Assessment of the Relationship Between C-Reactive Protein to Albumin Ratio and New-Onset Atrial Fibrillation in Patients with ST Elevation Myocardial Infarction

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ABSTRACT

Introduction: Previous studies reported that inflammatory markers are associated with the development of new-onset atrial fibrillation (NOAF) in patients with coronary artery disease. However, the predictive value of serum C-reactive protein (CRP) to serum albumin ratio (CAR) for the development of NOAF in patients with ST elevation myocardial infarction (STEMI) has not been investigated yet. Hence, the aim of the present study was to evaluate the potential utility of the CAR in predicting NOAF in patients with STEMI who underwent primary percutaneous coronary intervention (pPCI).

Patients and Methods: The present study was a retrospective analysis of the data related to 1153 patients with STEMI who underwent pPCI. CRP levels were measured according to the immunoturbidimetric method, and serum albumin levels were analyzed by the bromocresol green method. The CAR was defined as the serum CRP level divided by the serum albumin level.

Results: The incidence of NOAF during in-hospital stay was 5.2% (n= 62 patients). Patients with NOAF had higher CAR values than those without NOAF. Multivariate logistic regression analyses revealed that elevated CAR value was an independent predictor of NOAF (odds ratio 3.280, 95% confidence interval 1.564-6.878; p= 0.002). Furthermore, comparison of receiver operating characteristic curves yielded that the predictive performance of CAR was higher than CRP and albumin alone, respectively.

Conclusion: In the present study, we observed that elevated CAR values were independently associated with NOAF development in patients with STEMI treated with pPCI.

Key Words: C-reactive protein to albumin ratio; new-onset atrial fibrillation; ST elevation myocardial infarction

ST Yükselmeli Miyokart İnfarktüslü Hastalarda C-Reaktif Protein/Albumin Oranı ile Yeni Başlangıçlı Atriyal Fibrilasyon Arasındaki İlişkinin Değerlendirilmesi

ÖZET

Giriş: Daha önceden yapılan çalışmalar, inflamatuvar belirteçlerin koroner arter hastalığı olan hastalarda yeni başlayan atriyal fibrilasyon (AF)'un gelişimi ile ilişkili olduğunu göstermiştir. Bununla birlikte, ST yükselmeli miyokart infarktüslü (STEMİ) hastalarda AF gelişimi için serum C-reaktif protein (CRP)'in serum albumin oranı (CAR)'na göre prediktif değeri henüz araştırılmamıştır. Bu nedenle, bu çalışmada, primer perkütan koroner girişim (pPKG) yapılan STEMİ hastalarında AF'ı öngörmedeki değerini değerlendirmeyi amaçladık.

Hastalar ve Yöntem: Bu çalışma, pPKG uygulanan 1153 STEMİ hastasına ilişkin verilerin geriye yönelik analiz edilmesi ile yapıldı. CRP düzeyleri immünoturbidimetrik yönteme göre ölçüldü ve serum albumin düzeyleri bromkresol yeşili yöntemiyle analiz edildi. CAR, CRP değerlerinin serum albumin oranı olarak tanımlandı.

Bulgular: Hastanede kalış sırasında AF insidansı %5.2 (n= 62 hasta) idi. AF olan hastalar, AF olmayanlara kıyasla yüksek CAR değerlerine sahipti. Çok değişkenli lojistik regresyon analizleri, yüksek CAR değerinin AF'ın bağımsız bir belirleyicisi olduğunu ortaya koydu (OR: 3.280, %95 CI: 1.564-6.878; p= 0.002). Ayrıca, ROC curve eğrilerinin karşılaştırıldığında, CAR'ın öngörücü performansının sırasıyla CRP ve albuminden daha yüksek olduğunu göstermiştir.

Sonuç: Bu çalışmada pPKG ile tedavi edilen STEMİ hastalarında yüksek CAR değerlerinin AF gelişimi ile bağımsız olarak ilişkili olduğunu gözlemledik.

