Research Article / Araştırma Makalesi

Neutrophil/Lymphocyte Ratio and Platelet/Lymphocyte Ratio in Predicting Chronic Total Occlusion in ST-Segment Elevation Myocardial Infarction

ST-Segment Yükselmeli Miyokart Enfarktüsünde Kronik Total Oklüzyonu Öngörmede Nötrofil/Lenfosit ve Trombosit/Lenfosit Oranı

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Abstract

Background: Neutrophil to lymphocyte ratio (NLR) and platelet to lymphocyte ratio (PLR) are new systemic inflammation markers and predictor of adverse cardiovascular outcomes. Approximately 10% of patients with acute ST-segment elevation myocardial infarction (STEMI) undergoing primary percutaneous coronary intervention (PPCI) have chronic total occlusion (CTO) of the non-infarct-related artery. The presence of concurrent CTO is associated with short- and long-term morbidity and mortality. Here, we aimed to investigate the relationship of NLR and PLO with coexistent CTO in STEMI patients.
Materials and Methods: Ninety consecutive STEMI patients with concurrent CTO were included in the study group and 100 STEMI patients without CTO were included in the control group retrospectively. The relationship between inflammatory markers and concurrent CTO in STEMI was analyzed.
Results: STEMI patients with concurrent CTO had increased NLR, PLR, C-reaktive protein and troponin while decreased glomerular filtration rate, left ventricular ejection fraction (LVEF) in comparison with patients without CTO. In multivariate analysis, NLR (p=0.002), PLR (p=0.042), CRP (p=0.002), hypertension (p<0.001), Hyperlipidemia (p=0.002) and LVEF (p=0.012) were found to be the independent predictors for the presence of concurrent CTO. In the ROC (Receiver Operating Characteristic) curve analysis, ≥ 5.6 and ≥ 164 cut-off values were determined for NLR and PLR in detecting concurrent CTO in STEMI.
Conclusions: PLR and NLR, simple and easily calculated laboratory parameters, may permit prediction of concurrent CTO in patients with STEMI.

Key Words: STEMI, Non-infarct artery chronic total occlusion, Neutrophil/lymphocyte ratio (NLR), Platelet/lymphocyte ratio (PLR)

Öz.

Amaç: Nötrofil/lenfosit oranı (NLO) ve platelet/lenfosit oranı (PLO), yeni sistemik inflamasyon belirteçleri ve olumsuz kardiyovasküler sonuçların öngörücüleridir. Primer perkütan koroner girişime (PPKG) alınan, akut ST-segment yükselmeli miyokart enfarktüslü (STYME) hastaların yaklaşık %10'unda enfarktla ilişkili olmayan arterde kronik total okluzyon (KTO) mevcuttur. Eş zamanlı KTO varlığı, kısa ve uzun dönem morbidite ve mortalite ile ilişkilidir. Biz burada, NLO ve PLO' nun STYME hastalarında eş zamanlı KTO ile ilişkisini araştırmayı amaçladık.
Materyal ve Metod: STYME ile başvuran, eşzamanlı başka damarda KTO'su olan 90 hasta çalışma grubu ve KTO'su olmayan 100 STYME'li hasta ise kontrol grubu olarak geriye dönük olarak ardışık bir şekilde alındı. İnflamatuvar belirteçler ve STYME'de eşzamanlı KTO arasındaki ilişki analiz edildi.
Bulgular: Eşzamanlı KTO'su olan STYME'li hastalarda, KTO'su olmayan hastalara kıyasla NLO, PLO, C-reaktif protein ve troponin artarken, glomerüler filtrasyon hızı, sol ventrikül ejeksiyon fraksiyonu (SVEF) azaldı. Çok değişkenli analizde NLO (p=0.002), PLO (p=0.042), CRP (p=0.002), hipertansiyon (p<0.001), hiperlipidemi (p=0.002) ve SVEF (p=0.012) parametreleri eşzamanlı KTO varlığı için bağımsız öngörücüler olarak bulundu. ROC (Receiver Operating Characteristic) eğrisi analizinde STYME'de eşzamanlı KTO'yu saptamada NLO ve PLO için sırasıyla ≥ 5.6 ve ≥ 164 kestirim değerleri saptandı.
Sonuç: Basit ve kolay hesaplanan laboratuvar parametreleri olan PLO ve NLO, STYME'li hastalarda eşzamanlı KTO'nun öngörülmesine yardımcı olabilmektedir.

