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RESEARCH

The association between C-reactive protein to albumin ratio and infarct location in patients with ST-segment elevation myocardial infarction

ST segment yükselmeli miyokard infarktüsü olan hastalarda C-reaktif protein/albümin oranı ile infarktüs lokalizasyonu arasındaki ilişki

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Abstract

Purpose: Inflammation has a crucial role in the pathogenesis of ST segment elevation myocardial infarction (STEMI). Recently, the C-reactive protein to albumin ratio (CAR) has emerged as a novel parameter of systemic inflammation. Although studies have demonstrated the that anterior STEMI location is associated with a higher infarct size and worse prognosis, no study has investigated the CAR in relation to infarct location. We herein aimed to evaluate whether there is a difference regarding the CAR between patients with anterior and non-anterior STEMI location.

Materials and Methods: The study population comprised 273 consecutive STEMI patients who were divided into 2 groups based on the STEMI location, as the anterior STEMI group (n=114) and non-anterior STEMI group (n=159). Baseline characteristics were recorded, and the CAR was calculated for all patients.

Results: Both groups were similar in terms of the baseline clinical characteristics. However, syntax score ($p<0.001$) was significantly higher and ejection fraction was lower ($p<0.001$) in the anterior STEMI group compared to the non-anterior STEMI group. The anterior STEMI patients had also significantly higher CAR ($p<0.001$). Correlation analysis showed that CAR was positively correlated with the syntax score ($r = 0.249$, $p < 0.001$) and troponin ($r = 0.158$, $p = 0.009$) whereas negatively correlated with ejection fraction ($r = -0.124$, $p = 0.040$). In the linear regression analysis, anterior STEMI location and syntax score were independently associated with the CAR.

Conclusion: We found that STEMI patients with anterior location had significantly higher CAR. Also, anterior infarct location and syntax score were independently related to the CAR level. We suggest that a more widespread inflammatory response is triggered in patients

Öz

Amaç: ST yükselmeli miyokard enfarktüsünün (STYMİ) patogenezinde inflamasyonun önemli bir rolü vardır. Son zamanlarda, C-reaktif protein/albümin oranı (CAO), sistemik inflamasyonun yeni bir parametresi olarak ortaya çıkmıştır. Çalışmalar anterior STYMİ lokalizasyonunun daha fazla infarktüs boyutu ve kötü prognoz ile ilişkili olduğunu göstermesine rağmen, hiçbir çalışma CAO'nun infarktüs lokalizasyonu ile ilişkisini araştırmamıştır. Bu çalışmada, anterior ve anterior yerleşimli olmayan STYMİ hastalar arasında CAO açısından fark olup olmadığını değerlendirmeyi amaçladık.

Gereç ve Yöntem: Çalışma popülasyonu, STEMI lokasyonuna göre anterior STEMI grubu (n=114) ve anterior olmayan STEMI grubu (n=159) olmak üzere 2 gruba ayrılan 273 ardışık STEMI hastasından oluşmuştur. Temel özellikler kaydedildi ve tüm hastalar için CAO hesaplandı.

Bulgular: Her iki grup da bazal klinik özellikler açısından benzerdi. Ancak anterior STYMİ grubunda, anterior olmayan STYMİ grubuna kıyasla syntax skoru anlamlı derecede yüksek ($p<0.001$) ve ejeksiyon fraksiyonu daha düşüktü ($p<0.001$). Anterior STYMİ hastaları aynı zamanda anlamlı olarak daha yüksek CAO ($p<0.001$) seviyesine sahipti. Korelasyon analizi, CAO'nun syntax skoru ($r=0.249$, $p<0.001$) ve troponin ($r=0.158$, $p=0.009$) ile pozitif, ejeksiyon fraksiyonu ($r= -0.124$, $p=0.040$) ile negatif korelasyon gösterdiğini saptadı. Lineer regresyon analizinde anterior STYMİ lokalizasyonu ve syntax skoru, CAO seviyesi ile bağımsız olarak ilişkili idi.

