of major adverse cardiac events (MACE), within patients beset by NOAF, contrasting patients devoid of AF instances [5].

\*Corresponding author:

Email address: [mehdikarasu@yahoo.com](mailto:mehdikarasu@yahoo.com) (✉ Mehdi Karasu)

The prevalence of new-onset atrial fibrillation (NOAF) among individuals hospitalized due to acute coronary syndromes (ACS) displays significant variability, spanning from 2% to 37% [6]. Predictive factors associated with the emergence of NOAF within ACS patients encompass advancing age, female gender, prior history of diabetes mellitus (DM), hypertension (HT), elevated high-sensitivity C-reactive protein (hsCRP) and brain natriuretic peptides (BNP) levels, augmented left atrial dimensions, presence of heart failure symptoms, heightened heart rate, diminished blood pressure, and compromised left ventricular function [7]. Nonetheless, the current model for risk classification intended to gauge the likelihood of NOAF during instances of non-ST-segment elevation myocardial infarction (NSTEMI) remains obscured.

The CHA<sub>2</sub>DS<sub>2</sub>-VASc score is the most widely used clinical score for estimating the risk of stroke in patients with atrial fibrillation (AF) [8]. Notably, certain elements encapsulated within the CHA<sub>2</sub>DS<sub>2</sub>-VASc score, namely congestive heart failure (CHF), hypertension (HT), advanced age, female gender, and diabetes mellitus (DM), exhibit a strong association with the onset of atrial fibrillation (AF).

Our study aims to investigate the potential of the CHA<sub>2</sub>DS<sub>2</sub>-VASc score as a predictive factor for NOAF in NSTEMI patients. Our research seeks to enable the development of early intervention strategies to reduce NOAF incidence and improve patient outcomes.

## Materials and Methods

This retrospective investigation enrolled 670 consecutive patients diagnosed with non-ST-segment elevation myocardial infarction (NSTEMI) who underwent percutaneous coronary intervention (PCI) at our cardiovascular center between June 2020 and June 2022. Exclusions encompassed autoimmune disorders, hyperthyroidism, severe infections, neoplastic ailments, and chronic liver conditions. Patients with prior atrial fibrillation (AF) or atrial flutter, moderate to severe valve pathologies, or scheduled bypass surgery after Coronary Angiography (CAG) were also omitted. Accordingly, 232 subjects were eliminated, yielding a final study population of 438 patients. Ethical approval was obtained from the Firat University Ethics Committee (Number of sessions: 2023/ 04- 32).

Coronary Angiography and PCI procedures were conducted, adhering to contemporary guidelines for NSTEMI patients. Expert cardiologists, blind to patients' clinical details, evaluated fluoroscopic imagery. In cases of dissenting interpretations, a third-party observer provided input, culminating in a consensus-driven verdict. Lesions causing over 50% luminal occlusion in vessels larger than 1.5 mm contributed to the overall SYNTAX score (SS). The cumulative SS was computed utilizing online calculator version 2.11 ([www.syntaxscore.com](http://www.syntaxscore.com)). Revascularization determinations were left to the physician's discretion. Following PCI, patients received aspirin, clopidogrel, prasugrel, or ticagrelor, along with statins. The usage of angiotensin-converting enzyme inhibitors and adrenergic blocking agents was left to the interventional cardiologist's judgment.

NSTEMI diagnosis rested on characteristic prolonged ischemic symptoms, concurrent with coronary artery disease

(CAD), and distinctive elevation and reduction of serum cardiac troponin I (CTnI), coupled with ST segment depression and/or notable T wave inversion on initial electrocardiography (ECG) [9]. The first ECG was administered within the initial 10 minutes of admission, with continuous ECG monitoring during acute myocardial infarction (AMI) to detect arrhythmias. All patients underwent continuous ECG monitoring during the first 48 h following the pPCI procedure. When arrhythmia were detected 12-lead ECG records were obtained. Atrial fibrillation (AF) was identified by the absence of P waves lasting a minimum of 30 seconds, irregular R-R intervals, and an obscured isoelectric line [10]. New-onset atrial fibrillation (NOAF) denoted patients lacking prior AF diagnoses, presenting with sinus rhythm upon admission, and subsequently developing AF during hospitalization.