Anahtar kelimeler: STYME, Enfarkt dışı arter kronik total oklüzyonu, Nötrofil/lenfosit oranı (NLO), Platelet/lenfosit oranı (PLO)

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Introduction

Early primary percutaneous coronary intervention (PPCI) is an effective treatment strategy in patients with acute ST-segment elevation myocardial infarction (STEMI) and associated with successful outcomes. Primary percutaneous coronary intervention is superior from thrombolytic therapy with respect to morbidity and mortality rates (1). Multivessel disease (MVD) is detected in 40-60% of the STEMI patients whereas chronic total occlusion (CTO) in non-infarct-related artery (IRA) is present in 10% of these patients (1,2). Last studies showed that the prognostic effect of multivessel disease on short and long period mortality after primary PCI could be due to present a coexistent CTO (3,4–8). In the PPCI, the existence of a non-infarct related artery CTO was detected to be related with one year mortality independently, but MVD was not related (8). In a study, the existence of CTO in non-IRA was shown to be related with poor prognosis; however, after recovery of left ventricule ejection fraction (LVEF) and renal functions, one-year survival was dependent on the presence of MVD, irrespective of presence CTO (8,9). Furthermore, the independent clinical predictors for CTO were cardiogenic shock, prior myocardial infarction (MI), age> 65 years, and history of angina (9). Atherosclerosis is related with low stage systemic inflammatory effect in which leukocytes perform a critical role. Leukocytes play a role in thrombus formation alongside atherosclerosis. Neutrophil/lymphocyte ratio (NLR) is the most powerful white blood cell (WBC) predictor of bad results. NLR is a systemic inflammatory indicator that has been connected to mortality and morbidity in numerous cardiac diseases such as decompensated heart failure, stable coronary artery disease, and acute coronary syndrome. Partial lymphopenia conversely thinks the cortisol stimulated stress reaction (10). Platelets are also involved in the advancement of atherosclerosis. Platelets interconnect with endothelial cells, leukocytes and secrete the inflammatory agents that reason to adhesion and transmigration of monocytes (11,12). These monocytes promote to inflammatory activities in the vessel wall increasing atherosclerotic lesions (13). So, we hypothesized and evaluated that platelet/lymphocyte ratio (PLR) and NLR might have an association with the presence of concurrent CTO in STEMI patients.

Materials and Methods

Study population

This study was designed retrospectively. We evaluated retrospectively 1286 consecutive patients who were admitted with first STEMI and underwent PPCI within 12 hours of symptom onset. We detected non-IRA CTO lesions in 238 patients. The patients with active infection (n=21), chronic inflammatory disease (n=13), known malignancy (n=5), cardiogenic shock (n=3), patients with steroid therapy (n=11), end-stage liver and renal failure patients (n=18), no WBC data (n=42), history of PCI (n=24) and history of coronary artery bypass greft patients (n=11) were kept out from the study. After all, 90 STEMI patients with concurrent CTO (group 1) and age, sex suitable 100 STEMI patients without CTO as a control group (group 2) were added in our study. The study was confirmed by the corporate ethics committee (ethics committee of the Faculty of Medicine, Kars Kafkas University (Date:14/08/2013-No: 2013/49)) and adhered to the Declaration of Helsinki.

The baseline characteristics of patients such as age, gender, history of hypertension (HT), diabetes mellitus (DM), hyperlipidemia (HL), smoking, family history and total ischemic time were saved. Also, patients were evaluated for blood pressure, heart rate, Killip class, existence of cardiogenic shock, history of using drug, laboratuvary parameters (such as creatinine, glucose, lipid, and hematologic parameters). All patients were evaluated with transthoracic echocardiography within 24 hours after the coronary event. Left ventricular ejection fraction was measured using the biplane Simpson's technique. Echocardiographic parameters were evaluated in accordance with the guidelines of the American Society of Echocardiography (14). Venous blood was taken for laboratory analysis from all patients on admission to the hospital. Hemoglobin, White blood cell, neutrophil, lymphocyte, platelets, NLR and PLR were obtained with Coulter LH 780 Hematology Analyzer (Beckman Coulter Ireland Inc, Mervue, Galway, Ireland) as complete blood count (CBC).