Sonuç: Anterior yerleşimli STYMİ hastalarının anlamlı olarak daha yüksek CAR'a sahip olduğunu bulduk. Ayrıca, anterior enfarktüs yerleşimi ve syntaks skoru bağımsız olarak CAR düzeyi ile ilişkiliydi. Anterior STEMI hastalarında, anterior olmayan STEMI hastalarına kıyasla

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with anterior STEMI compared to the patients with non-anterior STEMI.
Keywords: Infarct location, inflammation, C-reactive protein to albumin ratio

daha geniş bir inflamatuvar yanıtın tetiklendiğini düşünüyoruz.
Anahtar kelimeler: Enfarktüs yeri, inflamasyon, C-reaktif protein/albümin oranı

INTRODUCTION

ST segment elevation myocardial infarction (STEMI) is a type of acute coronary syndrome (ACS) and is characterized by the presence of persistent chest pain, ST segment elevation on electrocardiography (ECG) and elevation in cardiac enzymes[1]. Studies have demonstrated that STEMI most likely develops in the location of the left anterior descending artery (LAD), that supplies a large portion of the whole heart blood[2,3]. The principal treatment option in STEMI patients is the application of reperfusion therapy as soon as possible[1]. Risk stratification has a crucial role in the management and prognosis of STEMI patients. The location of myocardial infarction (MI) may also provide important information to the clinician regarding the prognosis[4,5]. Previous studies compared the prognosis of anterior vs. non-anterior STEMI and reported that anterior STEMI location had larger infarct size, greater reduction in ejection fraction and worse outcomes compared to non-anterior STEMI location[2-6]. Therefore, anterior location has been accepted as a risk factor for the prediction of poor prognosis in STEMI patients[7]. Although the relationship between anterior STEMI and poor prognosis is mainly attributed to the infarct size, the full mechanisms underlying this association have not yet been exactly elucidated.

Inflammation has an important role in the pathogenesis, development and prognosis of STEMI[8]. So far, many inflammatory parameters including the neutrophil to lymphocyte ratio (NLR) and platelet to lymphocyte ratio (PLR) have been found to be related to the prognosis of ACS[8,9]. Recently, a novel marker of inflammation, the C-reactive protein (CRP) to albumin ratio (CAR), has emerged. This marker reflects the balance between the CRP and albumin level and is a more sensitive marker than CRP and albumin separately[10,11]. Previous studies showed that CAR was significantly related to adverse outcomes in patients with coronary artery disease and had a better predictive value in detecting the presence of coronary artery disease than the NLR and PLR[11-13]. Moreover, it was reported that an elevated CAR was associated with thrombus burden, no-reflow, acute kidney injury and long-term mortality in patients with STEMI[12].

As a larger infarct size develops in the anterior STEMI localization, we hypothesized that a more widespread inflammatory response may be triggered in anterior STEMI patients compared to the non-anterior patients. However, when we reviewed the literature, we found no study evaluating the relationship between STEMI location and CAR. Therefore, we wanted to make a contribution to the literature on this subject. Our aim in this study was to evaluate whether there is a difference regarding the inflammatory response measured by CAR between patients with and without anterior STEMI location.

MATERIALS AND METHODS

Study population

In this study, 273 consecutive patients who were admitted with the diagnosis of STEMI and underwent coronary angiography were included retrospectively. STEMI diagnosis was based on the current guideline, where the ST segment elevation criteria were as follows: ≥ 1 mm ST segment elevation in all leads other than leads V2–V3, in which the cut-off points of the ST-segment elevation were ≥ 2mm in males ≥ 40 years; ≥ 2.5 mm in males < 40 years, or ≥ 1.5 mm in females regardless of age[1]. All data were obtained from the hospital information system, archive records and coronary angiography imaging and report. All patients who were older than 18 years of age and whose laboratory, electrocardiographic and angiographic data were available from the hospital archives were included in the study. The exclusion criteria were as follows: patients with coronary artery bypass graft, a previous history of myocardial infarction, heart failure, non-interpretable ECG, left bundle branch block, hematologic/inflammatory diseases, malignancies, active infection, sepsis, and chronic renal and hepatic disorder. An approval was obtained from the Harran University Ethics Committee for the study (number: HRÜ.22/04/18, date: 21.02.2022).

