ARAŞTIRMA MAKALESİ / *RESEARCH ARTICLE*

Inflammatory Prognostic Index Predicts New-Onset Atrial Fibrillation in Patients After Primary Percutaneous Coronary Intervention in Patients with ST-Segment Elevation Myocardial Infarction

Enflamatuvar Prognostik Endeks, ST-Segment Yükselmeli Miyokard Enfarktüsü Olan Hastalarda Primer Perkütan Koroner Girişim Sonrası Yeni Başlangıçlı Atriyal Fibrilasyonu Öngörür

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ABSTRACT

Aim: *New-onset atrial fibrillation (NOAF) is an independent predictor of mortality and a strong indicator of poor prognosis following ST-segment elevation myocardial infarction (STEMI). The Inflammatory Prognostic Index (IPI) is clinically significant in predicting patient outcomes and serves as a novel inflammatory prognostic marker based on C-reactive protein (CRP), the neutrophil-to-lymphocyte ratio (NLR), and serum albumin levels. This study aimed to investigate the relationship between the IPI and NOAF in patients with STEMI who underwent primary percutaneous coronary intervention (pPCI).*

Material and Methods: *The population for this retrospective study consisted of 1.132 consecutive patients diagnosed with STEMI who underwent pPCI. Out of these, 946 patients were included in the study sample and were divided into two groups based on whether they developed NOAF or not.*

Results: *The study's primary outcome, that is, IPI was significantly higher in patients with NOAF than in those with No-AF (42.15 (17.6–81.7) vs. 12.77 (5.72–27.01), p<0.001) Univariate logistic regression analysis revealed significant correlations between NOAF; IPI, LVEF and age. Further analysis of these variables using the multivariate logistic regression analysis indicated that IPI (Odds Ratio [OR]: 2.026, 95% confidence interval [CI]: 1.081–3.799; p=0.028), LVEF and age were independent predictors for the development NOAF. Inflammatory prognostic index optimal cut-off value of >17.5 predicted NOAF with 76% sensitivity and 62.7% specificity (AUC: 0.740 [95% CI: 0.711–0.768, p<0.0001] (P <0.0001).*

Conclusions: *This study finds that the IPI independently predicts NOAF in STEMI patients treated with pPCI.*

Key words: *inflammatory prognostic index; ST-segment elevation myocardial infarction; atrial fibrillation*

ÖZET

Amaç: *Yeni başlangıçlı atriyal fibrilasyon (NOAF), ölümün bağımsız bir öngörücüsü ve ST-segment yükselmeli miyokard enfarktüsü (STEMI) sonrası kötü prognozun güçlü bir göstergesidir. Enflamatuvar Prognostik Endeks (IPI), hasta prognozunu tahmin etmede klinik öneme sahiptir ve C-reaktif protein (CRP), nötrofil-lenfosit oranı (NLR) ve serum albümin seviyelerine dayalı yeni bir enflamatuvar prognostik belirteç görevi görür. Bu çalışmanın amacı, STEMI geçiren ve primer perkütan koroner girişim (pPCI) geçiren hastalarda IPI ve NOAF arasındaki ilişkiyi araştırmaktır.*

Gereç ve Yöntem: *Bu retrospektif çalışmanın popülasyonu, pPCI geçiren STEMI tanısı almış 1132 ardışık hastadan oluşuyordu. Bunlardan 946 hasta çalışma örneğine dâhil edildi ve NOAF geliştirip geliştirmediklerine göre iki gruba ayrıldı.*

Bulgular: *Çalışmanın birincil çıktısı, yani IPI, NOAF'lı hastalarda AF'li olmayanlara kıyasla anlamlı derecede yüksekti (42,15(17,6–81,7) – 12,77 (5,72–27,01), p<0,001). Tek değişkenli lojistik regresyon analizi, NOAF, IPI, LVEF ve yaş arasında anlamlı korelasyonlar olduğunu ortaya koydu. Bu değişkenlerin çok değişkenli lojistik regresyon analizi kullanılarak daha ileri analizi, IPI'nin (Olasılık Oranı [OR]: 2,026, %95 güven aralığı [GA]: 1,081–3,799; p=0,028), LVEF'in ve yaşın NOAF gelişimi için bağımsız öngörücüler olduğunu gösterdi. IPI'nin >17,5'lik optimum kesme değeri, %76 duyarlılık ve %62,7 özgüllükle yeni başlangıçlı AF'yi öngördü (AUC: 0.740 [95% CI: 0,711–0,768, p<0,0001] (P <0,0001).*