Data encompassing patients' demographics, family and medical histories, weight, height, and smoking status were documented upon admission. Body mass index (BMI) was computed as weight divided by height squared (kg/m<sup>2</sup>). Estimated glomerular filtration rate (eGFR) was calculated via the Cockcroft-Gault formula [11]. Transthoracic echocardiography was executed within 4 hours of admission, measuring left atrial diameter (LAD) and determining left atrial volume index (LAVI) through the bi-plane area length method indexed by body surface area [12]. Left ventricular ejection fraction (LVEF) was assessed post-coronary intervention using the modified Simpson's method [13]. Coronary Angiography (CAG) results were used to identify the infarct-related artery (IRA), and laboratory analyses were obtained following initial ECG readings.

The CHA<sub>2</sub>DS<sub>2</sub>-VASc score, attributing 1 point to congestive heart failure (CHF), hypertension (HT), age 65-74, diabetes mellitus (DM), vascular disease, and female gender, and 2 points to age  $\geq$  75 years and prior stroke or transient ischemic attack (TIA), was computed [14]. Notably, existing NSTEMI event was not incorporated in the score calculation due to its universality among all patients.

## Statistical analysis

Continuous variables were assessed for conformity to a normal distribution using the Kolmogorov-Smirnov test. For normally distributed data, descriptive statistics were expressed as mean  $\pm$  standard deviation (SD) and subjected to Student's t-test. Non-normally distributed variables were presented as medians (interquartile range) and subjected to analysis through the Mann-Whitney U test. Binary variables were presented as proportions and analyzed using the Pearson Chi-square test.

Univariate analysis and multivariate logistic regression(enter) were employed to identify factors associated with the risk of new-onset atrial fibrillation (NOAF). To assess the discriminatory capability of the CHA<sub>2</sub>DS<sub>2</sub>-VASc score, Receiver Operating Characteristic (ROC) curves and corresponding Area Under the Curve (AUC) values were computed. Statistical significance was established at a two-tailed P-value of less than 0.05. The statistical software package SPSS version 24.0 (IBM Corp, Armonk, NY) was utilized for all data analyses.

## Results

A total of 438 consecutive patients diagnosed with acute myocardial infarction (AMI), of which 268 were male, were enrolled in the study. These patients had no prior history of atrial fibrillation (AF). The baseline characteristics of the participants are detailed in Table 1. During hospitalization, new-onset atrial fibrillation (NOAF) emerged in 55 patients (12.5%). Individuals experiencing NOAF were characterized by older age and elevated levels of high-sensitivity C-reactive protein (hs-CRP), left atrial volume index (LAVI), and peak cardiac troponin I (CTnI), as well as a higher CHA<sub>2</sub>DS<sub>2</sub>-VASc score and SYNTAX score (SS). Notably, the NOAF group exhibited a lower left ventricular ejection fraction (LVEF). The utilization of angiotensin-converting enzyme inhibitors (ACEI)/angiotensin receptor blockers (ARBs), beta blockers, statins, and calcium antagonists displayed no significant inter-group differences.

Angiographic assessments, as presented in Table 2, revealed a higher incidence of right coronary artery (RCA) lesions among patients with NOAF, yet no substantial variance was noted concerning the diseased vessel distribution.

Upon univariate logistic regression analysis to ascertain predictors of NOAF, variables including the CHA<sub>2</sub>DS<sub>2</sub>-VASc score, post-PCI TIMI grade <3, hemoglobin levels, hsCRP levels, and SS emerged as predictive factors. However, subsequent multivariate analysis highlighted that the CHA<sub>2</sub>DS<sub>2</sub>-VASc score, post-PCI TIMI grade <3, and hemoglobin levels retained determining influence. To mitigate multicollinearity, risk factors within the CHA<sub>2</sub>DS<sub>2</sub>-VASc score were excluded from this analysis.

Further analyses encompassing individual risk factors from the CHA<sub>2</sub>DS<sub>2</sub>-VASc score, conducted through both univariate and multivariate logistic regression, identified age  $\geq 75$  years, history of stroke, transient ischemic attack (TIA), or thromboemboli (TE), vascular disease, and age range of 65-74 years as independent determinants of NOAF incidence (Table 4).