Data collection

Baseline clinical characteristics were recorded for all patients and initial blood sample on admission, before the coronary angiography, were examined. Complete blood counts and biochemical parameters including creatinine, cardiac markers, lipid panel, CRP and albumin were recorded. The CAR was calculated as the ratio of the CRP to that of the albumin. Moreover, the NLR was calculated as the ratio of the neutrophil count to that of the lymphocyte count and the PLR was calculated as the ratio of the platelet count to that of the lymphocyte count.

Electrocardiography

A 12-lead ECG was obtained from all patients at admission. After the diagnosis of STEMI according to the current guideline, the location of ST elevation was recorded, and patients were divided groups based on the location of the STEMI (1). The anterior STEMI group (n = 114) included patients who had all leads located in anteroseptal, anterior, and anterolateral, whereas the non-anterior STEMI group (n = 159) included patients who had all leads located in the lateral, inferior, posterior, and right ventricle[14].

Coronary angiography

Coronary angiography was conducted with standard techniques via the radial or femoral route. The infarct related artery was determined, and primary percutaneous coronary intervention (PCI) was performed. The STEMI location was also confirmed by infarct related artery: LAD-related STEMI (anterior MI) versus non-LAD-related (circumflex artery [CX] and right coronary artery [RCA] related) STEMI (non-anterior MI). Critical stenosis was defined as ≥ 50% stenosis for the left main coronary artery and ≥ 70% for the remaining major epicardial arteries. The Synergy between PCI with TAXUS and Cardiac Surgery (syntax) score was measured as previously described in the literature[15].

Statistical analysis

Normality of the study data was evaluated with the Kolmogorov-Smirnov. Continuous data with normal distribution were defined as mean ± SD and compared with student t test. Continuous data without normal distribution were defined as median (25^{th} -75^{th} interquartile range) and compared with Mann-Whitney U test. Categorical data were defined as number and percentage and compared with chi-square test. Spearman correlation coefficient was applied for the correlation analysis. Linear regression analysis was used to identify the independent parameters that may be associated with the CAR. Parameters found to be significant in univariate analyses and possible clinical factors that may be associated with STEMI and inflammation were included in the regression model. P value of <0.05 was defined as significant. A post hoc power analysis with G-power program was performed and indicated that the power of study was 97.6%.

RESULTS

The mean age of the patients was 61.88 ± 11.87 and 60.4% (165) were male. Primary PCI was performed on all patients. The LAD was the infarct related artery in 114 (41.8%) patients, CX in 61 (22.3%) patients and RCA in 98 (35.9%) patients (Figure 1). Our study population was divided into two groups based on the STEMI location. Of the patients, 114 (41.8%) patients had anterior STEMI whereas 159 (58.2%) patients had non- anterior STEMI.

Baseline characteristics and angiographic variables of patients with and without anterior STEMI are presented in Table 1. Age, sex, comorbidities, body mass index, blood pressures and the frequency of 3-vessel disease were not different between anterior and non-anterior STEMI groups. However, the syntax score (18 [11-22] vs. 12 [8-18], $p < 0.001$) was significantly higher in the anterior STEMI group compared to the non-anterior STEMI group. On the other hand, patients with anterior STEMI had significantly lower ejection fraction compared to the non-anterior STEMI patients (35.84 ± 6.49 vs. 42.58 ± 6.36, $p < 0.001$) (Table 1).

