Original Article

Naples Prognostic Score Predicts New-Onset Atrial Fibrillation in Patients with ST-Elevated Myocardial Infarction Undergoing Primary Angioplasty

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Abstract

Background: **New-onset atrial fibrillation (NOAF) is a typical complication in patients with ST-segment elevated myocardial infarction (STEMI) patients undergoing primary percutaneous coronary intervention (pPCI). Previous studies have investigated inflammation as a NOAF predictor. The Naples prognostic score (NPS) is a novel marker of inflammation and nutritional status.**

Objective: **The objective of this study was to evaluate the predictive power of the NPS for NOAF.**

Methods: **This study enrolled 1537 consecutive STEMI who underwent pPCI. The patients who presented NOAF during hospital admission and those who remained in sinus rhythm (RSR) were compared in terms of baseline characteristics. Univariate and multivariate analyses were carried out to identify variables predicting NOAF development, and $p < 0.05$ was considered statistically significant.**

Results: **NOAF was detected in 7.74% (n: 119) of the participants. The mean age (67.03±13.48 vs 57.84±11.31; $p < 0.001$) and NPS (2.53±1.17 vs 2.25±1.10, $p=0.008$) were significantly higher in the NOAF group. Multivariate analysis revealed age (Odds ratio [OR]: 1.045 for a year, 95% confidence interval [CI]: 1.019–1.071, $p=0.001$), NPS (OR: 1.645, 95% CI: 0.984–2.748, $p=0.037$) and left atrial dimensions (OR: 2.542 for cm, 95% CI: 1.488–4.342, $p=0.001$) as independent predictors of NOAF.**

Conclusions: **The NPS was an independent predictor of NOAF in STEMI patients, in addition to classical factors such as age and left atrial dimensions. This score, mostly related to an inflammatory burden, may help to predict NOAF incidence and select better potential therapies aimed at abating inflammation after myocardial infarction.**

Keywords: **Prognosis; Atrial Fibrillation; Myocardial Infarction; Angioplasty.**

Introduction

ST-segment elevation myocardial infarction (STEMI) is still associated with significant mortality and morbidity despite advancements in pharmacological and interventional techniques.[1,2] Atrial fibrillation (AF) is a typical type of heart arrhythmia with an increased prevalence in older adults. It is a leading cause of stroke. Typical causes of AF include advanced age, ischemic or structural heart disease, hypertension, hyperthyroidism, and alcohol consumption.[3] Increased frequencies of supraventricular and ventricular arrhythmias have been reported in patients with acute coronary syndromes. AF is the major supraventricular arrhythmia in patients with STEMI, leading to a 4–14% increase in primary percutaneous coronary interventions (pPCI) opportunities.[4]

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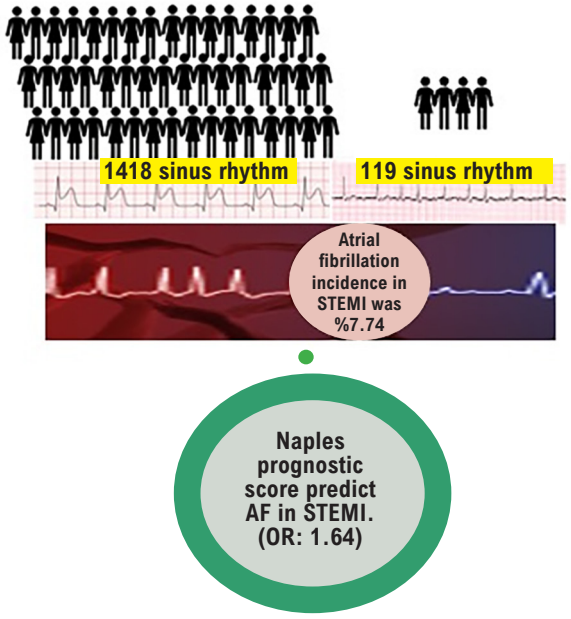
The pathophysiology of new-onset AF (NOAF) in the acute phase of STEMI depends on factors such as atrial ischemia, increased sympathetic activity, and inflammation during myocardial infarction.[5] The acute loss of atrial contractions during STEMI leads to diastolic deterioration and serious hemodynamic deterioration. In addition, NOAF may lead to a high ventricular rate, negatively impacting cardiac output. NOAF also increases the risk of stroke. Therefore, NOAF is associated with higher mortality rates in patients with STEMI.[6]