Definitions

STEMI was described as existence of ST-segment elevation ≥1 mm in two or more continuous electrods (≥2 mm for V1-V3), ongoing myocardial ischemia ≥30 min, new beginning left bundle branch block or true posterior infarction. CTO was described as existence of TIMI 0 or 1 flow stage in non-infarcted coronary artery, not associated with acute coronary event (15). CTOs inside branches of major epicardial coronary arteries were recorded according to its main branches such as left anterior descending (LAD) coronary artery, right coronary artery (RCA) and circumflex artery (CX). The distinction between the critical lesion for STEMI and CTO was arised from morphology of the occlusion (absence of new thrombus, presence of bridge, septal or epicardial collaterals) and a possible history of previous myocardial infarction. Hypertension was described as systolic blood pressure> 140 mmHg and/or diastolic blood pressure was >90 mmHg at least twice or if the patient was receiving antihypertensive drugs. Smoking was described as current smoking. Diabetes mellitus was described as previous taking antidiabetic drug or insulin or having fasting glucose values above 126 mg/dl. Hyperlipidemia was described as LDL>100 mg/dl, TG> 200 mg/dl, or taking current statin therapy. Cockcroft-Gault formula was used for calculating the estimated glomerular filtration rate (eGFR) (16).

Coronary angiography and primary PCI

All patients underwent PPCI within half an hour after admis-

sion. All patients were immediately loaded with acetylsalicylic (300 mg) and clopidogrel (600 mg) and were transferred to angiography laboratory for PPCI. Due to the equality of the groups and accessibility, all patients were loaded with clopidogrel. Heparin (100 IU/kg) was given before PCI, but the glycoprotein IIb/IIIa inhibitor (only tirofiban) was used according to the choice of cardiologist. All angiographic approaches were made using the femoral way with a 7-Fr guiding catheter. The IRA was only target of the procedure. Direct stenting was applied if possible; balloon predilatation was applied for unsuitable lesions. Post-dilatation was applied the cardiologist choice after evaluated the final stent position and angiographic view. Thrombolysis in myocardial infarction (TIMI) flow stage was evaluated for all patients before the procedure. Coronary collateral circulation (CCC) was evaluated according to the Rentrop classification (17). According to; Grade 0 staged as no flow; Grade 1 staged as flow of side vessels via collateral roads without visualization of the epicardial area; Grade 2 staged as incomplete flow of the epicardial big coronary artery via collateral roads; Grade 3 staged as full flow of the epicardial big coronary artery. The patients were seperated into impaired CCC (Group-1, Rentrop grades 0–1) or good CCC (Group-2, Rentrop grades 2–3). After PCI, antiaggregant (clopidogrel, acetylsalicylic acid) and other treatments were given according to the guidelines. All angiographic images we

Statistical analysis

Continuous parameters are given as mean ± standard deviation, and categorical parameters are given as percentage. Student-t test or Mann-Whitney-U test or Chi-square tests were applied for compare between the two groups, as suitable. The Kolmogorov Smirnov test was applied for evaluation the normality of dispersion of continuous parameters. One way ANOVA was performed to compare NLR and PLR ratio according to vessel types in patients with concurrent CTO. Multivariable logistic regression test was made to define the independent predictors of coexistent CTO in STEMI patients; the parameters were fitted from variables found to have marginal relations with it on univariate testing ($p<0.05$). Receiver operating characteristic (ROC) test was applied to determine the cut-off value of NLR and PLR in estimation of concurrent CTO in STEMI patients. Comparison of area under curves (AUC) was made by MedCalc program, version 7.3.0.1 (MedCalc Software, Belgium). Relationship between parameters were made using Pearson or Spearman correlation. The P value of <0.05 was accepted important. All other analyses were made with SPSS 15.0 statistical program (SPSS Inc., Chicago, IL, USA).

Results

A total of 190 STEMI patients (mean age was 67.5 ± 11.8 years and male ratio was 81.1%) were included the study. There was no variation among the groups about age and gender. The baseline clinic and angiographic parameters of groups are presented in Table 1. The prevalence of single and two-vessel CTO in the non-IRAs was 8% and 0.5%, respectively. The patients with concurrent CTO exhibited higher frequency of hypertension, hyperlipidemia, NLR, PLR, WBC, CRP, troponin; as well as lower estimated GFR and LVEF in comparison with patients without CTO (Table 1).