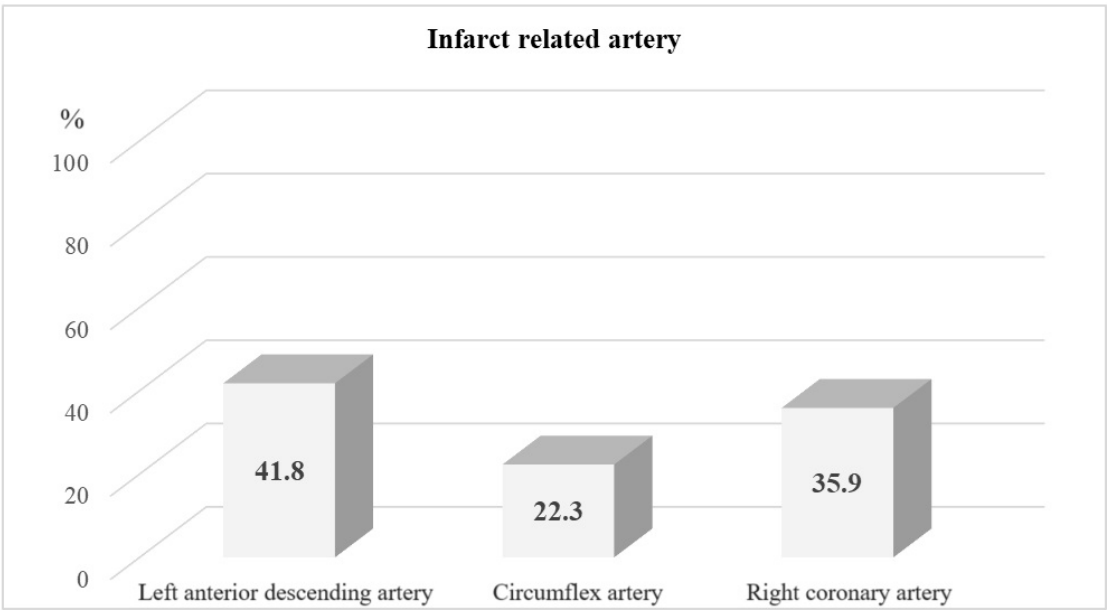

Figure 1. Distribution of infarct related artery

Table 1. Comparison of baseline characteristics and angiographic variables between anterior versus non-anterior myocardial infarction.

	Anterior STEMI (n = 114)	**Non-anterior STEMI (n = 159)**	**P**
Age, years	62.34 ± 13.27	61.55 ± 10.78	0.589
Gender, male (%)	74 (64.9)	91 (57.2)	0.201
BMI, kg/m²	27.85 ± 3.57	28.19 ± 4.71	0.678
Hypertension (%)	61 (53.5)	93 (58.5)	0.500
Diabetes mellitus (%)	38 (33.3)	52 (32.7)	0.413
Smoking (%)	74 (64.9)	90 (56.6)	0.167
Heart rate, /min.	83.97 ± 17.75	80.16 ± 15.61	0.066
Systolic BP, mmHg	129.41 ± 19.71	127.84 ± 19.89	0.587
Diastolic BP, mmHg	77.31 ± 11.21	75.63 ± 10.58	0.292
Ejection fraction (%)	35.84 ± 6.49	42.58 ± 6.36	**<0.001**
Syntax score	18 (11-22)	12 (8-18)	**<0.001**
Three-vessel disease (%)	25 (21.9)	49 (30.8)	0.103

BMI: body mass index, BP: blood pressure, STEMI: ST segment elevation myocardial infarction

Complete blood count and biochemical parameters of the study groups are listed in Table 2. The anterior STEMI patients had significantly higher CK-MB (62 [22-128] vs. 24 [6-46], $p < 0.001$), troponin (4000 [3401-8257] vs. 1895 [695-5700], $p < 0.001$) and lactate dehydrogenase (296 [205-444] vs. 239 [195-300], $p = 0.006$) levels. Also, CRP ($p < 0.001$) level was importantly higher, whereas the albumin level was significantly lower ($p = 0.031$) in the anterior STEMI patients compared to the non-anterior STEMI patients. Moreover, anterior STEMI patients had a higher CAR level (1.18 [0.44-3.30] vs. 0.45 [0.12-1.25], $p < 0.001$) and NLR (3.67 [2.22-5.59] vs. 2.94 [2.12-4.35], $p = 0.021$).