A close relationship between AF and systemic inflammation has been demonstrated.[7] The Naples Prognostic Score (NPS) was initially designed to predict prognosis in colorectal cancer using biomarkers of inflammation and nutrition.[8] The Naples prognostic score (NPS) is a feasible prognostic score that shows immunity, inflammation, and nutritional status, including total cholesterol, albumin, lymphocyte, monocyte, and neutrophil variables, and has been frequently included in recent publications on cardiovascular outcomes.[8-10] The higher neutrophil-to-lymphocyte ratio (NLR) and lower lymphocyte-to-monocyte ratio (LMR) indicate a pro-inflammatory state, while reduced serum albumin and cholesterol suggest poor nutritional status. These combined parameters provide a comprehensive inflammatory and

Central Illustration: Naples Prognostic Score Predicts New-Onset Atrial Fibrillation in Patients with ST-Elevated Myocardial Infarction Undergoing Primary Angioplasty



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nutritional assessment, which has been increasingly recognized as valuable in predicting cardiovascular outcomes, including NOAF in STEMI patients.[9,10]

Notably, to avoid mortality and morbidity in patients with STEMI, urgent action should be taken to prevent adverse events and predict complications such as NOAF. Therefore, in this study, we evaluated the predictive power of NPS for NOAF in patients with STEMI who underwent pPCI.

Materials and methods

Study population

This is a single-center, retrospective, cross-sectional study and contains patients between March 2013 and December 2019. In total, 1706 consecutive patients who underwent pPCI for STEMI were enrolled in this study. Notably, all patients over 18 years old and presented with ST-segment elevation in electrocardiography with at least two contiguous derivations and were treated with pPCI were included. The exclusion criteria were a history of paroxysmal and/or permanent AF, previous arrhythmia or ablation history, previous cardiac surgery, cardiac arrest, defibrillation or resuscitation before admission, early stent thrombosis, iatrogenic or spontaneous coronary dissection, coronary perforation, tamponade, end-stage chronic organ failure, end-stage malignancy, receiving chemotherapy, drug abuse, and refusing to undergo intervention or participate in the study. Overall, 169 patients were excluded based on the inclusion criteria, and the remaining 1537 patients were included in this study (Figure 1). The participants were divided into two groups: patients with NOAF and those who remained in sinus rhythm (RSR).

This study protocol was approved by the local ethics committee of Istanbul Cerrahpasa University Institute of Cardiology, and it was conducted according to the ethical subjects of the Helsinki Declaration and Good Clinical Practice Guidelines. Informed consent was obtained from all the participants included in this study.

Primary angioplasty procedure

Participants who were admitted to the emergency department with angina pectoris or angina-equivalent symptoms underwent electrocardiography (ECG) urgently within 10 min. Patients with ST-segment elevation in at least two contiguous derivations or a newly developed right or left bundle branch block were diagnosed with STEMI. Notably, all patients diagnosed with STEMI were administered 300 mg aspirin and a P2Y12 inhibitor (loading doses for clopidogrel: 600 mg; ticagrelor: 180 mg; prasugrel: 60 mg). All the medical treatments were administered in accordance with European Society of Cardiology (ESC) guidelines.[2]

The femoral artery access route was mostly used. However, radial artery access was used occasionally in patients undergoing anticoagulant treatment when an operator could not access femoral arteries due to peripheral artery