Sonuç: *Bu çalışmanın bulguları, IPI'nin pPCI ile tedavi edilen STEMI hastalarında NOAF'ın bağımsız bir öngörücüsü olduğunu göstermektedir.*

Anahtar kelimeler: *enflamatuvar prognostik endeks, ST-segment yükselmeli miyokard enfarktüsü, atriyal fibrilasyon*

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Introduction

Research indicates that significant inflammation is a major factor in the onset of new atrial fibrillation (NOAF) among individuals experiencing acute ST-elevation myocardial infarction (STEMI)[1,2]. The occurrence of NOAF, a common outcome of acute STEMI, varies between 2.3% and 21%[3]. NOAF is a key predictor of mortality and strongly indicates a poor prognosis after STEMI[4,5]. Inflammation plays a significant role in developing and maintaining NOAF through myocyte necrosis, fibrosis, and infiltrating inflammatory markers[6]. Studies have shown that individuals with various subtypes of atrial fibrillation (AF) exhibit higher levels of certain inflammatory markers, including Interleukin-6 (IL-6) and High-Sensitivity C-Reactive Protein (Hs-CRP), compared to individuals in sinus rhythm[7].

A novel hematological biomarker that indicates a patient's inflammatory and immunological condition is the inflammatory prognostic index (IPI), which is calculated by combining the levels of CRP, neutrophil to lymphocyte ratio (NLR), and serum albumin (ALB) (IPI=CRP×NLR/ALB)[8]. Research has shown that this novel biomarker provides valuable insights into the prognosis of cancer patients, with elevated levels associated with worse outcomes. However, to our knowledge, no research has examined the impact of IPI on the likelihood of patients with STEMI developing NOAF. Therefore, we investigated the potential of IPI to predict NOAF in patients with STEMI who underwent primary percutaneous coronary intervention (pPCI).

Material and Methods

Population and Sample

This retrospective study included a population of 1.132 consecutive patients who were admitted to the emergency department of a tertiary heart center within 12 hours of the onset of symptoms and underwent pPCI between January 2019 and December 2023. Patients were excluded from the study if they were undergoing chemotherapy, had a history of concomitant inflammatory disorders, had received glucocorticoid therapy within the past three months, were pregnant or nursing, were in cardiogenic shock, had undergone cardiopulmonary resuscitation due to cardiopulmonary arrest, were receiving treatment with a thrombolytic agent, or were referred for emergency coronary artery bypass grafting. Additional exclusions included those with acute or chronic inflammatory diseases, neoplastic hematologic disorders, immunosuppressive medication use, major trauma or surgery within the last three months, missing blood cell count data, severe liver or kidney dysfunction, or those lost to follow-up. Ultimately, a total of 946 patients were included in the study sample. Among these patients, 892 did not present with AF, while 54 were identified as having NOAF. Patients were categorized into these two groups. Baseline demographic and clinical characteristics were gathered from the hospital's electronic database, and IPI was calculated for each patient. The study protocol received approval from the hospital's ethics and research committee and was conducted by the ethical principles outlined in the Declaration of Helsinki.

Laboratory Tests

The data included laboratory information such as neutrophil, lymphocyte, platelet, and hemoglobin counts, as well as biochemical parameters like serum albumin and C-reactive protein (CRP). The Inflammatory Prognostic Index (IPI) was calculated using the following formula: CRP × Neutrophil-Lymphocyte Ratio (NLR) / serum albumin[8].