Table 2. Comparison of hematological and biochemical parameters between anterior versus non-anterior myocardial infarction.

	Anterior STEMI (n = 114)	Non-anterior STEMI (n = 159)	P
Glucose, mg/dl	130 (106-183)	134 (111-187)	0.288
Creatinine, mg/dl	0.86 ± 0.28	0.87 ± 0.22	0.773
CK-MB, ng/mL	62 (22-128)	24 (6-46)	**<0.001**
Troponin, pg/mL	4000 (3401-8257)	1895 (695-5700)	**<0.001**
LDH, U/L	296 (205-444)	239 (195-300)	**0.006**
Triglyceride, mg/dl	110 (82-175)	120 (86-200)	0.117
Total cholesterol, mg/dl	180.82 ± 43.16	174.48 ± 42.73	0.230
LDL- cholesterol, mg/dl	122 (98-144)	116 (91-140)	0.302
HDL- cholesterol, mg/dl	36.79 ± 9.61	35.09 ± 9.22	0.141
WBC, $x10^3/\mu l$	11.23 (9.28-14.67)	10.05 (7.73-13.68)	0.063
Neutrophil, $x10^3/\mu l$	8.80 (5.96-10.95)	6.48 (4.90-9.80)	**0.010**
Lymphocyte, $x10^3/\mu l$	2.10 (1.41-3.30)	2.20 (1.70-3.13)	0.381
Platelet, $x10^3/\mu l$	261 (220-303)	251 (213-287)	0.143
Hemoglobin, g/dl	13.71 ± 1.87	13.38 ± 1.72	0.135
MPV, fl	8.98 ± 1.37	8.78 ± 1.59	0.334
CRP, mg/dl	3.67 (1.88-12.50)	1.90 (0.56-5.10)	**<0.001**
Albumin, g/dl	3.91 ± 0.50	4.03 ± 0.38	**0.031**
CAR	1.18 (0.44-3.30)	0.45 (0.12-1.25)	**<0.001**
NLR	3.67 (2.22-5.59)	2.94 (2.12-4.35)	**0.021**
PLR	117.22 (81.67-168.06)	113.75 (78.79-114.37)	0.337

CK-MB: Creatine Kinase MB, LDH: Lactate dehydrogenase, LDL: low density lipoprotein, HDL: high density lipoprotein, WBC: white blood cell, MPV: mean platelet volume, CRP: C-reactive protein, CAR: CRP to albumin ratio, NLR: neutrophil to lymphocyte ratio, PLR: platelet to lymphocyte ratio, STEMI: ST segment elevation myocardial infarction

Correlation analysis was applied to identify the correlation of CAR with the clinical and angiographic parameters. It was found that CAR was positively correlated with syntax score ($r = 0.249$, $p < 0.001$), troponin ($r = 0.158$, $p = 0.009$) and CK-MB ($r = 0.128$, $p = 0.035$), whereas it was negatively correlated with ejection fraction ($r = -0.124$, $p = 0.040$).

Linear regression analysis was applied to identify the independent parameters that may be associated with the CAR. It was found that anterior STEMI and syntax score were independently associated with the CAR (Table 3).