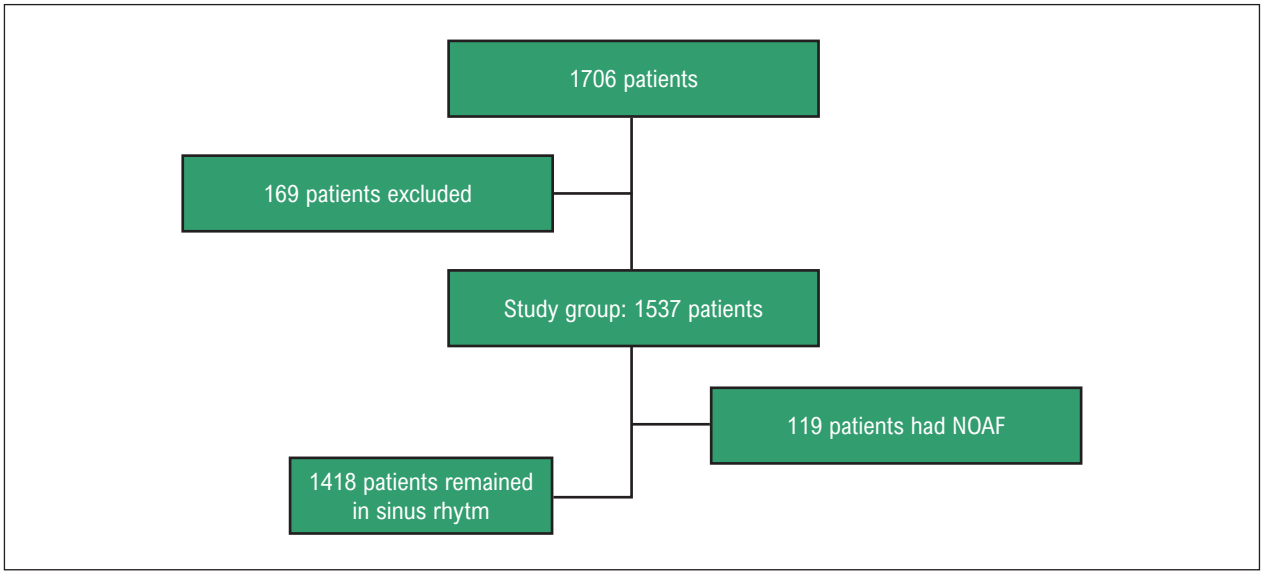

Figure 1 – *Study design and workstream. NOAF: new-onset atrial fibrillation.*

disease or any disability, morbid obesity, or based on the patient's request. Coronary angiography was performed before wiring the coronary arteries, and 70–100 U/kg heparin was administered intracoronary to achieve an activated clotting time of 250–300 s. Furthermore, only culprit lesions underwent revascularization, except for cases of cardiogenic shock or acute myocardial infarction with more than one culprit lesion. Coronary bypass surgery is recommended for patients with critically unprotected left main lesions to prevent revascularization. After rewiring the balloon angioplasty (predilatation – postdilatation), thrombus aspiration and glycoprotein IIb/IIIa inhibitor usage were left at the discretion of experienced operators. After pPCI, the patients were followed up in the coronary intensive care unit. Medical treatment was arranged according to current guidelines.[2] The pPCI was accepted as successful when the residual obstruction was < 30% and Thrombolysis in Myocardial Infarction (TIMI) III flow was achieved.

Definitions, diagnosis, and clinical endpoint

Patient demographics, characteristics, angiographic data, laboratory data, and outcomes were achieved from the medical data recording system. Hypertension (HT) was described as systolic blood pressure higher than 140 mmHg and/or diastolic blood pressure over 90 mmHg or patients already on hypertension treatment. Diabetes mellitus (DM) was defined as having fasting glucose readings >126 mg/dL in two measurements, glycated hemoglobin >6.5 %, or patients previously diagnosed with DM and on treatment. The estimated glomerular filtration rate (eGFR) was measured using the Cockcroft-Gault equation.[11] Peripheral venous blood samples were taken from the patients at their first hospital admission. Biochemical parameters and hemograms were studied from these samples. Within 24 hours of admission, all patients underwent conventional transthoracic echocardiography, and the left ventricle ejection fraction (LVEF) was estimated with Simpson's method. The primary endpoint of this study was the presence of NOAF. Patients with histories of paroxysmal or persistent AF were excluded. AF was defined as the absence of P-waves, fibrillatory activity recorded in the atria, and irregular R – R intervals.

Total cholesterol and albumin levels in the peripheral blood sample, lymphocyte-to-monocyte ratio (LMR), and neutrophil-to-lymphocyte ratio (NLR) were used to calculate the NPS.[8]

Statistical analysis

Statistical analysis was performed using SPSS 23.0 (IBM SPSS Statistics, Version 23.0, Armonk, NY, USA, 2015) software package. Continuous variables were described using mean ± standard deviation or median and interquartile range, according to the normality of the data. Categorical variables were described using absolute and relative frequencies. The distribution characteristics of the variables were assayed using the Kolmogorov–Smirnov test. For comparisons of continuous variables between two groups, the unpaired Student's t-test was used if the data were normally distributed, while the Mann–Whitney U test was applied for non-normally distributed data. Categorical variables were compared using the Chi-Square test. Further stratified analyses were performed on the variables age, e-GFR, hemoglobin, LVEF, NPS, and left atrial dimension based on the significance determined in univariate statistical analysis. A multivariate logistic regression model with a forward elimination method was used to identify variables significantly predicting NOAF development. Results from the logistic regression analysis were reported as odds ratios (ORs) with 95% confidence intervals (CI). Furthermore, receiver operating characteristic (ROC) curve analysis was applied to demonstrate the predictive power of NPS for NOAF development. The area under the curve (AUC) was approved with a 95% confidence interval (CI). $P < 0.05$ was accepted as statistically significant.