The median total ischemic time was 4±2.1 hours for the patients who underwent primary PCI and there was no statistical variation among the groups (4.2±2.2 vs 3.8±2, p = 0.237). Of the 90 patients with concurrent CTO, target vessels involving CTO were RCA in 48.9 %, followed by Cx in 30% and LAD in 28.9% of the cases. Six patients (7.8%) had two-vessel CTOs. Coronary stent implantation was made in 71 patients with concurrent CTO, whereas 18 patients underwent elective surgery, and 1 patient underwent medical therapy. In-hospital mortality rate was 11.6% (22 patients), which included 18 STEMI patients with concurrent CTO. Both NLR and PLR were importantly higher in subjects with in-hospital mortality, when compared to those who survived (11 ± 7.7 vs 7.5 ± 5, p=0.042 and 296 ± 163 vs. 258 ± 335, respectively, p=0.012). The patients with good CCC were detected importantly higher NLR values (8.1±5.5 vs 6.8 ± 5.3, p=0.042) in comparison with the patients with impaired CCC.

The patients with concurrent CTO had importantly higher NLR values (10.1 ± 5.7 vs 5.9 ± 4.3, p<0.001) and PLR values (353 ± 389 vs. 181 ± 212, p<0.001), compared to those without CTO (Table 1). In one way ANOVA analysis, which compared the NLRs and PLRs according to vessel types in patients with concurrent CTO, no statistical association was observed between the vessel type and these two parameters (p = 0.545 and p = 0.138, respectively). Multivariable logistic regression test was applied for detecting the independent parameters for coincident CTO. The parameters that were found to be important in the univariate test (NLR, PLR, estimated GFR, HT, CRP, HL, WBC, troponin, LVEF) were added in the multivariate model. Between those, NLR, PLR, CRP, HT, HL and LVEF were found independent predictors for concurrent CTO in patients with STEMI (p<0.05) (Table 2).

Receiver-operating characteristic (ROC) curve evaluation was made to determine the best cut-off threshold of NLR and PLR in the prediction of concurrent CTO. NLR value ≥ 5.6 was obtained an AUC (Area Under the Curve) value of 0.737 (95% CI 0.667-0.808) (p<0.001) and PLR value ≥ 164 was obtained an AUC value of 0.804 (95% CI 0.743-0.865) (p<0.001) (Figure 1). Moreover, NLR of ≥ 5.6 value showed a sensitivity of 73% and specificity of 63% and PLR of ≥ 164 value showed a sensitivity of 80% and specificity of 68% for the prediction of concurrent CTO (Figure 1).

We compared both AUC of ROC curves for define the most proper parameter, and PLR ratio was found more accurate against the NLR in prediction of concurrent CTO in STEMI patients (p=0.037) (Figure 2). There was a mild correlation among NLR and eGFR (r =-0.241 p=0.001) and a moderate correlation among NLR and PLR (r = 0.393, p<0.001) (Figure 3).

Table 1. Baseline clinical and angiographic characteristics of the groups

Variables	STEMI with concurrent CTO (n=90)	STEMI without CTO (Control group) (n=100)	P value
Age (years)	68 ± 11	66 ± 12	0.196
Sex, Male n (%)	74 (82.2)	80 (80)	0.145
Diabetes mellitus, n (%)	18 (20)	11(11)	0.085
Hypertension, n (%)	72 (80)	52 (52)	<0.001
Hyperlipidemia, n (%)	50 (55.6)	41 (41)	0.045
Smoking, (%)	16 (17.8)	25 (25)	0.227
Total ischemic time, hours	4.2 ± 2.2	3.8 ± 2	0.237
LVEF (%)	41.9 ± 7.4	46.1±8	<0.001
Rentrop grade 0-1, (%)	72.2	64	0.226
White blood cell count, (K/uL)	12.3 ± 3	11 ± 2.8	0.003
Neutrophil/Lymphocyte ratio (NLR)	10.1 ± 5.7	5.9 ± 4.3	<0.001
Platelet/Lymphocyte ratio (PLR)	353 ± 389	181 ± 212	<0.001
Estimated GFR, mL/min/1.73 m²	74.2 ± 23.5	82.7 ± 23.6	0.014
LDL-Cholesterol, mg/dl	135 ± 43	141 ± 39	0.364
HDL-Cholesterol, mg/dl	37 ± 10	39 ± 9	0.155
Triglyceride, mg/dl	140 ± 78	123 ± 69	0.631
CRP (mg/l)	4 ± 3.9	2.8 ± 2.7	0.028
TIMI 0-1, (%)	67.8	77	0.155
IRA			
LAD, (%)	44.4	41	0.527
Cx, (%)	23.3	17	0.577
RCA, (%)	31.1	39	0.256