Coronary Angiography and Percutaneous Coronary Intervention

All patients underwent coronary angiography via a femoral artery within 90 minutes of admission. They received 300 mg acetylsalicylic acid and a 180 mg oral loading dose of ticagrelor, 60 mg oral loading dose of prasugrel or 300 to 600 mg oral loading dose of clopidogrel, if not suitable for ticagrelor, on admission, as recommended in the recent European Society of Cardiology myocardial revascularization guidelines[9]. Standard intravenous bolus unfractionated heparin (70–100 U/kg) and additional doses were given to achieve an activated clotting time of >250 seconds before coronary intervention[9]. Stenting of the infarct-related artery with a drug-eluting stent was completed in suitable patients immediately after the coronary angiography.

Definitions

ST-segment elevation myocardial infarction (STEMI) was diagnosed based on the presence of typical chest pain lasting >30 min and/or other angina-equivalent symptoms, e.g., fainting, shortness of breath, dizziness, and sweating, with at least one of the following

electrocardiographic (ECG) findings, i. e., at least two contiguous leads with ST-segment elevation of ≥2.5 mm in men <40 years, ≥2 mm in men ≥40 years, or ≥1.5 mm in women in leads V2-V3 and/or ≥1 mm in other leads [in the absence of left ventricular hypertrophy or left bundle branch block. In patients with inferior myocardial infarction (MI), right precordial leads (V3R and V4R) should be recorded for ST-segment elevation to determine concurrent right ventricular infarction. Similarly, ST-segment depression in leads V1-V3 signals myocardial ischemia, particularly when the terminal T-wave is positive (ST-segment elevation equivalent), and confirmation by simultaneous ST-segment elevation ≥0.5 mm in leads V7-V9 could be regarded as a way of identifying posterior acute myocardial infarction (AMI)[10].

To diagnose AF, ECG recordings obtained during hospital stays were analyzed. The evaluation focused on various parameters, such as irregular RR intervals, the presence of fibrillation waves, and the absence of P waves. Additionally, biochemical markers and morning venous blood samples collected during these hospital stays were reviewed afterward.

Statistical Analysis

IBM Statistical Package for Social Sciences (SPSS, Statistical Product and Service Solutions for Windows) program version 22.0 (IBM Corp., Armonk, NY, U. S., 2013) software package was used for statistical analyses. The descriptive statistics obtained from the collected data were expressed as mean ± standard deviation in case of continuous variables determined to conform to the normal distribution, as median with 0.25 and 0.75 quantiles in case of continuous variables determined not to conform to the normal distribution, and as percentage values in the case of categorical variables. The t-test or Mann-Whitney U test was used to compare continuous variables between the groups, whereas Fisher's exact or chi-square test was used to compare categorical variables between the groups. Univariate Cox proportional hazards analyses were conducted for all clinically relevant variables that can potentially predict NOAF. Multivariate Cox regression analysis of variables found to be significant in univariate analyses, with stepwise backward conditional elimination, was performed to determine independent predictors of NOAF ($p < 0.05$). The significance level of selected variables was deemed 0.05 (a ¼ 0.05), and of the significant variables was deemed 0.10 (a ¼ 0.10). The IPI was analyzed using the multivariate Cox proportional hazards model as a continuous variable. The receiver operating characteristic (ROC) analysis was used to determine the optimal IPI score cut-off value for predicting NOAF.

Results

The baseline demographic, laboratory, and angiographic characteristics of the patients are presented in Table 1. The study included a sample of 946 patients with STEMI who underwent pPCI. The mean age of the participants was 56 years (±12 years), and 156 patients (16.6%) were female. Among the 946 patients, 892 were classified as No-AF patients, while 54 were categorized as having NOAF. There were no significant differences in mean systolic blood pressure (SBP), platelet count, creatinine levels, or the presence of an infarct-related left anterior descending coronary artery (LAD) between the No-AF and NOAF groups. However, patients with NOAF were older and had a higher proportion of females compared to those No-AF. The prevalence of hypertension (HT) and diabetes mellitus (DM) was significantly greater in the NOAF group, while smoking was more common among No-AF patients. The number of patients with a Killip class of 2 to 4 upon admission was also significantly higher in those with NOAF. Regarding laboratory findings, hemoglobin (Hgb), albumin, lymphocyte count, and left ventricular ejection fraction (LVEF) were all lower in NOAF patients. Conversely, white blood cell (WBC) counts, neutrophil counts and CRP levels were higher in this group. Additionally, heart rate, peak creatine kinase-myocardial band (CK-MB), glucose levels, time-to-treatment, and the SYNTAX (SYNergy between percutaneous coronary intervention with TAXus and cardiac surgery) score were significantly elevated in patients with NOAF (see Table 1).