Results

In this study, we analyzed 1537 patients who underwent pPCI for STEMI. NOAF was detected in 7.74% (n: 119) of participants and 92.26% (n: 1418) of the patients with RSR. Table 1 demonstrates the descriptive and patient characteristics. The mean age was significantly higher in the NOAF group ($p < 0.001$), and more females were in the NOAF group ($p = 0.002$). However, a higher percentage of patients with RSR were smokers ($p < 0.001$). There were no significant differences between the groups regarding hypertension, DM, previous coronary artery disease, body mass index, or vitality signs. The eGFR was significantly lower in the NOAF group ($p < 0.001$). However, the serum total cholesterol, low-density lipoprotein cholesterol (LDL-C), and triglyceride (Tg) levels were significantly higher in the RSR group ($p < 0.001$ for all). Hemoglobin levels were lower in the NOAF group than in the RSR group ($p < 0.001$). LVEF was lower in the NOAF group; however, left atrium (LA) dimensions were higher in the NOAF group ($p < 0.001$ for both). NPS was significantly higher in the NOAF group (2.53 ± 1.17 vs 2.25 ± 1.10, $p = 0.008$).

Hospital mortality, ventricular arrhythmias, third-degree atrioventricular block, cardiopulmonary resuscitation, and blood transfusion rates were significantly higher in the NOAF group than in the RSR group (Table 2).

Independent NOAF predictors were evaluated using a multivariate logistic regression model, including variables such as age, eGFR, hemoglobin, LVEF, NPS, and LA (Table 3). The findings from the regression analysis confirmed that age, NPS, and left atrial dimension were independent predictors of NOAF development.

ROC analysis was used to determine the predictive power of NPS for NOAF development. Accordingly, the NPS cut-off value of 2.2 predicted NOAF development with a sensitivity and specificity of 83.5% and 72.0%, respectively (AUC: 0.768; 95% CI: 0.511–0.925; $p = 0.013$).

Discussion

This study demonstrated the concomitance of STEMI and NOAF in patients undergoing pPCI and mainly aimed to present the NPS as an effective predictor of NOAF in patients with STEMI who underwent pPCI. In addition to the NPS score, advanced age and increased left atrial dimensions were significant predictors of NOAF. In our cohort of 1537 patients with STEMI, the incidence of NOAF was 7.74%, consistent with the previous literature.[6,12,13] NOAF is observed in approximately 7–8% of STEMI patients undergoing pPCI, with reported rates ranging from 4% to 14% in the literature. The variation in prevalence is influenced by factors such as study design, population demographics, and diagnostic criteria.[4,14] The multicenter randomized Harmonizing Outcomes With Revascularization and Stents in Acute Myocardial Infarction (HORIZONS-AMI) study consisted of 3281 STEMI patients, and NOAF incidence was observed 4.5% (n: 147) after pPCI.[14] NPS showed superior prognostic factors for classical risk factors, such as hemoglobin levels, eGFR, and LVEF. Furthermore, NPS was not inferior to age and LA (Central Illustration).

However, despite the widespread use of pPCI and catheter laboratories, STEMI remains the most fatal subgroup of cardiovascular diseases. Invasive cardiology is developing daily; however, it has a high mortality rate and causes serious morbidity. The pathophysiology of STEMI includes inflammation due to the rupture of fibrin and lipid-containing plaques and adhesion and platelet aggregation, which consequently forms a thrombus.[10]

Inflammation is involved in every stage of thrombus formation and lumen occlusion due to atheroma plaque complications. Cytokine release, acute phase reactants, oxidation end products, neutrophil migration, and lymphocyte apoptosis occur during STEMI due to atherosclerosis and atheroma plaque complications, as well as in the pathophysiology of AF.[15,16] Increased oxidation during STEMI and revascularization injury leads to atrial myofibril damage. Inflammation and oxidative stress affect atrial remodeling. Therefore, agents that increase myocyte remodeling and contain antioxidants are beneficial in avoiding NOAF and other cardiovascular outcomes.[17] Studies have shown that increased inflammation and high monocyte counts increase the frequency of major adverse cardiac events (MACE) in patients with STEMI. In a study examining the incidence of NOAF after STEMI, 346 patients were included, and NOAF was observed in 9.5% of patients. Total oxidative status and oxidative stress index were statistically significantly higher in the NOAF group. Oxidative stress and high sensitivity C-reactive protein (CRP) levels have been reported as independent predictors of NOAF.[18] In a prospective cohort study of 625 patients, the effects of inflammatory markers on outcomes after pPCI were investigated, and only the NLR was shown to be an independent variable to predict the MACE in hospital.[19] In a previous meta-analysis, increased CRP levels were significantly associated with NOAF after acute myocardial infarction. However, one study reported that sex, age, revascularization time, and site of vessel occlusion contributed to elevated CRP levels while predicting NOAF.[20]