Anahtar Kelimeler: C-reaktif protein/albumin oranı; yeni başlayan atriyal fibrilasyon; ST yükselmeli miyokart infarktüsü

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INTRODUCTION

New-onset atrial fibrillation (NOAF) is the most commonly observed arrhythmic complication in patients who presented with ST elevation myocardial infarction (STEMI). Depending on the study design, diagnostic method, and treatment modality used, the incidence of NOAF may vary from 2.3% to as high as 21%[1]. Prior clinical studies showed that the occurrence of NOAF is related with poor short- and long-term prognoses in patients with STEMI[2,3]. Therefore, some risk factors, such as older age, female gender, diabetes mellitus, increased heart rate, decreased systolic blood pressure, number of diseased vessels, and impaired left ventricular ejection fraction (LVEF), are demonstrated to be related with the development of NOAF in patients with STEMI[2-4]. However, the knowledge in this field is not in the desired range.

The C-reactive protein (CRP), as a marker of systemic inflammation, plays a major role in all stages of atherosclerosis, and it has been found to be associated with thromboembolic events[5]. Furthermore, many studies reported a strong independent association between elevated CRP levels and the presence of atrial fibrillation, as well as the development of NOAF[6,7]. In the body, the inflammatory processes may cause hypoalbuminemia due to the reduced synthesis and increased catabolism of serum albumin. Hypoalbuminemia is associated with the development of a new myocardial infarction in addition to increased mortality in patients with acute coronary syndrome[8,9]. A newly introduced inflammatory risk index, namely serum CRP to serum albumin ratio (CAR), may reflect the inflammatory condition better than serum CRP and serum albumin alone. Therefore, several studies have recently investigated whether the CAR can effectively reflect the prognosis of patients with cancer and critical illness[10,11]. These studies demonstrated that elevated CAR values measured on admission were related to poor prognosis among these patients. In addition, the CAR has been examined in patients with acute coronary syndrome, and it has been found to be associated with the severity of coronary artery disease (CAD)[12]. Therefore, the aim of the present study was to evaluate the potential utility of the CAR in predicting NOAF in patients with STEMI who underwent primary percutaneous coronary intervention (pPCI).

PATIENTS and METHODS

Study Patients

The present study was a retrospective analysis of the data related to 1153 patients with STEMI who underwent pPCI from January 2010 to April 2016. Patients who were having an active malignancy, acute or chronic infection(s), connective tissue disorders, proteinuria, presented with atrial fibrillation, cardiomyopathy, valvular heart disease, and previous diagnosis of CAD were excluded from the study. Patients with incomplete or missing data and those treated with emergency coronary artery bypass graft surgery due to pPCI failure were also excluded in the final study population. After the evaluation according to the above-mentioned exclusion criteria, a total of 1153 patients constituted the final study population. The diagnosis of STEMI was based on the following criteria: (I) patients presented with ongoing ischemic symptoms within 12 h, (II) a typical increase or decrease in the serum cardiac biomarkers, and (III) a new ST elevation ≥ 2 contiguous leads with the leads V1, V2, and V3 measuring ≥ 0.2 mV or ≥ 0.1 mV in the remaining leads or a newly developed left bundle-branch block pattern[13]. In the present study, the patients were diagnosed with NOAF if their electrocardiograms (ECGs) during the hospital course show irregular RR intervals, the absence of identifiable P waves with an unidentifiable isoelectric line, and atrial rhythm > 300 bpm[14]. In the present study, all patients post-pPCI were admitted to an intensive care unit with ECG monitoring for 48-72 h. According to the current guidelines, all patients received the standard medical therapy, including acetylsalicylic acid, clopidogrel, enoxaparin, beta-blockers, statins, and angiotensin-converting enzyme inhibitor/angiotensin receptor blocker unless contraindicated. The study protocol was approved by the local ethics committee (Kafkas University Faculty of Medicine, Ethic Committee, 80576354-050-99134) in accordance with the Declaration of Helsinki and Good Clinical Practice guidelines. Informed consent was not needed owing to the retrospective design of the study.