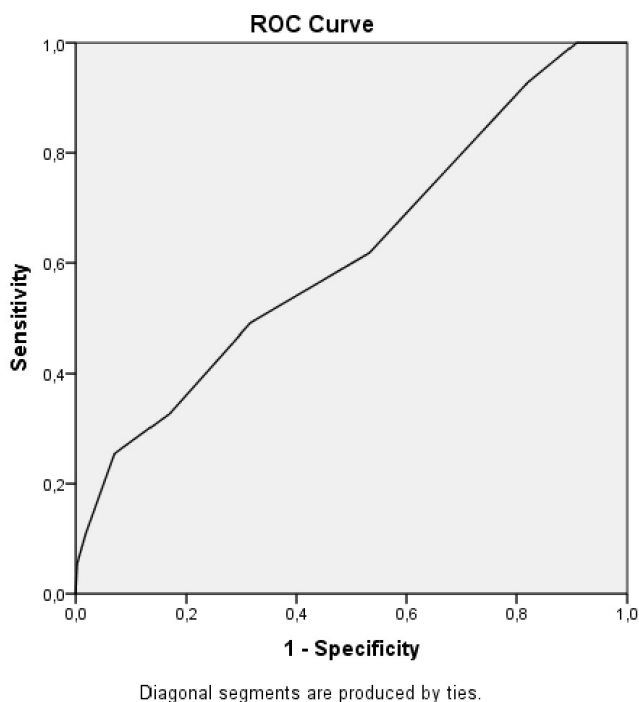
Significantly higher CHA<sub>2</sub>DS<sub>2</sub>-VASc scores were observed in NOAF patients compared to those without NOAF. Receiver Operating Characteristic (ROC) curve analysis demonstrated that the CHA<sub>2</sub>DS<sub>2</sub>-VASc score possessed acceptable discriminatory capability for prognosticating post-AMI NOAF, illustrated by an Area Under the Curve (AUC) of 0.619 (95% CI, P = 0.004), as depicted in Table 5 and Figure 1.

## Discussion

Our study findings underscore that the CHA<sub>2</sub>DS<sub>2</sub>-VASc score serves as a valuable predictor of new-onset atrial fibrillation (NOAF) following percutaneous coronary intervention (PCI) in patients with non-ST-segment elevation myocardial infarction (NSTEMI). Remarkably, a CHA<sub>2</sub>DS<sub>2</sub>-VASc score of  $\geq 2.5$  displayed promise as a potential threshold value, yielding a sensitivity of 61.8% and specificity of 46.7%. Our observed NOAF incidence rate of 12.5% among NSTEMI patients aligned with findings from comparable studies [2,15].

Predictive factors associated with the emergence of NOAF within ACS patients encompass advancing age, female gender, prior history of diabetes mellitus (DM), hypertension (HT), elevated high-sensitivity C-reactive protein (hsCRP) and brain natriuretic peptides (BNP) levels, augmented left atrial dimensions, presence of heart failure symptoms, heightened heart rate, diminished blood pressure, and compromised left ventricular function [7]. Notably, certain elements encapsulated within the CHA<sub>2</sub>DS<sub>2</sub>-VASc score, namely congestive heart failure (CHF), hypertension (HT), advanced age, female gender, and diabetes mellitus (DM), exhibit a strong association with the onset of atrial fibrillation (AF).

The predictive capacity of the CHA<sub>2</sub>DS<sub>2</sub>-VASc score for post-NSTEMI NOAF development was demonstrated through acceptable discrimination, as evidenced by a Receiver Operating Characteristic (ROC) curve analysis. These findings align with those from other investigations, such as Huang SS et al., who evidenced a heightened incidence of new-onset AF in acute myocardial infarction (AMI) patients with higher CHADS<sub>2</sub> scores [16]. Likewise, Lau KK et al. demonstrated the utility of CHADS<sub>2</sub> and CHA<sub>2</sub>DS<sub>2</sub>-VASc scores in identifying post-ST elevation myocardial infarction (STEMI) patients at elevated risk for AF and ischemic stroke [17]. A study focusing on the elderly population revealed the predictive potential of the CHA<sub>2</sub>DS<sub>2</sub>-VASc score, in conjunction with high-sensitivity C-reactive protein (hs-CRP), for NOAF development in acute coronary syndrome (ACS) patients [18]. F. Aksoy et al. highlighted the relative robustness of CHADS<sub>2</sub> and CHA<sub>2</sub>DS<sub>2</sub>-VASc scores in predicting NOAF subsequent to STEMI [19]. However, our study's distinctiveness stems from its exclusive inclusion of NSTEMI patients, devoid of age limitations.