Table 3. Linear regression analysis showing the independent variables related to CAR

	Unstandardized Coefficients		Standardized Coefficients	t	P value
	B	Std. Error	β (beta)		
Anterior STEMI	1.371	0.627	0.202	2.186	0.030
Syntax score	0.104	0.041	0.219	2.532	0.012

STEMI: ST segment elevation myocardial infarction, CAR: CRP to albumin ratio
Included variables: Age, gender, hypertension, diabetes mellitus, heart rate, STEMI location, left ventricular ejection fraction, three-vessel disease, syntax score, creatinine, LDL-cholesterol, HDL-cholesterol, CK-MB, troponin, LDH, hemoglobin

DISCUSSION

We aimed to compare the CAR between anterior and non-anterior STEMI patients. The main findings of our study were that (I) CAR was importantly higher in the anterior STEMI patients compared to the non-anterior STEMI patients, (II) CAR was positively correlated with the syntax score, CK-MB and troponin whereas it was negatively correlated with ejection fraction (III) anterior STEMI and syntax score were independently related to CAR level.

Acute coronary syndromes are the leading cause of mortality worldwide[16]. STEMI is one of the most urgent ACS types requiring intervention, and reperfusion therapy should be provided as soon as possible. Because it is often related to the acute total occlusion of an epicardial coronary artery including the LAD, CX and RCA[1]. LAD supplies the anterior wall of the myocardium which constitutes a large proportion of whole myocardium, and its acute occlusion leads to anterior STEMI. It has been reported that STEMI most likely develops in the location of the LAD[2,3]. Similar to the literature, we also found that LAD (41.8%) was the most common infract related artery in patients with STEMI (the frequency of CX and RCA was 22.3% and 35.9%, respectively).

Previous studies compared anterior versus non-anterior STEMI patients in terms of baseline and angiographic characteristics. In these studies, it was detected that the anterior and non-anterior STEMI patients had similar baseline clinical characteristics[2,7,17]. We also found that patients with and without anterior STEMI had similar baseline characteristics. On the other hands, studies regarding the frequency of 3-vessels between anterior vs. non-anterior STEMI patients are contradictory. Some previous studies found that the 3-vessel disease frequency was higher in non-anterior STEMI[18,19], whereas others found no significant difference between the two groups[17,20]. In the current study, although the frequency of the 3-vessel disease tended to be higher in non-anterior STEMI (30.8% vs. 21.9%), we observed that this difference did not reach significance. ($p = 0.103$).

Inflammation plays an important role in the development, progression and prognosis of ACS[8,9]. The ischemic injury during myocardial infarction induces an intense inflammatory response that initiates cardiac repair process. Inflammatory cells infiltrate the infarct area to remove the necrotic cell residues from the infarct zone and contribute to the wound healing process and cardiac repair[21]. However, this inflammatory response is also implicated in the pathogenesis of post-infarct remodeling and heart failure[9,21]. Neutrophils are the first infiltrated cells the infarct area following the onset of infarction. Subsequently, they activate all reparative pathways required[22]. On the other hand, an excessive inflammatory response may also play pathological roles such as adverse remodeling that cause heart failure[23].

To date, many inflammatory parameters have been used to evaluate prognosis in patients with STEMI[8,9,24]. Most of these studies evaluated the NLR and PLR due to the fact that they can be easily measured. In the present study, the NLR and PLR were also assessed, and it was found that the NLR was significantly higher in anterior STEMI patients than in the non-anterior STEMI patients. However, the CAR has recently emerged as a novel marker of inflammation. CRP is also released due to an inflammatory response during ischemic injury and plays a significant role in prothrombotic events. Contrary to this, the inflammatory response result in a decrease in the albumin level due to reduced synthesis and increased catabolism[12,25]. CAR reflects the balance between these two parameters and can also easily be calculated from the laboratory analysis[10-12]. Studies reported that increased CAR was related to adverse outcomes in STEMI[12].