Laboratory Measurements

All venous blood parameters, including plasma glucose, serum creatinine, serum albumin, serum CRP, and troponin I levels, were obtained upon admission. Serum albumin and CRP levels were measured using a Roche Diagnostics Cobas 8000 c502 analyzer (Indianapolis, USA). CRP levels were measured according to the immunoturbidimetric method, and serum albumin levels were analyzed by the bromocresol green method. The CAR was defined as the serum CRP level divided by the serum albumin level. The estimated glomerular filtration rate (eGFR) was calculated using the modified diet renal disease equation.

Angiographic Analysis

In all patients, coronary angiography was performed via transfemoral route using the standard diagnostic catheters. All patients received a loading dose of 600 mg clopidogrel along with 300 mg acetylsalicylic acid before the procedure. Intravenous unfractionated heparin (70 IU/kg as loading dose, but not > 10.000 IU/kg) and additional dose, if necessary, were given to

obtain an activated clotting time of 250-300 s during the pPCI procedure. A successful pPCI was defined as a post-procedural residual-diameter stenosis < 30%, the thrombolysis myocardial infarction (TIMI) 3 flow in the infarct-related artery (IRA) after the procedure, and no procedural complications. Angiographic no-reflow was accepted if there is an acute transient or persistent coronary flow reduction (final TIMI flow grade < 3) at the target vessel lesion in the absence of spasm, thrombus, dissection, and/or significant residual stenosis[15].

Statistical Analysis

Data were performed using IBM SPSS Statistics for Windows, version 19.0. (IBM Corp., Armonk, NY, USA) for statistical analyses. Data were expressed as mean ± standard deviation for continuous variables and frequency and percentage for categorical variables. The chi-square or Fisher's exact test was performed for comparison of categorical data. The Kolmogorov-Smirnov test was used to test the normal distribution of continuous variables. If appropriate, the Pearson and Spearman tests were performed to assess the correlations of continuous variables. The Student's T-test or Mann-Whitney U test was performed for comparison of continuous variables between the two groups. A one-way ANOVA or Kruskal-Wallis test was used for comparison of continuous variables of the three groups, whereas the chi-square test was used for comparison of categorical variables. Multivariate logistic regression analysis was used to identify the independent predictors of NOAF. A two-sided p value of < 0.05 was considered as significant. A receiver operating characteristic (ROC) curve analysis was performed to determine the best cut-off value of CAR, serum CRP, and serum albumin in predicting NOAF. The De Long test was used for comparison of the ROC curves of CAR, serum CRP, and serum albumin levels.

RESULTS

In the present study, the mean age of the study population was 57 ± 12 years, and 81.2% (n= 936) of the study patients were male. The incidence of NOAF during in-hospital stay was 5.2% (n= 62 patients) in the present study. The baseline characteristics, laboratory, and angiographic findings of all patients are compared and shown in Table 1. Patients with NOAF were older, and the frequency of hypertension and diabetes mellitus was more common among these patients (p< 0.05 for each). Upon admission, we noted that patients with NOAF had higher prevalence of Killip class > 1, total ischemia time, and heart rate than those without NOAF (p< 0.05 for each). With respect to the laboratory finding, creatine kinase myocardial band (CK-MB) isoenzyme level, white blood cell (WBC) count, and CRP level were significantly higher, whereas serum albumin, hemoglobin, and eGFR levels were significantly lower in patients with NOAF than in those without NOAF (p< 0.05 for each). There was no statistically significant difference with respect to IRA.

The study subjects were divided into three groups according to their CAR tertiles: patients with CAR < 1.85 were assigned to the low tertile group (n= 384), those with 1.85 < CAR < 3.92 were assigned to the intermediate group (n = 385), and those with CAR > 3.92 were assigned to the high tertile group (n= 384). The frequencies of NOAF in the low, intermediate, and high tertile CAR groups were 1.3% (n= 5), 4.7% (n= 18), and 9.9% (n= 38), respectively (p< 0.001). Patients in the high tertile group were older in addition to having a more frequent history of hypertension and diabetes mellitus than those in the intermediate and low tertile groups. Upon admission, patients in the high tertile group had higher prevalence of Killip class > 1 and heart rate than those in the intermediate and low tertile groups. In addition, the indicators of larger infarct size, such as the presence of the left anterior descending artery as the IRA, the incidence of no-reflow, longer total ischemia time, higher peak CK-MB, and lower LVEF, were more frequently observed in the high tertile group. The comparisons of the three tertile groups are presented in Table 2.