In-Hospital Outcomes

The study's primary outcome, that is, IPI was significantly higher in patients with NOAF than in those with No-AF (42.15 (17.6–81.7) vs. 12.77 (5.72–27.01), $p<0.001$) (Table 1).

Independent Predictors of NOAF

Univariate logistic regression analysis revealed significant correlations between NOAF; IPI, LVEF and age (Table 2). Further analysis of these variables using the multivariate logistic regression analysis indicated that IPI (Odds Ratio [OR]: 2.026, 95% confidence interval [CI]: 1.081–3.799; p=0.028), LVEF (OR: 0.888, 95%

Table 1. *The baseline demographic, laboratory and angiographic characteristics of the patients with p-values*

	No AF (n: 892)		New-onset AF (n: 54)		All Patients (n: 946)		p-value
Age (years)	56	±12	64	±13	56	±12	**<0.001**
Patients with DM, n (%)	201	22.5	20	37	221	23.4	**0.015**
Gender, n (%) (Female)	142	15.9	15	27.8	157	16.6	**0.023**
Patients with HT, n (%)	351	39.3	35	64.8	386	40.8	**<0.001**
Active smokers, n (%)	518	58.1	20	37	538	56.9	**0.002**
Patients with Killip class >1 at admission, n (%)	123	13.8	23	42.6	146	15.4	**<0.001**
SBP (mmHg)	131	±31	131	±40	131	±31	0.734
Heart rate (bpm)	76	±16	87	±17	77	±16	**<0.001**
Hemoglobin level (g/L)	13.7	±1.74	12.8	±2.1	13.6	±1.77	**0.009**
WBC count (10^3/ml)	12.06	±3.6	13.6	±4	12.1	±3.6	**0.002**
Platelet count (10^3/ml)	259	±68	251	±71	258	±68	0.474
Neutrophil count (10^3/ml)	9.3	±3.4	11.01	±3.5	9.4	±3.4	**<0.001**
Lymphocyte count (10^3/ml)	0.17	(0.125-0.24)	0.135	(0.11-0.21)	0.170	(0.12-0.239)	**0.006**
Glucose level (mg/dL)	125	(103-163)	145	(112-210)	126	(104-168)	**0.025**
CRP (mg/dL)	9.6	(5.3-16.7)	16.6	(12–32.1)	9.9	(5.6-17.1)	**<0.001**
Albumin level (g/L)	36.89	±4.25	35.19	±5.76	36.79	±4.36	**0.018**
Creatine level (mg/dL)	0.92	±0.26	1.03	±0.39	0.93	±0.27	0.135
Peak CK-MB level (ng/Ml)	165	(98-298)	365	(210-443)	171	(100-311)	**<0.001**
LVEF (%)	47.7	±7.98	38.8	±8.2	46.7	±8.2	**<0.001**
Time-to-treatment (min.) or Total ischemic time	175	(110-265)	209	(128-325)	178	(112-267)	**0.001**
Patients with infarct-related LAD coronary artery , n (%)	453	50.8	34	63	487	51.5	0.082
SYNTAX score	16.6	±4.6	18.4	±3.8	16.7	±4.5	**<0.001**
IPI	12.77	(5.72-27.01)	42.15	(17.6-81.7)	13.29	(5.97-28.87)	**<0.001**

AF: atrial fibrillation, p: probability statistic, DM: diabetes mellitus, HT: hypertension, SBP: systolic blood pressure, bpm: beats per minute, WBC: white blood cell, CRP: C-reactive protein, CK-MB: creatine kinase-myocardial band, min: minute, LVEF: left ventricular ejection fraction, LAD: left anterior descending, SYNTAX: SYNergy between percutaneous coronary intervention with TAXus and cardiac surgery, IPI: Inflammatory prognostic index.