**Figure 1.** Receiver operating characteristic curves for CHA<sub>2</sub>DS<sub>2</sub>-VASc score in study groups.

**Table 1.** The baseline characteristics of patients with and without new-onset atrial fibrillation (NOAF).

	In hospital NOAF		p
	Without NOAF (n=383)	With NOAF (n=55)	
Age (years)	60.93 ± 10.7	66.25 ± 10.4	<b>0.001</b>
Gender (F/M)	143/240	27/28	0.094
Smoking (%)	216 (56.4)	22 (40)	0.205
Heart rate (bpm)	77.1 ± 11.4	79.9 ± 13.4	0.394
SBP (mm Hg)	138.7 ± 18.5	143.1 ± 16.2	0.325
DBP (mmHg)	81.7 ± 12	84.2 ± 10.3	0.267
Creatinine (mg/dl)	0.97 ± 0.1	0.95 ± 0.1	0.066
eGFR (ml/min)	83.6 ± 23.6	82.5 ± 23.3	0.243
WBC count (10 <sup>3</sup> /μl)	9.6 ± 2.4	10.4 ± 3.5	<b>0.014</b>
Haemoglobin (g/dl)	13.3 ± 1.8	12.6 ± 1.5	<b>0.001</b>
Platelets/mm <sup>3</sup>	235 ± 56	245 ± 51	<b>0.015</b>
Peak troponin I (ng/ml)	1235 ± 1456	3458 ± 2875	<b>0.002</b>
hs-CRP (mg/L)	3.5 ± 2.1	4.4 ± 2.2	<b>0.008</b>
Uric acid (mmol/l)	5.6 ± 1.5	6.2 ± 1.6	0.015
Total cholesterol, mg/dl	195 ± 35	200 ± 38	0.338
Triglyceride, mg/dl	163 ± 56	168 ± 44	0.759
LDL-C, mg/dl	135 ± 30	136 ± 31	0.853
HDL-C, mg/dl	45 ± 10	42 ± 9	0.447
E/E'	13.2 ± 1.8	14.1 ± 1.8	<b>0.003</b>
LAVI (mL/m <sup>2</sup> )	24.4 ± 4.2	27.7 ± 5.3	<b>&lt;0.001</b>
Ejection fraction (%)	51.9 ± 8.1	46.5 ± 7.2	<b>0.012</b>
Post PCI TIMI grade <3 n (%)	67 (17.5)	36 (65.4)	<b>&lt;0.001</b>
SYNTAX score	15.2 ± 4.2	18.3 ± 4.7	<b>&lt;0.001</b>
CHA <sub>2</sub> DS <sub>2</sub> -VASc score	2.84 ± 1.6	3.78 ± 2.0	<b>&lt;0.001</b>
<b>Comorbidity</b>			
Hypertension (%)	263 (68.7)	36 (65.5)	0.644
Diabetes Mellitus (%)	165 (43.1)	33 (60.0)	<b>0.021</b>
CAD	216 (56.5)	31 (56.3)	0.205
Dyslipidemia (%)	132 (34.5)	19 (34.5)	0.991
PAD (%)	180 (47.0)	32 (58.2)	0.121
Stroke, TIA,TE (%)	60(15.7)	24(43.6)	<b>&lt;0.001</b>
<b>Medications</b>			
ACE-I/ARB	221 (57.7)	33 (60.0)	0.735
Beta blocker	218 (56.9)	35 (63.6)	0.383
Statin	207 (54.0)	25 (45.5)	0.233
Calcium antagonist	127 (33.2)	19 (34.5)	0.657
ASA	232 (60.6)	42 (76.4)	0.066

Abbreviations: ASA, acetylsalicylic acid; ACE-I, angiotensin-converting enzyme inhibitor; ARB, angiotensin receptor blocker; CAD, coronary artery disease; DBP, diastolic blood pressure; eGFR, estimated glomerular filtration rate; hsCRP, high-sensitivity C reactive protein; HDL-C, high-density lipoprotein cholesterol; LAVI, left atrial volume index; LDL-C, low density lipoprotein cholesterol; PAD, peripheral artery disease; PCI, percutaneous coronary intervention; SBP, systolic blood pressure; SYNTAX, Synergy between PCI with Taxus and Cardiac Surgery; TIMI, thrombolysis in myocardial infarction; WBC, white blood cell.