Multivariate logistic regression analysis was performed to identify the independent predictors of NOAF with variables that showed marginal association in univariate analysis, including age, hypertension, diabetes mellitus, smoking, Killip class > 1, heart rate, hemoglobin, WBC count, CAR, eGFR, peak CK-MB, LVEF, total ischemia time, and no-reflow. The CAR (odds ratio (OR) 3.280, 95% confidence interval (CI) 1.564-6.878; p= 0.002), age (OR 1.029, 95% CI 1.006-1.053; p= 0.014), LVEF (OR 0.932, 95% CI 0.894-0.972; p= 0.001), and heart rate (OR 1.022, 95% CI 1.005-1.040; p= 0.010) were found to be independent predictors of NOAF (Table 3).

The CAR value of > 2.68 predicted an NOAF with a sensitivity of 85.2% and a specificity of 51.1% (area under the curve (AUC) 0.716, 95% CI 0.689-0.742, p< 0.001). The ROC curves were compared to assess whether CAR had an additional discriminative value over serum CRP and serum albumin levels. An AUC value of CAR was significantly higher than those of serum CRP (AUC 0.716, 95% CI 0.689-0.742 vs. AUC 0.676, 95% CI 0.648-0.703; p= 0.027) and serum albumin levels (AUC 0.716, 95% CI 0.689-0.742 vs. AUC 0.621, 95% CI 0.592-0.649; p= 0.016) (Figure 1).

DISCUSSION

In the present study, we mainly focused on the potential diagnostic utility of the CAR for the prediction of NOAF in patients who underwent pPCI for STEMI. The study results

Table 1. The baseline characteristics and laboratory findings of the study groups with and without NOAF

		Total patients (n= 1153)	NOAF (–) (n= 1092)	NOAF (+) (n= 61)	p
Age (years)		57 ± 12	56 ± 12	63 ± 13	< 0.001
Male gender, n (%)		936 (81.2)	891 (81.6)	45 (73.8)	0.128
Diabetes mellitus, n (%)		274 (23.8)	253 (23.2)	21 (34.4)	0.044
Hypertension, n (%)		477 (41.4)	437 (40.0)	40 (65.6)	< 0.001
Hyperlipidemia, n (%)		519 (45.0)	497 (45.5)	22 (36.1)	0.149
Family history of CAD, n (%)		270 (23.4)	260 (23.8)	10 (16.4)	0.183
Smoking, n (%)		631 (54.7)	607 (55.6)	24 (39.3)	0.013
Killip class > 1 on admission, n (%)		189 (16.4)	163 (14.9)	26 (42.6)	< 0.001
Systolic blood pressure (mmHg)		132 ± 32	132 ± 32	134 ± 43	0.527
Heart rate (bpm)		77 ± 16	77 ± 16	87 ± 17	< 0.001
Hemoglobin (g/dL)		13.7 ± 1.8	13.7 ± 1.8	13.1 ± 2.1	0.038
WBC count ($\times 10^3/\mu L$)		12.3 ± 3.9	12.3 ± 3.8	13.6 ± 4.4	0.012
C-reactive protein (mg/dL)		9.8 (5.6-17.6)	9.5 (5.4-17)	16.1 (11.0-32.1)	< 0.001
Serum albumin (g/dL)		3.74 ± 0.49	3.75 ± 0.48	3.53 ± 0.55	0.001
C-reactive protein to albumin ratio		2.72 (1.47-4.77)	2.61 (1.42-4.64)	4.86 (3.27-8.23)	< 0.001
Estimated GFR, mL/min/1.73 m^2		88.2 ± 25.8	88.7 ± 25.6	78.1 ± 28.4	0.006
Peak CK-MB (ng/mL)		179 (100-321)	171.5 (98-305)	369 (199-444)	< 0.001
Left ventricular ejection fraction (%)		46.7 ± 8.3	47.1 ± 8.1	39.2 ± 8.1	< 0.001
Total ischemia time (min)		178 (115-270)	176 (114-268)	208 (128-330)	0.033
Infarct-related artery, n (%)	LAD	606 (52.6)	567 (51.9)	39 (63.9)	0.105
	CX	140 (12.1)	136 (12.5)	4 (6.6)	
	RCA	391 (33.9)	373 (34.2)	18 (29.5)	
	Other	16 (1.4)	16 (1.5)	0	
No-reflow, n (%)		145 (12.6)	130 (11.9)	15 (24.6)	0.004