Galizia et al. used serum albumin, total cholesterol, NLR, and LMR markers to evaluate nutrition and inflammation and predict prognosis in patients undergoing colorectal cancer surgery.[8] The NPS is an effective prognostic marker for other cancer surgeries and has been included in cardiological studies. Erdogan et al.[10] retrospectively examined 1887 patients with STEMI, and NPS was observed to be a statistically significant independent predictor of all-cause mortality at a median follow-up of 15 months (hazard ratio: 2.49, 95% CI: 1.75–3.5, $p < 0.001$). NPS is more sensitive in predicting the prognosis than other inflammation markers like high sensitivity-CPR, oxidative indices, and isolated NLR or LMR. In addition, the NPS reflects patient nutrition. Recent studies have demonstrated that inflammation is crucial in AF etiology; therefore, we investigated NPS to predict NOAF after pPCI in patients with STEMI. Our study observed that NPS was a significant predictor of NOAF (OR: 1.64, 95% CI: 0.98–2.74, $p = 0.037$).

In addition to NPS, we identified other independent predictors of NOAF, such as age and LA size. LA size can be used to predict and determine the prognosis of AF. In our study, LA was larger in the NOAF group, and multivariate analysis

Table 1 – Demographic findings and patient characteristics

Variables	NOAF (n: 119)	No NOAF at hospitalization (n: 1418)	p-value
Age, years	67.03 ± 13.48	57.84 ± 11.31	<0.001
Female Gender, %(n)	26.9 (32)	16.1 (230)	0.002
Smoking, %(n)	27.7 (33)	50.8 (727)	<0.001
HT, %(n)	50.4 (60)	48.6 (689)	0.324
DM, %(n)	27.7 (33)	27.4 (392)	0.937
CAD, %(n)	28.6 (34)	21.1 (302)	0.057
BMI, kg/m^2	28.70 ± 4.73	27.89 ± 4.83	0.504
Heart Rate, bpm	81.04 ± 28.31	78.63 ± 17.93	0.194
SBP, mmHg	124.03 ± 26.02	128.13 ± 25.24	0.092
DBP, mmHg	75.92 ± 15.35	78.10 ± 14.22	0.113
Pain to needle time, hours	3.13 ± 3.28	2.82 ± 2.41	0.204
Creatinine, mg/dL**	1.04 [0.72 – 1.24]	0.96 [0.69 – 1.03]	0.005
eGFR, ml/min/1.73m^2	70.55 ± 23.33	84.11 ± 21.96	<0.001
HbA1c, %	6.49 ± 1.24	6.59 ± 1.61	0.525
Total – C, mg/dL	171.12 ± 40.90	188.90 ± 42.70	<0.001
LDL – C, mg/dL	114.62 ± 36.88	130.09 ± 38.18	<0.001
HDL – C, mg/dL	40.44 ± 11.31	38.55 ± 10.19	0.055
Triglyceride, mg/dL	126.97 ± 72.53	156.89 ± 96.66	<0.001
Hemoglobin, g/dL	13.44 ± 1.99	14.13 ± 1.75	<0.001
Lym,K/µL	2.46 ± 1.69	2.60 ± 1.54	0.343
Mon, K/µL **	0.79 [0.63 – 0.96]	0.76 [0.45 – 1.1]	0.563
Neu, ,K/µL	8.10 ± 3.92	8.70 ± 4.08	0.119
PLT, ,K/µL	242.65 ± 62.44	259.65 ± 76.02	0.017
EF, %	42.17 ± 9.80	46.78 ± 8.68	<0.001
LA, mm	42.45 ± 5.51	38.11 ± 4.12	<0.001
LVDD, mm	52.21 ± 5.95	49.75 ± 5.36	0.027
IVS, mm	10.91 ± 1.34	10.64 ± 1.19	0.271
NEU/LYM	5.35 ± 5.53	4.84 ± 4.77	0.270
LYM/MON	3.87 ± 2.78	3.97 ± 2.75	0.715
Albumin, g/dL	3.81 ± 0.55	3.92 ± 0.41	0.001
Total protein, K/µL	6.26 ± 0.65	6.45 ± 0.59	0.006
Naples Score	2.53 ± 1.17	2.25 ± 1.10	0.008

*** Data are presented as percentage, mean ± standard deviation, or median (interquartile range). HT: hypertension; DM: diabetes mellitus; CAD: coronary artery disease; BMI: body mass index; SBP: systolic blood pressure; DBP: diastolic blood pressure; eGFR: estimated glomerular filtration rate; C: cholesterol; LDL: light density lipoprotein; HDL: high-density lipoprotein; Lym: lymphocyte; Mon: monocyte; PLT: platelet; EF: ejection fraction; Neu: neutrophile; NOAF: new-onset atrial fibrillation; AF: atrial fibrillation.*