Our investigation determined that age ≥75 years, history of stroke, transient ischemic attack (TIA), or thromboemboli (TE), vascular disease, and age range of 65-74 years—components of the CHA<sub>2</sub>DS<sub>2</sub>-VASc score—served as autonomous risk factors for NSTEMI-NOAF. Advanced age's role in NOAF development, corroborated by previous studies [7,15], was consistent with our findings. Yuan FU et al. demonstrated a comparable outcome, revealing advanced age as an independent risk factor for NOAF in ACS patients [18].

Likewise, a history of stroke, TIA, or TE has been consistently

linked to NOAF occurrence in ACS patients [20], as mirrored in our study. Notably, the occurrence of stroke-TIA history was more prevalent in NOAF patients, reinforcing previous research's conclusions.

Post PCI TIMI grade <3 emerged as an independent risk factor for NSTEMI-NOAF in our study, complementing earlier findings that patients exhibiting any form of AF frequently present with an initial TIMI grade <3 [21]. This concurrence was also noted regarding PCI TIMI <3 grades among NOAF patients.

**Table 2.** The angiographic characteristics of patients with and without new onset atrial fibrillation (NOAF).

	In hospital NOAF		p
	No (n=383)	Yes (n=55)	
<b>Infarct-related artery (%)</b>			
LAD	131 (34.2)	23 (41.8)	0.432
LCx	101 (26.4)	19 (34.5)	0.243
RCA	73 (18.4)	18 (35.3)	0.02
Other	14 (3.8)	1 (2)	0.420
LMCA	18 (4.7)	0	0.101
<b>Number of diseased vessels (%)</b>			
1	205 (53.5)	30 (55.8)	0.778
2	98 (25.6)	20 (36.4)	0.214
3	80 (21.1)	5 (7.8)	0.062

Abbreviations: LAD, left anterior descending coronary artery; LCx, left circumflex coronary artery; LMCA, left main coronary artery; RCA, right coronary artery.

**Table 3.** Univariate and Multivariate Regression Analysis of Predictors of new-onset atrial fibrillation (NOAF) in Study Population.

Variables	Univariate analysis		Multivariate analysis	
	OR (95% CI)	p	OR (95% CI)	p
CHA <sub>2</sub> DS <sub>2</sub> -VASc score	1.348 (1.150-1.581)	<b>&lt;0.001</b>	1.246 (1.051-1.477)	<b>0.011</b>
Post PCI TIMI grade <3	0.232 (0.154-0.349)	<b>0.001</b>	0.297 (0.191-0.461)	<b>0.021</b>
Hemoglobin	0.749 (0.634-0.885)	<b>0.001</b>	0.813 (0.679-0.972)	<b>0.024</b>
hsCRP	1.227 (1.074-1.401)	<b>0.003</b>	1.108 (0.952-1.290)	0.184
SYNTAX score	1.126 (1.066-1.188)	<b>&lt;0.001</b>	1.060 (0.998-1.125)	0.058

Abbreviations: CI, confidence interval; hsCRP, high-sensitivity C reactive protein; OR, odds ratio; PCI, percutaneous coronary intervention; SYNTAX, Synergy between PCI with Taxus and Cardiac Surgery; TIMI, thrombolysis in myocardial infarction.

**Table 4.** Univariate and Multivariate Analysis of Predictive Power of Individual Risk Factors in CHA<sub>2</sub>DS<sub>2</sub>-VASc Score for new-onset atrial fibrillation (NOAF).