NOAF: New-onset atrial fibrillation, CAD: Coronary artery disease, CK-MB: Creatine kinase myocardial band, Cx: Circumflex artery, GFR: Glomerular filtration rate, LAD: Left ascending artery, RCA: Right coronary artery, WBC: White blood cell.
Continuous variables with normal distribution were expressed as mean ± standard deviation, and continuous variables without normal distribution were expressed as median (25^{th}-75^{th} percentiles).

suggested that elevated CAR levels were independently associated with NOAF development in patients with STEMI. In addition, this new index might be a better parameter than serum CRP and serum albumin levels alone for the prediction of NOAF in these patients.

In patients who presented with STEMI, atrial fibrillation is the most commonly observed supraventricular arrhythmia following pPCI[16]. In previous studies, it was demonstrated that the development of NOAF in the acute setting of myocardial infarction is associated with poor short- and long-term prognoses in patients undergoing pPCI[2-4]. Previous clinical studies showed that some risk factors, such as left or right ventricular dysfunction, atrial ischemia, pericarditis, excessive catecholamine release, drugs, acute hypoxia, and hypopotassemia, were associated with the development of NOAF after myocardial infarction[17]. In the present study, patients with NOAF had larger infarct size than those without NOAF. Furthermore, we observed that the presence of hypertension and diabetes mellitus, lower LVEF, older age, and elevated heart rate were independent predictors of NOAF, which was consistent with previous studies[3,18-20].

The inflammatory status is closely associated with an increased risk of atrial fibrillation in patients with acute myocardial infarction. The serum CRP, which is a positive acute pha-

Table 2. Demographic, clinical, laboratory, and coronary angiographic characteristics of all patients according to CAR tertiles

		Tertile groups of CAR			
		CAR < 1.85 (n= 384)	1.85 < CAR < 3.92 (n= 385)	CAR > 3.92 (n= 384)	p
Age (years)		53 ± 11	57 ± 12	59 ± 13	< 0.001
Female gender, n (%)		331 (86.2)	313 (81.3)	292 (76)	0.002
Diabetes, n (%)		76 (19.8)	89 (23.1)	109 (28.4)	0.019
Hypertension, n (%)		121 (31.5)	167 (43.4)	189 (49.2)	< 0.001
Hyperlipidemia, n (%)		179 (46.6)	179 (46.5)	161 (41.9)	0.330
Family history of CAD, n (%)		90 (23.4)	100 (26)	80 (20.8)	0.243
Smoking, n (%)		220 (57.3)	216 (56.1)	195 (50.8)	0.155
Killip class > 1 on admission, n (%)		38 (9.9)	50 (13)	101 (26.3)	< 0.001
Systolic blood pressure (mmHg)		130 ± 26	134 ± 30	132 ± 39	0.199
Heart rate (bpm)		75 ± 15	76 ± 15	80 ± 18	< 0.001
Hemoglobin (g/dL)		13.8 ± 1.7	13.7 ± 1.6	13.4 ± 2	0.014
WBC count ($\times 10^3/\mu L$)		11.8 ± 3.3	11.9 ± 3.2	13.3 ± 4.7	< 0.001
C-reactive protein (mg/dL)		4.29 (2.87-5.60)	9.87 (8.1-12.4)	21.50 (17.55-32.4)	< 0.001
Serum albumin (g/dL)		3.87 ± 0.48	3.76 ± 0.43	3.60 ± 0.51	< 0.001
CAR		1.11 (0.73-1.47)	2.72 (2.19-3.37)	6.06 (4.78-9.09)	< 0.001
Estimated GFR, mL/min/1.73 m^2		94.72 ± 24.33	87.29 ± 23.42	80.69 ± 26.96	< 0.001
Peak CK-MB (ng/mL)		127 (78-234)	158 (99-269)	288 (162-432)	< 0.001
Left ventricular ejection fraction (%)		49.8 ± 6.9	48.4 ± 7.1	41.83 ± 8.63	< 0.001
Total ischemia time (min)		151 (102-228)	174 (111-260)	221 (135-332)	0.028
Infarct-related artery, n (%)	LAD	183 (47.7)	183 (47.5)	240 (62.5)	< 0.001
	CX	51 (13.3)	51 (13.2)	38 (9.9)	
	RCA	144 (37.5)	143 (37.1)	104 (27.1)	
	Other	6 (1.6)	8 (2.1)	2 (0.5)	
No-reflow, n (%)		23 (6)	39 (10.1)	83 (21.6)	< 0.001
New-onset atrial fibrillation		5 (1.3)	18 (4.7)	38 (9.9)	< 0.001