Table 2. *Results of the univariate and multivariate analyses of the variables in terms of their prognostic value in predicting new-onset AF in patients with STEMI treated with pPCI*

	Univariate Analysis			Multivariate Analysis		
	Univariate OR, 95% CI		p-value	Multivariate OR, 95% CI		p-value
IPI	2.007	(1.038-4.011)	**<0.001**	2.026	(1.081-3.799)	**0.028**
LVEF	0.884	(0.853-0.916)	**<0.001**	0.888	(0.856-0.922)	**0.001**
Age	1.052	(1.029-1.076)	**<0.001**	1.034	(1.009-1.059)	**0.008**

AF: atrial fibrillation, STEMI: ST-segment elevation myocardial infarction, pPCI: primary percutaneous coronary intervention, OR: odds ratio, CI: confidence interval, p: probability statistic, LVEF: left ventricular ejection fraction, IPI: Inflammatory prognostic index.

CI: 0.856–0.922; p=0.001) and age (OR: 1.034, 95% CI: 1.009–1.059; p=0.008) were independent predictors for the development NOAF (Table 2).

Inflammatory prognostic index optimal cut-off value of >17.5 predicted new-onset AF with 76% sensitivity and 62.7% specificity (AUC: 0.740 [95% CI: 0.711–0.768, p<0.0001] (P <0.0001) (Figure 1).

Discussion

This study's findings indicate that the IPI is an independent predictor of NOAF in patients with STEMI treated with pPCI. To the best of the authors' knowledge, this is the first study to report the association between NOAF and IPI in patients with STEMI undergoing pPCI.

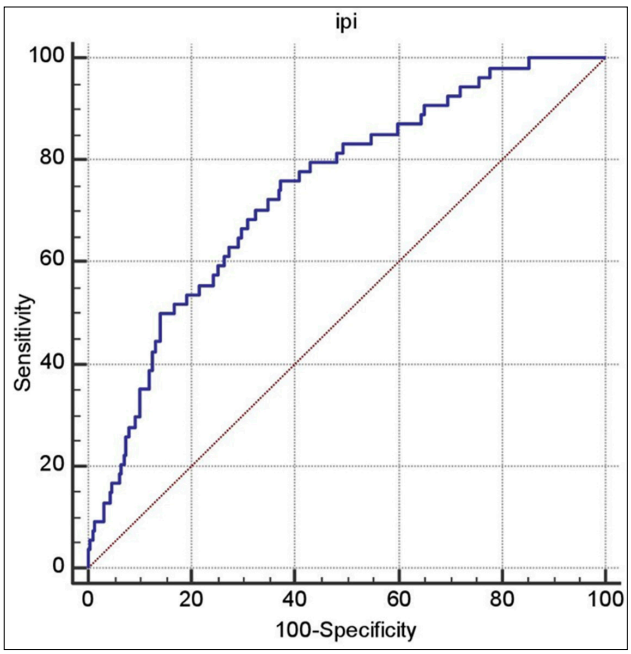

Figure 1. *ROC curve analysis of IPI index to predict new-onset atrial fibrillation in STEMI.*