CRP: C reactive protein, IRA: Infarct related artery, GFR: Glomerular filtration rate, LVEF: Left ventricular ejection fraction, NLR: Neutrophil lymphocyte ratio, PLR: Platelet lymphocyte ratio, TIMI: Thrombolysis in myocardial infarction

Table 2. Independent predictors of concurrent CTO in multivariate analysis

Variables	Univariate OR and 95% CI	P value	Multivariate OR and 95% CI	P value
LVEF	0.985 (0.972-0.997)	<0.001	0.935 (0.8887-0.985)	0.012
NLR	1.194 (1.111-1.284)	<0.001	1.168 (1.058-1.289)	0.002
PLR	1.005 (1.003-1.008)	<0.001	1.002 (1.000-1.004)	0.042
CRP (mg/l)	1.119 (1.020-1.228)	0.028	1.209 (1.071-1.366)	0.002
GFR (mL/min/1.73m²)	0.985 (0.972-0.997)	0.014	0.998 (0.982–1.014)	0.796
HT	3.692 (1.930-7.063)	<0.001	5.128 (2.203-11.936)	<0.001
HL	1.799 (1.011-3.200)	0.045	3.438 (1.583-7.465)	0.002

CI: Confidens interval, CRP: C reactive protein, GFR: Glomerular filtration rate, HL: Hyperlipidemia, HT: Hypertension, LVEF: Left ventricular ejection fraction, NLR: Neutrophil lymphocyte ratio, PLR: Platelet lymphocyte ratio, OR: Odds ratio.

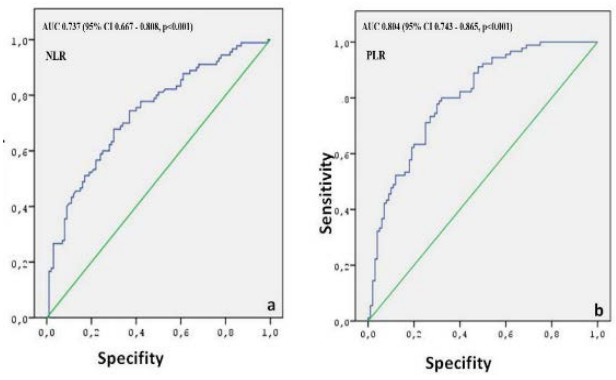

Figure 1. ROC analyses of NLR (a) and PLR (b) in prediction of concurrent CTO in patients with STEMI patients. (NLR= Neutrophil/lymphocyte ratio, PLR= Platelet/lymphocyte ratio, CTO= Chronic total occlusion)

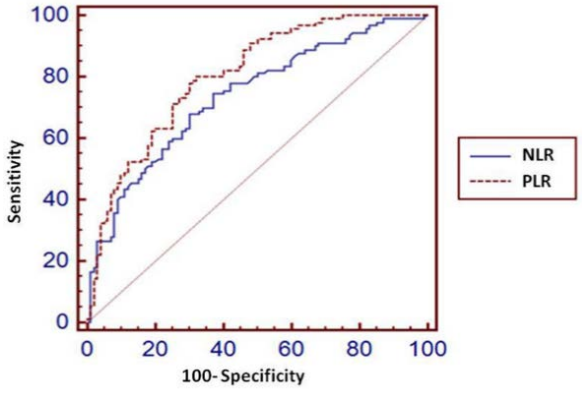

Figure 2. Comparasion of Receiver-operating characteristic (ROC) curves of NLR and PLR for identification of patients with concurrent CTO. (NLR= Neutrophil/lymphocyte ratio, PLR= Platelet/lymphocyte ratio, CTO= Chronic total occlusion)