CAD: Coronary artery disease, CAR: C-reactive protein to albumin ratio, CK-MB: Creatine kinase myocardial band, Cx: Circumflex artery, GFR: Glomerular filtration rate, LAD: Left ascending artery, RCA: Right coronary artery, WBC: White blood cell.
Continuous variables with normal distribution were expressed as mean ± standard deviation, and continuous variables without normal distribution were expressed as median (25^{th}-75^{th} percentiles).

Table 3. Independent predictors of new-onset atrial fibrillation in multivariate logistic regression analysis

	Univariate analyses			Multivariate analyses		
Variables	**OR**	**95% CI**	**p**	**OR**	**95% CI**	**p**
High age	1.047	1.025-1.069	< 0.001	1.029	1.006-1.053	0.014
Increased heart rate	1.043	1.026-1.060	< 0.001	1.022	1.005-1.040	0.010
Decreased left ventricular ejection fraction	0.894	0.865-0.923	< 0.001	0.932	0.894-0.972	0.001
Increased C-reactive protein to albumin ratio	1.130	1.082-1.181	< 0.001	3.280	1.564-6.878	0.002

CI: Confidence interval, OR: Odds ratio.

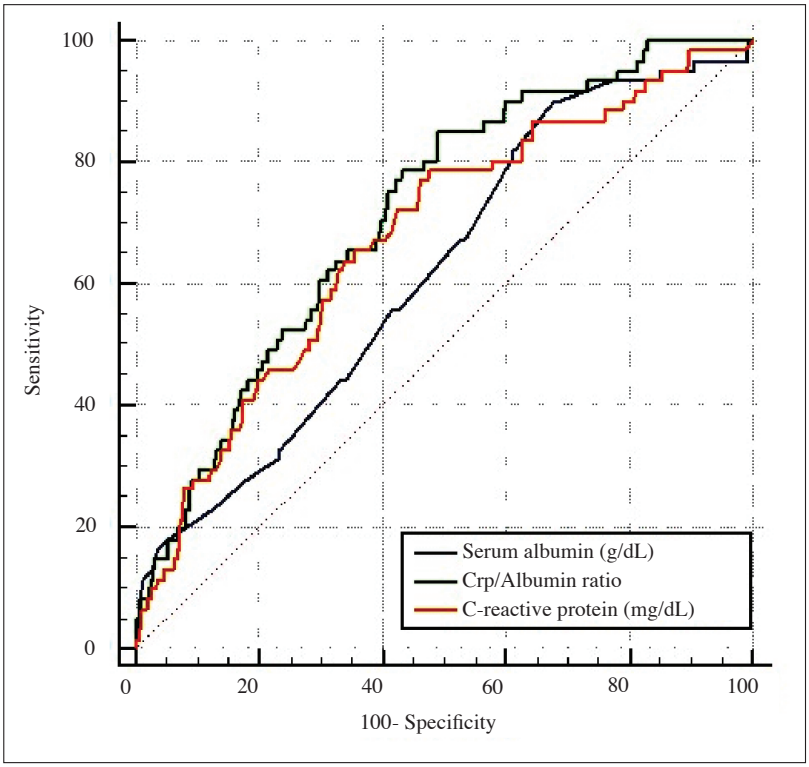

Figure 1. In receiver operating characteristic curve analyses, the comparison of the AUC value of CAR was higher than that of serum CRP and serum albumin alone (0.716 vs. 0.676 and 0.621, p= 0.027 and p= 0.016, respectively).

se reactant, is produced by the hepatocytes upon activation by cytokines, such as interleukin 6 and tumor necrosis factor-alpha. The levels of serum CRP increase considerably with inflammation or infection[21]. The CRP is also accepted as a potent cardiovascular risk factor. Many studies have shown that high levels of CRP are associated with the development of NOAF[6,7,22]. In our study, the NOAF group had higher levels of CRP than those without NOAF. The inflammation could also cause a decrease in serum albumin, which is the most abundant blood plasma protein that is produced by the liver. Serum albumin is a negative acute phase reactant; thus, its blood level decreases during inflammation. In previous studies, serum albumin was shown to be associated with the severity of inflammation, poor prognosis, and elevated mortality rates[23-25]. Previously, serum albumin was reported to be associated with adverse cardiovascular events in patients with STEMI[8,9,26]. However, the possible relationship between serum albumin levels and the occurrence of NOAF is unknown. In the present study, we were able to demonstrate that the incidence rate of NOAF was associated with decreased albumin levels.