Variables	Univariate analysis		Multivariate analysis	
	OR (95% CI)	p	OR (95% CI)	p
Congestive heart failure	1.815 (0.652-5.052)	0.254	0.940 (0.302-2.926)	0.915
Hypertension	0.865 (0.476-1.569)	0.632	0.575 (0.292-1.133)	0.110
Age ≥75	2.385 (1.188-4.787)	0.015	2.444 (1.619-3.368)	<b>0.011</b>
Diabetes mellitus	1.982 (1.114-3.526)	0.020	1.336 (0.638-2.799)	0.442
Stroke, TIA, or TE	4.168 (2.288-7.593)	<0.001	6.717 (2.903-15.541)	<b>&lt;0.001</b>
Vascular disease	0.768 (0.513-1.150)	0.200	0.451 (0.224-0.909)	<b>0.026</b>
Age 65-74	2.068 (1.162-3.681)	0.013	2.203 (1.097-4.425)	<b>0.026</b>
Female gender	0.618 (0.350-1.090)	0.097	0.619 (0.336-1.141)	0.124

Abbreviations: CI, confidence interval; OR, odds ratio; TE, thromboembolic event; TIA, transient ischemic attack.

**Table 5.** Sensitivity, specificity, AUC, cut-off and asymptotic significance of CHA<sub>2</sub>DS<sub>2</sub>-VASc score in study group.

	Sensitivity (%)	Specificity (%)	AUC	Cut-off	p
CHA <sub>2</sub> DS <sub>2</sub> -VASc score	61.8	46.7	0.619	2.5	<b>0.004</b>

Abbreviations: AUC, area under curve.

Associations between low hemoglobin levels, anemia, and NOAF development have been established in prior studies [22]. Anemia's influence on myocardial hypertrophy, chamber enlargement, heart rate elevation, oxygen deliv-

ery reduction, and hypoxia intensification can contribute to heart failure and AF, consistent with our observations [23,24].

Inflammatory processes' roles in atherosclerosis and AF

complications are well-established [25], particularly in the context of ACS-AF coexistence [26]. Our study corroborated this, with NOAF patients presenting higher levels of hsCRP, uric acid, white blood cells (WBC), and platelets.

While the number of diseased vessels showed no substantial link with NOAF in our study, patients experiencing NOAF exhibited elevated SYNTAX scores (SS). RCA lesions were more prevalent among NOAF patients, possibly due to RCA-related ischemia affecting nodes or atria [27].

Higher left atrial volume index (LAVI) values among NOAF patients resonated with literature highlighting LAVI's representation of true atrial size and its association with abnormal filling pressures [12,28].

Our study also unveiled the significance of diastolic dysfunction, reflected through E/E' values, in influencing NOAF development, likely via increased left atrial pressure [29,30]. This revelation adds to the multifactorial nature of NOAF development in the context of ACS.

In summary, our study highlights the clinical significance of the CHA<sub>2</sub>DS<sub>2</sub>-VASc score as a valuable predictive tool for NOAF following PCI in NSTEMI patients. It indicates that a CHA<sub>2</sub>DS<sub>2</sub>-VASc score of  $\geq 2.5$  may serve as a potential threshold, with notable sensitivity and specificity. These findings align with previous research and emphasize the importance of early intervention strategies to mitigate the emergence of NOAF and improve patient outcomes in this specific patient population. Additionally, our study provides insights into the contributions of individual risk factors within the CHA<sub>2</sub>DS<sub>2</sub>-VASc score and other clinical parameters to the development of NOAF in NSTEMI patients.

### Limitations

Several limitations are inherent to our study. Firstly, its single-center nature restricts the generalizability of the findings to broader populations. Additionally, the study's lack of auxiliary tools like fractional flow reserve and intravascular ultrasound precludes comprehensive assessment of coronary artery disease (CAD) severity and extent.

### Conclusion

Our study underscores the utility of the CHA<sub>2</sub>DS<sub>2</sub>-VASc score as a predictive tool for new-onset atrial fibrillation (NOAF) following percutaneous coronary intervention (PCI) in patients without ST-segment elevation myocardial infarction (STEMI). Beyond the components encompassed by the CHA<sub>2</sub>DS<sub>2</sub>-VASc score, the presence of post-PCI TIMI grade  $< 3$  and lower hemoglobin levels emerge as autonomous risk factors for NOAF in the context of non-STEMI. These findings accentuate the potential of the CHA<sub>2</sub>DS<sub>2</sub>-VASc score in risk assessment and the identification of NOAF-prone individuals, thereby contributing to the development of targeted prevention and management strategies for this patient population.