Table 2 – Per or post pPCI complications

Variables	NOAF (n: 119)	No NOAF at hospitalization (n: 1418)	p-value
In-hospital mortality, % (n)	8.4 (10)	4.4 (63)	0.048
VT/VF, % (n)	16.8 (20)	5.2 (75)	<0.001
AV block, % (n)	7.6 (9)	3.4 (48)	0.037
Cardiogenic shock, % (n)	7.6 (9)	4.5 (65)	0.138
CPR, % (n)	4.2 (5)	1.6 (23)	0.041
Major bleeding, % (n)	1.7 (2)	1.1 (16)	0.582
Hemodialysis, % (n)	2.5 (3)	1.0 (14)	0.121
Access site complication, % (n)	5 (6)	2.5 (26)	0.181
Blood transfusion, %(n)	3.4 (4)	0.8 (12)	0.009

pPCI: primary percutaneous coronary intervention; VT: ventricular tachycardia; VF: ventricular fibrillation; AV: atrioventricular; CPR: cardiopulmonary resuscitation.

Table 3 – Multivariate logistic regression analysis to demonstrate the predictors of new-onset atrial fibrillation after pPCI in patients admitted with STEMI

	Exp(B)	CI	p value
Age	1.045	1.019 – 1.071	0.001
eGFR	1.007	0.995 -1.020	0.250
Hemoglobin	1.142	0.982 – 1.328	0.086
LVEF	0.978	0.952 – 1.004	0.102
NPS	1.645	0.984 – 2.748	0.037
Left atrium	2.542	1.488 – 4.342	0.001

Nagelkerke R square: 0.66. LVEF: left ventricular ejection fraction; NPS: Naples prognostic score.

demonstrated LA dimensions as an independent predictor (OR: 2.54, 95% CI: 1.48–4.34, p=0.001). Li et al. analysis of 4713 patients with acute myocardial infarction showed that LA enlargement was associated with an increased NOAF risk.[21] Advanced age is the most well-known risk factor for AF. Recent studies have also demonstrated similar findings regarding age, which is clearly associated with NOAF.[22] Albumin is a negative acute-phase reactant with anti-inflammatory properties and provides information about nutritional status. Furthermore, recent studies have shown that low serum albumin levels are associated with a higher risk of developing NOAF after STEMI.[23] Another study on the inflammatory effects of NOAF demonstrated that the uric acid-to-albumin ratio is an independent predictor of NOAF in patients with STEMI.[24]

Inflammation after myocardial infarction is crucial in cardiovascular complications and pathophysiological repair. During STEMI, many inflammatory processes are triggered, increasing catecholamines, cortisol, and many oxidation products. Therefore, with this pathophysiology, the risk of developing NOAF increases during the occlusion and revascularization of the coronary arteries. Thus, determining the level of inflammation in patients with STEMI and deciding to use drugs that can reduce inflammation, such as colchicine, may reduce the frequency of NOAF. To our knowledge, this is the first study to investigate the relationship between NPS and NOAF after pPCI in patients with STEMI.

Limitations

This study has some limitations. Notably, several patients were enrolled in this study; however, some data could not be obtained due to its retrospective nature. In addition, there was a lack of clinical follow-up of patients in this study. The severity of coronary artery disease, reporting of flow rates after pPCI, and reporting AF attacks separately as symptomatic/asymptomatic and paroxysmal/permanent could have also increased the scientific value of this study. Furthermore, owing to the retrospective nature of this study, reliable data on drug use could not be obtained. Therefore, a prospective multicenter study with many patients can provide valuable data from a scientific perspective.

Conclusion

STEMI is a cardiovascular emergency that requires urgent intervention. NOAF is a frequent complication of STEMI and has poor outcomes; therefore, predicting the risk of NOAF using predictive markers is crucial. NOAF incidence is approximately 7–8%. NPS is an essential marker that provide information on inflammation and nutrition. Recent studies have demonstrated an obvious association between NOAF and inflammation. In patients with STEMI undergoing pPCI, the NPS was an independent predictor of NOAF, in addition to classical factors such as age and left atrial dimensions. This score, mostly related to an inflammatory burden, may help to predict NOAF incidence in patients after STEMI and select better potential therapies aimed at abating inflammation after myocardial infarction.