The CAR, a newly introduced inflammatory risk index, is thought to be a better indicator of inflammation than serum CRP and serum albumin alone. This idea has been validated in patients with cancer and acute medical illnesses[10,11]. Cagdas et al. recently demonstrated that the predictive performance of CAR is higher than that of serum CRP and serum albumin alone in the prediction of high Synergy between PCI with Taxus and Cardiac Surgery score in patients with acute coronary syndrome. In our study, higher CAR values were associated with NOAF. As the patients were divided into low, intermediate, and high CAR tertiles, the frequency of NOAF was gradually increased through CAR tertiles. In our study, the CAR was also associated with increased age, hypertension, diabetes mellitus, and female gender. The factors related to larger infarct size, such as increased peak CK-MB, longer total ischemia time, left anterior descending coronary artery as the IRA, the frequency of no-reflow, and lower LVEF, were more frequently observed in patients with high CAR values than in those with lower CAR values. In previous studies, it has been shown that these factors were associated with the development of NOAF[12]. A close relationship between the CAR and the above-mentioned factors may have contributed to the significance of our results.

Limitations

The present study has several limitations. The frequency of NOAF could have been higher than is observed, as it is possible to miss some silent NOAF attacks. Furthermore, the lack of ability to identify the silent/asymptomatic paroxysmal NOAF episodes in patients prior to admission is also a limitation of the

present study. Moreover, this was a nonrandomized, retrospective study that was conducted in a single center. The prognostic value of CAR could not be assessed in the present study owing to the limited number of event rates. Finally, our finding needs to be verified by further larger sample studies.

CONCLUSION

In conclusion, the CAR was associated with the development of NOAF in patients undergoing pPCI for STEMI. This simple, cheap, and easily obtained parameter may be used in risk stratification of patients with STEMI for the development NOAF following pPCI.

CONFLICT of INTEREST

The authors reported no conflict of interest related to this article.

AUTHORSHIP CONTRIBUTIONS

Concept/Design: MY, MÇ
Analysis/Interpretation: YK, MK
Data Acquisition: MY, MÇ, YK
Writting: MY, TÇ, İR
Critical Revision: İT, OG
Final Approval: All of authors.

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