### Ethical approval

Ethical approval was obtained from the Firat University Ethics Committee (Number of sessions: 2023/ 04- 32).

### References

1. Kundu A, O'Day K, Shaikh AY, et al. Relation of atrial fibrillation in acute myocardial infarction to in-hospital complications and early hospital readmission. *Am J Cardiol.* 2016;117(8):1213–1218.
2. Schmitt J, Duray G, Gersh BJ, et al. Atrial fibrillation in acute myocardial infarction: a systematic review of the incidence, clinical features and prognostic implications. *Eur Heart J.* 2009;30(9):1038-1045.
3. Rene AG, Genereux P, Ezekowitz M, et al. Impact of atrial fibrillation in patients with ST-elevation myocardial infarction treated with percutaneous coronary intervention (from the HORIZONS-AMI [Harmonizing Outcomes with Revascularization and Stents in Acute Myocardial Infarction] trial). *Am J Cardiol.* 2014;113(2):236–242.
4. Kober L, Swedberg K, McMurray JJ, et al. Previously known and newly diagnosed atrial fibrillation: a major risk indicator after a myocardial infarction complicated by heart failure or left ventricular dysfunction. *Eur J Heart Fail.* 2006;8(6):591-98.
5. Worme MD, Tan MK, Armstrong DWJ, et al. Previous and New Onset Atrial Fibrillation and Associated Outcomes in Acute Coronary Syndromes (from the Global Registry of Acute Coronary Events). *Am J Cardiol.* 2018 Sep 15;122(6):944-951.
6. Ben Halima M, Yaakoubi W, Boudiche S, et al. New-onset atrial fibrillation after acute coronary syndrome: prevalence and predictive factors. *Tunis Med.* 2022 Feb;100(2):114-121.
7. He J, Yang Y, Zhang G, et al. Clinical risk factors for new-onset atrial fibrillation in acute myocardial infarction: a systematic review and meta-analysis. *Medicine.* 2019;98(26):e15960.
8. Hindricks G, Potpara T, Dagres N, et al. 2020 ESC Guidelines for the diagnosis and management of atrial fibrillation developed in collaboration with the European Association for Cardio-Thoracic Surgery (EACTS): The Task Force for the diagnosis and management of atrial fibrillation of the European Society of Cardiology (ESC) Developed with the special contribution of the European Heart Rhythm Association (EHRA) of the ESC [published correction appears in *Eur Heart J.* 2021 Feb 1;42(5):507] [published correction appears in *Eur Heart J.* 2021 Feb 1;42(5):546-547] [published correction appears in *Eur Heart J.* 2021 Oct 21;42(40):4194]. *Eur Heart J.* 2021;42(5):373-498.
9. Roffi M, Patrono C, Collet JP, et al. ESC Guidelines for the management of acute coronary syndromes in patients presenting without persistent ST-segment elevation: Task Force for the Management of Acute Coronary Syndromes in Patients Presenting without Persistent ST-Segment Elevation of the European Society of Cardiology (ESC) *Eur Heart J.* 2016;37(3):267–315.
10. Vardas P, Agewall S, Camm J, et al. ESC Guidelines for the management of atrial fibrillation developed in collaboration with EACTS. *Europace Eur Pacing Arrhythm Card Electrophysiol J Work Groups Card Pacing Arrhythm Card Cell Electrophysiol Eur Soc Cardiol.* 2016;18(11):1609–1678.
11. Cockcroft DW, Gault H. Prediction of creatinine clearance from serum creatinine. *Nephron.* 1976;16(1):31-41.
12. Lester SJ, Ryan EW, Schiller NB, et al. Best method in clinical practice and in research studies to determine left atrial size. *Am J Cardiol.* 1999;84(7):829-32.
13. Lang RM, Bierig M, Devereux RB, et al. Recommendations for chamber quantification: a report from the American Society of Echocardiography's Guidelines and Standards Committee and the Chamber Quantification Writing Group, developed in conjunction with the European Association of Echocardiography, a branch of the European Society of Cardiology. *J Am Soc Echocardiogr.* 2005;18(12):1440-63.
14. January C, Wann L, Alpert J, et al. AHA/ACC/HRS guideline for the management of patients with atrial fibrillation: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines and the Heart Rhythm Society. *J Am Coll Cardiol.* 2014;64(21):e1–76.
15. Wang J, Yang YM, Zhu J. Mechanisms of new-onset atrial fibrillation complicating acute coronary syndrome. *Herz.* 2015; 40(suppl 1):18-26.
16. Huang SS, Chan WL, Leu HB, et al. Association between CHADS2 score and the preventive effect of statin therapy on new-onset atrial fibrillation in patients with acute myocardial infarction. *PLoS ONE.* 2013;8(8):e74709.