Author Contributions

Conception and design of the research: Oksen D, Oktay V, Abaci O; Acquisition of data and Analysis and interpretation of the data: Arslan S, Gecit MH; Statistical analysis: Oksen D, Gecit MH, Abaci O; Obtaining financing: Oksen D, Gecit MH; Writing of the manuscript: Oksen D, Arslan S, Tekin EE, Oktay V; Critical revision of the manuscript for content: Oksen D, Tekin EE, Oktay V.

Potential conflict of interest

No potential conflict of interest relevant to this article was reported.

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Study association

This article is part of the thesis of doctoral submitted by Dogac Oksen, from Istanbul University Cerrahpasa Institute of Cardiology.

Ethics approval and consent to participate

This study was approved by the Ethics Committee of the Istanbul University Cerrahpasa Institute of Cardiology. under the protocol number 2024/195. All the procedures in this study were in accordance with the 1975 Helsinki Declaration, updated in 2013. Informed consent was obtained from all participants included in the study.

Use of Artificial Intelligence

The authors did not use any artificial intelligence tools in the development of this work.

Data Availability

All datasets supporting the results of this study are available upon request.

References

1. Okşen D, Sarılar M, Demirci G, Haberal İ, Abaci O. In-Hospital and Long-Term Outcomes of ST-Segment Elevation Myocardial Infarction Patients Undergoing Primary Percutaneous Coronary Intervention. Koşuyolu Heart J. 2022;25(1):23-32. doi: 10.51645/khj.2021.m54.
2. Byrne RA, Rossello X, Coughlan JJ, Barbato E, Berry C, Chieffo A, et al. 2023 ESC Guidelines for the Management of Acute Coronary Syndromes. Eur Heart J Acute Cardiovasc Care. 2024;13(1):55-161. doi: 10.1093/ehjacc/zuad107.
3. Gaudino M, Di Franco A, Rong LQ, Piccini J, Mack M. Postoperative Atrial Fibrillation: From Mechanisms to Treatment. Eur Heart J. 2023;44(12):1020-39. doi: 10.1093/eurheartj/ehad019.
4. Dal Zotto B, Barbieri L, Tumminello G, Saviano M, Gentile D, Lucreziotti S, et al. New Onset Atrial Fibrillation in STEMI Patients: Main Prognostic Factors and Clinical Outcome. Diagnostics. 2023;13(4):613. doi: 10.3390/diagnostics13040613.
5. Ulus T, Isgandarov K, Yilmaz AS, Vasi I, Moghanchızadeh SH, Mutlu F. Predictors of New-Onset Atrial Fibrillation in Elderly Patients with Acute Coronary Syndrome Undergoing Percutaneous Coronary Intervention. Aging Clin Exp Res. 2018;30(12):1475-82. doi: 10.1007/s40520-018-0926-9.
6. Arslan Ş, Batıt S, Kılıçarslan O, Doğan Ö, Yumuk MT, Arslan Ş, et al. Incidence of Atrial Fibrillation and Its Effects on Long-Term Follow-Up Outcomes in Patients Undergoing Primary Percutaneous Coronary Intervention for ST-Elevation Myocardial Infarction. Anatol J Cardiol. 2021;25(9):609-16. doi: 10.5152/AnatolJCardiol.2021.26020.
7. Guo Y, Lip GY, Apostolakis S. Inflammation in Atrial Fibrillation. J Am Coll Cardiol. 2012;60(22):2263-70. doi: 10.1016/j.jacc.2012.04.063.
8. Galizia G, Lieto E, Auricchio A, Cardella F, Mabilia A, Podzemny V, et al. Naples Prognostic Score, Based on Nutritional and Inflammatory Status, is an Independent Predictor of Long-Term Outcome in Patients Undergoing Surgery for Colorectal Cancer. Dis Colon Rectum. 2017;60(12):1273-84. doi: 10.1097/DCR.0000000000000961.
9. Birdal O, Pay L, Aksakal E, Yumurtaş AÇ, Çinier G, Yücel E, et al. Naples Prognostic Score and Prediction of Left Ventricular Ejection Fraction in STEMI Patients. Angiology. 2024;75(1):36-43. doi: 10.1177/00033197231161903.
10. Erdogan A, Genc O, Ozkan E, Goksu MM, Ibisoglu E, Bilen MN, et al. Impact of Naples Prognostic Score at Admission on In-Hospital and Follow-Up Outcomes among Patients with ST-Segment Elevation Myocardial Infarction. Angiology. 2023;74(10):970-80. doi: 10.1177/00033197231151559.
11. Cockcroft DW, Gault MH. Prediction of Creatinine Clearance from Serum Creatinine. Nephron. 1976;16(1):31-41. doi: 10.1159/000180580.
12. Mazzone A, Scalese M, Paradossi U, Del Turco S, Botto N, De Caterina A, et al. Development and Validation of a Risk Stratification Score for New-Onset Atrial Fibrillation in STEMI Patients Undergoing Primary Percutaneous Coronary Intervention. Int J Clin Pract. 2018;72(4):e13087. doi: 10.1111/ijcp.13087.
13. Rencuzogullari I, Çağdaş M, Karakoyun S, Yesin M, Gürsoy MO, Artaç İ, et al. Propensity Score Matching Analysis of the Impact of Syntax Score and Syntax Score II on New Onset Atrial Fibrillation Development in Patients with ST Segment Elevation Myocardial Infarction. Ann Noninvasive Electrocardiol. 2018;23(2):e12504. doi: 10.1111/anec.12504.
14. Rene AG, Généreux P, Ezekowitz M, Kirtane AJ, Xu K, Mehran R, 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):236-42. doi: 10.1016/j.amjcard.2013.09.016.