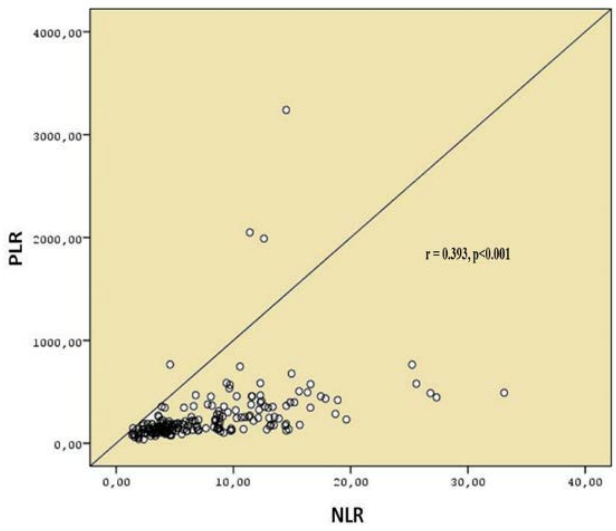

Figure 3. The graphic showing correlation analysis between NLR and PLR. (NLR= Neutrophil/lymphocyte ratio, PLR= Platelet/lymphocyte ratio).

Discussion

In our study, we evaluated the association among NLR, PLR and concurrent CTO in STEMI patients. This study demonstrated that existence of concurrent CTO in STEMI patients related with an increased in inflammatory markers such as NLR and PLR and had a more frequent incidence of hypertension, hyperlipidemia and a lower LVEF and eGFR. These parameters, except eGFR, were independent predictors for concurrent CTO in STEMI patients.

Prior studies have shown a strong association among higher inflammatory parameters and raised mortality in both healthy persons and patients with symptomatic and/or asymptomatic coronary artery disease (18,19). White blood cell subtypes such as neutrophils, lymphocytes, and monocytes are identified as inflammatory agents in the cardiovascular diseases and are played an important role in inflammatory response (20). Neutrophils increase in early phase of acute inflammation and involve with adaptive myocardial infarct healing, whereas lymphocytes play specific role in late phase of immune response. The reduction of lymphocytes in inflammation was thought due to stress-related steroid exposure and increased apoptosis (21). Neutrophil to lymphocyte ratio was displayed more powerful predictor than leukocyte values for inflammation (22). It was also related with morbidity and mortality in heart failure, stable or unstable coronary artery diseases (10). Recently published studies showed that PLR, a novel inflammatory marker, predicted long-time mortality in patients with malignancies and unstable atherosclerotic processes (23,24). Atherosclerosis is a disease that progresses with chronic inflammatory events and appears with thrombotic complications (25). Platelets play an important role in progression of atherosclerosis. Platelets effect the endothelial cells, leukocytes and extricate the inflammatory agents providing to adhesion and transmigration of monocytes (11,12). Therefore, increased PLR due to elevated platelet and reduced lymphocyte counts might be related with increased vascular complications in STEMI patients.

The previous studies showed that existence of CTO in a non-IRA was associated with poor prognosis in patients undergoing primary PCI for acute STEMI (3,5,29). If distal coronary flow of CTO depended on collateral blood flow from IRA, the infarct area related IRA will spread more wide myocardial area. In addition, presence of concurrent CTO in the non-infarct-related artery is higher in terms of cardiovascular risk factors and comorbidities. Increased mortality in this population could be related to low PCI success (6). So, some simple hematological parameters could permit interpretation of PCI outcomes and mortality risk preprocedurally. However, time should not be wasted waiting for the results of hematological parameters in STEMI.

This is the first study that showed the impact of hematological parameters such as NLR and PLR, in estimation of concurrent CTO for STEMI patients. Previous studies showed that existence of concurrent CTO was related with impaired left ventricular functions in STEMI patients when compared to those without concurrent CTO (3,5). The most likely mechanism may be that patients with concurrent CTO, lose of their potential coronary collaterals in the coronary artery, resulting in less blood supply to the infarct-related artery and larger infarct size. Lee et al. (27) have reported that increased neutrophil numbers predict a higher infarction area, worse angiographic results, and poor short-time prognosis in patients with STEMI. Tanrıverdi et al. (28) showed that STEMI patients with NLR ≥ 5.47 had significantly higher infarct area and NLR ≥ 5.47 was an independent predictor of in-hospital mortality. The results of our study are compatible with these findings. Elevation in NLR in STEMI patients with concurrent CTO is probably due to association between large infarction size and increased neutrophil count. Buyukkaya et al. (28) demonstrated that existence and severity of metabolic syndrome was correlated with changes in NLR. In our study, STEMI patients with concurrent CTO were found to have increased prevalance of atherosclerotic and metabolic syndrome risk factors (HT, HL) except diabetes. So, the association between elevated NLR and presence of concurrent CTO may also be attributed to higher frequency of metabolic syndrome risk parameters in this group (9). The poor prognostic impact of NLR in STEMI patients, which was shown in previous studies, may be stemmed from the relationship between NLR and existence of CTO in non-IRA. Chronic total occlusion was related with raised mortality in STEMI patients. Data about PLR and its relationship with coronary events are lacked. Several cytokines such as interleukin (IL)-1, IL-3 and IL-6 which are secreted in pro-inflammatory phase of systemic inflammation stimulate the production of megakaryocytes, lead to increase in platelet counts. Some studies showed that increased platelet counts are related with development of atherosclerotic processes and related complications. Morever, platelets induce the leukocyte differentiation for