17. Lau KK, Chan PH, Yiu KH, et al. Roles of the CHADS2 and CHA2DS2-VASc scores in post-myocardial infarction patients: risk of new occurrence of atrial fibrillation and ischemic stroke. *Cardiol J.* 2014;21(5):474–483.
18. Yuan Fu, Yuxia Pan, Yuanfeng Gao, et al. Predictive value of CHA2DS2-VASc score combined with hs-CRP for new-onset atrial fibrillation in elderly patients with acute myocardial infarction. *BMC Cardiovasc Dis.* volume 21, Article number: 175 (2021).
19. Aksoy F, Bař HA, Bařcı A, et al. The CHA2DS2-VASc score for predicting atrial fibrillation in patients presenting with ST elevation myocardial infarction: prospective observational study. *Med J.* 2019 Jul 22;137(3):248-254.
20. Yi JE, Seo SM, Lim S, et al. Gender Differences in the Impact of New-Onset Atrial Fibrillation on Long-Term Risk of Ischemic Stroke after Acute Myocardial Infarction. *J Clin Med.* 2021 Nov 1;10(21):5141.
21. Pizzetti F, Turazza FM, Franzosi MG, et al. Incidence and prognostic significance of atrial fibrillation in acute myocardial infarction: the GISSI-3 data. *Heart.* 2001;86(5):527-32.
22. Woo-Hyun L, Eue-Keun C, Kyung-Do H, et al. Impact of hemoglobin levels and their dynamic changes on the risk of atrial fibrillation: a nationwide population-based study. *Sci Rep.* 2020;10:1–8.
23. Schwartz AJ, Converso-Baran K, Michele DE, et al. A genetic mouse model of severe iron deficiency anemia reveals tissue-specific transcriptional stress responses and cardiac remodeling. *J Biol Chem.* 2019;294:14991–15002.
24. Metivier F, Marchais SJ, Guerin AP, et al. Pathophysiology of anaemia: focus on the heart and blood vessels. *Nephrol Dial Transplant.* 2000;15(suppl 3):14-8.
25. Van Wagoner DR. Oxidative stress and inflammation in atrial fibrillation: role in pathogenesis and potential as a therapeutic target. *J Cardiovasc Pharmacol.* 2008;52(4):306-13.
26. Sayın MR, Özderya A, Konuş AH, et al. The use of systemic immune-inflammation index to predict new onset atrial fibrillation in the context of acute coronary syndrome. *Kardiologija.* 2022 Aug 30;62(8):59-64.
27. Alasady M, Abhayaratna WP, Leong DP, et al. Coronary artery disease affecting the atrial branches is an independent determinant of atrial fibrillation after myocardial infarction. *Heart Rhythm.* 2011;8(7):955-60.
28. Wi J, Shin DH, Kim JS, et al. Transient new-onset atrial fibrillation is associated with poor clinical outcomes in patients with acute myocardial infarction. *Circ J.* 2016;80(7):1615-23.
29. Celik S, Erdođ C, Baykan M, et al. Relation between paroxysmal atrial fibrillation and left ventricular diastolic function in patients with acute myocardial infarction. *Am J Cardiol.* 2001;88(2):160-2.
30. Geske JB, Sorajja P, Nishimura RA, et al. Evaluation of left ventricular filling pressures by Doppler echocardiography in patients with hypertrophic cardiomyopathy: correlation with direct left atrial pressure measurement at cardiac catheterization. *Circulation.* 2007;116(23):2702-8.