15. Mourouzis K, Oikonomou E, Siasos G, Tsalamadris S, Vogiatzi G, Antonopoulos A, et al. Pro-Inflammatory Cytokines in Acute Coronary Syndromes. Curr Pharm Des. 2020;26(36):4624-47. doi: 10.2174/1381612826666200413082353.

16. Karam BS, Chavez-Moreno A, Koh W, Akar JG, Akar FG. Oxidative Stress and Inflammation as Central Mediators of Atrial Fibrillation in Obesity and Diabetes. Cardiovasc Diabetol. 2017;16(1):120. doi: 10.1186/s12933-017-0604-9.

17. Ozaydin M, Peker O, Erdogan D, Akcay S, Yucel H, Icli A, et al. Oxidative Status, Inflammation, and Postoperative Atrial Fibrillation with Metoprolol vs Carvedilol or Carvedilol Plus N-Acetyl Cysteine Treatment. Clin Cardiol. 2014;37(5):300-6. doi: 10.1002/clc.22249.

18. Bas HA, Aksoy F, Icli A, Varol E, Dogan A, Erdogan D, et al. The Association of Plasma Oxidative Status and Inflammation with the Development of Atrial Fibrillation in Patients Presenting with ST Elevation Myocardial Infarction. Scand J Clin Lab Invest. 2017;77(2):77-82. doi: 10.1080/00365513.2016.1244857.

19. Machado GP, Araújo GN, Carpes CK, Lech M, Mariani S, Valle FH, et al. Comparison of Neutrophil-to-Lymphocyte Ratio and Mean Platelet Volume in the Prediction of Adverse Events after Primary Percutaneous Coronary Intervention in Patients with ST-Elevation Myocardial Infarction. Atherosclerosis. 2018;274:212-7. doi: 10.1016/j.atherosclerosis.2018.05.022.

20. Ren Y, Zeng RX, Li JJ, Guo LH, He DY, Li Y, et al. Relation of C-Reactive Protein and New-Onset Atrial Fibrillation in Patients with Acute Myocardial Infarction: A Systematic Review and Meta-Analysis. Int J Cardiol. 2015;190:268-70. doi: 10.1016/j.ijcard.2015.04.152.

21. Li Z, Liu Q, Liu F, Hidru TH, Yang Y, Wang S, et al. Atrial Cardiomyopathy Markers and New-Onset Atrial Fibrillation Risk in Patients with Acute Myocardial Infarction. Eur J Intern Med. 2022;102:72-9. doi: 10.1016/j.ejim.2022.04.019.

22. He J, Yang Y, Zhang G, Lu XH. Clinical Risk Factors for New-Onset Atrial Fibrillation in Acute Myocardial Infarction: A Systematic Review and Meta-Analysis. Medicine. 2019;98(26):e15960. doi: 10.1097/MD.0000000000015960.

23. Gao Z, Bao J, Wu L, Shen K, Yan Q, Ye L, et al. A Predictive Model of New-Onset Atrial Fibrillation after Percutaneous Coronary Intervention in Acute Myocardial Infarction Based on the Lymphocyte to C-Reactive Protein Ratio. J Inflamm Res. 2023;16:6123-37. doi: 10.2147/JIR.S443319.

24. Selçuk M, Çınar T, Şaylık F, Akbulut T, Asal S, Çiçek V, et al. Predictive Value of Uric Acid/Albumin Ratio for the Prediction of New-Onset Atrial Fibrillation in Patients with ST-Elevation Myocardial Infarction. Rev Invest Clin. 2022;74(3):156-64. doi: 10.24875/RIC.22000072.