Patients who receive thrombolysis or pPCI have a worse prognosis and higher death rates in the short, mid, and long term if they experience any additional AF events while hospitalized for acute STEMI[4,11]. The development of NOAF in individuals with STEMI has been linked to several clinical factors, consistent with earlier research[3,4]. We discovered that several factors are associated with an increased risk of NOAF in patients with STEMI. These factors include older age, reduced left ventricular ejection fraction (LVEF), and increased infarct size. The causes of NOAF are complex and involve several triggers. These include atrial ischemia, atrial dilation, increased sympathetic nervous system activity, and reduced vagal tone. Arrhythmic events can be initiated or worsened by acute hypoxia, hypokalemia, systemic and localized inflammation, and hormonal changes. Ultimately, this series of events leads to structural and electrical remodeling of the atria, resulting in AF[12]. After it develops, AF can lead to worsened ischemia, increased oxygen demand, and a higher heart rate. In addition to factors such as age, gender, obesity, smoking, diabetes mellitus, renal failure, chronic obstructive pulmonary disease, and history of arrhythmias, other clinical parameters that are recognized as risk factors for the onset of NOAF include elevated heart rate, increased size and volume of the left atrium, decreased LVEF, and HT[13]. In line with existing literature, our study found that the group that developed NOAF was older and had a higher prevalence of HT and DM. This group also exhibited higher heart rates and reduced LVEF. Notably, we found that smoking was more prevalent in the group without atrial fibrillation compared to the NOAF group.

The onset of new-onset atrial fibrillation (NOAF) and the rupture of atherosclerotic plaques, which can worsen STEMI, are significantly influenced by inflammation. Research has examined the ability of various inflammatory biomarkers to predict NOAF in patients with acute coronary syndrome (ACS). In individuals with STEMI, factors such as the neutrophil-to-lymphocyte ratio, systemic immune-inflammatory index, and prognostic nutritional index have been identified as independent predictors associated with NOAF[14–16]. Furthermore, while the CAR has not been documented in NOAF associated with STEMI, its relationship with postoperative atrial fibrillation following coronary artery bypass graft (CABG) has been reported[17].

The Inflammatory Prognostic Index (IPI) is a novel measure of inflammation that holds clinical significance for assessing prognosis. It is based on C-reactive protein (CRP), neutrophil-to-lymphocyte ratio (NLR), and serum albumin levels. Dirican et al. initially developed the IPI using hematological and biochemical markers to evaluate the prognosis of patients with non-small cell lung cancer[8]. Inflammatory prognostic index (IPI) is a simple, affordable, accessible, and noninvasive metric for predicting prognosis. Numerous studies on cancer patients have utilized it as a significant new measure to assess survival[8,18]. To our knowledge, no published research has examined the connection between IPI and NOAF. This study is the first to demonstrate a correlation between NOAF and IPI in STEMI patients undergoing pPCI.

Advanced age is recognized as an independent risk factor for AF[19]. Studies indicate elderly individuals are more likely to develop NOAF following a STEMI[20]. Our recent findings confirm that advanced age has significant prognostic value and is an independent risk factor for NOAF in STEMI patients undergoing initial pPCI. Furthermore, research by Asanin et al. demonstrated that patients who experienced AF after an acute myocardial infarction had a significantly lower LVEF compared to those who did not develop AF[21]. Our study also found that decreased LVEF is an independent risk factor for NOAF in the STEMI patient cohort.

Limitations of the Study

This study had several limitations. First, it was a single-center, retrospective study. Second, we only measured CRP, lymphocyte, neutrophil, and albumin levels at admission; we did not investigate how these levels changed over time or how those changes might have influenced the outcomes of patients with STEMI. Lastly, there is a need for comprehensive prospective studies to evaluate the impact of anti-CRP and other anti-inflammatory interventions in larger groups of STEMI patients, as the effectiveness of these treatments in improving outcomes for STEMI patients remains uncertain.

Conclusion

This study is the first to explore the association between the development of NOAF and the IPI in patients with STEMI who are treated with pPCI. To our knowledge, no prior research has investigated the relationship between IPI and NOAF. The increase in IPI may serve as an effective clinical indicator for assessing the risk of NOAF, exhibiting good sensitivity and specificity. The ability to predict the risk of NOAF in STEMI patients can be significantly enhanced by validating our findings through multicenter, prospective trials involving larger sample sizes.

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Conflict of Interest Statement

The authors have no conflicts of interest to declare.

Ethical Considerations

The study protocol was approved by the local ethics committee.

Informed Consent

Written informed consent was obtained from all patients.

Data Availability Statement

The authors confirm that the data supporting this study's findings are available within the article and that they have no additional data to share.

Contributorship

All authors contributed to the research's planning, conducting, and reporting. All contributors are responsible for the overall content.

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