more proadhesive and promigratory phenotype, and leukocytes produce mediators that more activate the platelets. It is important to know that the relation among platelets, leukocytes and endothelial cells are frequently two-way (30,31). Accordingly, in current study there was moderate correlation among PLR and NLR. Platelet to lymphocyte ratio, mediator of acute inflammatory response in atherosclerotic processes, was elevated in STEMI patients with concurrent CTO and was related with larger infarct size. The superiority of PLR over NLR in prediction of presence concurrent CTO in STEMI patients was showed in ROC curve analysis. This result may have been stemmed from higher sensitivity of PLR in promoting inflammatory response (32). Cicek and et al (33) showed together of PLR and NLR can be helpful for the estimation of in-hospital and long-time mortality in STEMI patients undergoing primary PCI. Again, Turkmen et al. (34) demonstrated that PLR was a better predictor of systemic inflammatory response in uremic patients compared to NLR. In our study, mean eGFR was within normal limits in both groups; however, the group with CTO had relatively worse renal functions. This may explain why PLR was a better predictor for CTO rather than NLR in STEMI patients.

In this study the prevalence of single and two-vessel CTO in the non-IRA was 8 % and 0.5%, respectively. This was appropriate with data provided in prior studies with STEMI patients (3–6). As the existence of CTO in STEMI patients was related with worse prognosis and PCI failure, it was significant to suspect the existence of CTO in non-infarct-associated artery prior processing to angiography in patients refering with STEMI. Therefore, we performed a basic, quick test, complete blood count, without wasting time, to obtain NLR and PLR before primary PCI, which could predict the presence of concurrent CTO. Furthermore, we described several basic clinical predictors for the existence of CTO in non-infarct associated artery. We found out that history of HT and HL, and lower LVEF were related with the existence of concurrent CTO.

Limitations

The retrospective design of our study was unable to assess the longitudinal relationships. We could not investigate the benefit of recanalization of the concurrent CTO lesion and short or long period survival. In many patients, the time of the CTO could not be detected obviously, but it was considered after attentive evaluation of the plaque formation to be ≥3 months old and, therefore, it assumed as correct CTO (15). But there was a possibility of the inclusion the patients who had the occlusion for less than 3 months. Additionally, the patients with prior MI and CABG were excluded from the study. So, the frequency of coexistent CTO in STEMI patients could be underestimated as some of these patients could had probably CTO.

Conclusion

Several risk stratification systems have been developed for patients who were referred to hospital with STEMI. The presence of CTO at non-IRA has been detected to indicate the high-risk patients due to increased mortality and low PCI success. PLR and NLR are simple laboratory parameters that could be easily calculated. These parameters can permit prediction of high short and long period mortality and low procedural success before the primary PCI in STEMI patients.

Ethical Approval: The protocol was accepted by the ethics committee of the Faculty of Medicine, Kars Kafkas University (Date:14/08/2013) No: 2013/49) and adhered to the Declaration of Helsinki.

Author Contributions:
Concept: T.G, F.B.
Literature Review: T.G, F.B, M.A.
Design : T.G, F.B.
Data acquisition: T.G, F.B.
Analysis and interpretation: T.G, F.B, M.A.
Writing manuscript: T.G, F.B, M.A.
Critical revision of manuscript: T.G, F.B, M.A.

Conflict of Interest: *There are no conflicts of interest to declare. All authors have participated in the work and could publicly defend its contents and have read the manuscript prior to its submission for publication and agree with its contents.*

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