Risk stratification is important to determine the prognosis in patients with acute STEMI[1]. Traditionally, the anterior location has been considered a risk factor because anterior STEMI has been shown to be associated with worse prognosis than non-anterior STEMI[7,17]. This is mainly attributed to a larger infarct size, greater reduction in the ejection fraction and worse epicardial and microvascular reperfusion compared with non-anterior infarcts[18,19]. Similar to previous studies, it was also found herein that the enzymatic infarct size was higher and the ejection fraction was lower in the anterior STEMI patients. However, the underlying mechanism of the worse prognosis in anterior STEMI may not only be due to these factors and there are probably different mechanisms that have not yet been characterized. It is our belief that inflammation may also be a variable that contributes to these mechanisms. Although studies have shown that inflammatory parameters were an important predictor of the prognosis[9,24], very limited studies have assessed the link between the STEMI location and inflammatory parameters. In a previous study, Kiris et al. assessed the link between left ventricular function, inflammatory cytokine levels and location of myocardial infarction in patients undergoing primary coronary artery bypass grafting. They found that adrenomedullin, which is a pro-inflammatory cytokine, was importantly higher in the anterior STEMI compared to the posterior/inferior STEMI group[26]. However, no study has compared the CAR between anterior and non-anterior STEMI in literature.

In the present study, the CAR was compared between patients with and without anterior STEMI and it was detected that CAR was importantly higher in the anterior STEMI patients than in the non-anterior STEMI. We also also observed that patients with anterior STEMI had lower ejection fraction, and higher infarct area measured by the troponin and CK-MB levels. Similarly, it was reported in previous studies that anterior STEMI patients had higher level of cardiac enzymes than non-anterior STEMI patients[5,17,27]. These results suggest that the inflammatory response during myocardial infarction may be related to the infarct location and infarct size, as well as a more widespread inflammation is triggered in anterior STEMI. Indeed, the fact that the CAR was positively correlated with the syntax score and troponin, and negatively correlated with ejection fraction in our study supports this idea. It can be concluded that because a larger portion of the myocardium is under risk in anterior STEMI, a more widespread inflammatory response develops.

The syntax score which an anatomical scoring system is used to determine the angiographic properties of coronary lesions and the complexity coronary artery disease, and provides information regarding prognosis in coronary artery disease[15,28]. It was reported that the CAR was significantly increased in the high syntax score group and was an independent predictor of the syntax score[25]. Similar to the previous findings, the current study also found that the CAR was positively correlated with syntax score. We also detected that anterior STEMI patients had higher syntax score compared to non-anterior STEMI patients. Moreover, we demonstrated that anterior STEMI location and syntax score were independently associated with CAR. When all these findings evaluated together, it was considered that infarct location, size and lesion complexity may be related to the amount of inflammatory response and especially the CAR. Further studies are required to better identify the association of the CAR with the infarct location.

The major limitation of this study was the small number of patients. We did not perform a priori power test for sample size because of retrospective design of our study. However, when we performed a post hoc power analysis, we calculated the power of the study as 97.6% (effect size: 0.49, α=0.05). Second, we in hospital mortality and long-term prognosis were not evaluated. Following up the patients and assessing the association of the CAR with clinical outcomes according to STEMI location could have provided an additional contribution to the study. Third, we could not evaluate the duration of chest pain on admission and door-to-ballon time. Inflammatory and biochemical parameters may have been affected from by these clinical variables. Finally, patients with a previous history of coronary artery disease and coronary artery bypass graft were not included. Therefore, our results may not reflect all STEMI patients. We think that further prospective studies evaluating total ischemic time and measuring infarct size by imaging methods will guide to a better understanding of the relationship between STEMI location and inflammation.

In this study, we compared the CAR between anterior and non-anterior STEMI location. We observed that patients with an anterior STEMI location had importantly higher CAR. In addition, CAR was significantly correlated with the syntax score and infarct size measured by troponin and ejection fraction. Moreover, anterior STEMI location and syntax score were independently associated with CAR level. According to the findings of our study, we suggest that a more widespread inflammatory response is triggered in anterior STEMI patients due to the larger infarct area in these patients.

Author Contributions: Concept/Design : ZT, HF; Data acquisition: ZT, HF; Data analysis and interpretation: ZT, HF; Drafting manuscript: ZT, HF; Critical revision of manuscript: ZT, HF; Final approval and accountability: ZT, HF; Technical or material support: ZT, HF; Supervision: ZT, HF; Securing funding (if available): n/a.
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