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ORIGINAL ARTICLE / ÖZGÜN ARAŞTIRMA

The relationship between neutrophil to lymphocyte ratio and SYNTAX score in patients with ST-segment elevation myocardial infarction

ST-segment yükselmeli miyokard infarktüsünde nötrofil lenfosit oranı ve syntax skoru arasındaki ilişki

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ABSTRACT

Objective: We aimed to assess relationship between the severity of coronary atherosclerosis assessed by SYNTAX score (SS) and neutrophil to lymphocyte ratio (NLR) in patients with ST elevation myocardial infarction (STEMI.

Methods: After accounting for exclusion criteria, a total of 291 patient with STEMI in whom primary percutaneous coronary intervention was performed were retrospectively included (216 male, 75 female; mean age 61.6+14.0 years). Total and differential leukocyte counts and other biochemical markers were measured at admission. Patients were categorized into tertiles on the basis of SS. Monitoring for major adverse cardiac events (MACEs) was performed during the in hospital follow-up period.

Results: The SS high group leukocyte (p=0.009), neutrophil (p=0.008), NLR (p=0.048), peak troponin (p<0.001), peak CK-MB (p=0.001) lactate dehydrogenase (p=0.005), aspartate aminotransferase (p=0.004) values were significantly higher compared with SSlow and SSmid groups. SS was increased, left ventricular ejection fraction was decrease (p<0.001) and left ventricular systolic diameter was increased (p=0.007). The in-hospital death rate and MACEs were greater in the high SS group than in the other groups (p<0.001 both of).

Conclusion: We found that high NLR was significantly and correlated increased with SS. In addition, high SS were significantly associated with increased in-hospital MACE and in-hospital death. Further prospective studies assessing the predictive role of both SS and NLR in conjunction for risk stratification might improve risk prediction in patients with STEMI. *J Clin Exp Invest 2014; 5 (2): 211-218*

Key words: Neutrophil to lymphocyte ratio, Syntax score, STEMI

ÖZET

Amaç: Biz bu çalışmada STEMİ hastalarında nötrofil lenfosit oranı (NLO) ve Syntax skoru (SS) ile değerlendirilen koroner aterosklerozun şiddeti arasındaki ilişkiyi ST yükselmeli miyokard infarktüsü (STEMİ)'ünde değerlendirmeyi amaçladık.

Yöntemler: Dışlama kriterleri sonrası primer perkütan koroner girişim uygulanan geriye dönük olarak toplamda 291 STEMI hastası çalışmaya alındı. (216 erkek,75 kadın, ortalama yaş 61,6±14,0 yıl). Toplam ve diferensiyel lökosit sayımı ve diğer biyokimyasal belirteçler hasta kabulünde alındı. Hastalar SS göre üç tertile ayrıldı. Majör istenmeyen kardiyak olaylar (MİKO) için izleme hastanede yatış döneminde yapıldı

Bulgular: SSyüksek grup, lökosit (p=0,009), nötrofil (p=0,008), NLO (p=0,048), zirve troponin (p<0,001), zirve CK-MB (p=0,001) laktat dehidrogenaz (p=0,005), aspartat aminotransferaz (p=0,004) değerleri SSdüşük ve SSorta grupları ile karşılaştırıldığında anlamlı derecede yüksek saptandı. SS artığında, Sol ventrikül ejeksiyon fraksiyonunda düşme (p<0,001) ve sol ventrikül sistolik çapında (p=0,007) artış izlendi. Hastane içi ölüm oranı ve MİKO diğer gruplara (p<0,001 her ikisi için) oranla SS yüksek grubunda daha fazlaydı.

Sonuçlar: Çalışmamızda NLO artışı ile SS artışı arasında anlamlı bir ilişki bulduk. Buna ek olarak, SS artışı ile hastane içi MİKO ve hastane içi ölüm arsındaki artan önemli bir ilişki saptadık. İleriye dönük çalışmalarda NLO ve SS birlikte STEMİ hastalarının sınıflandırılmasında risk tahmininde değerlendirilebilir.

Anahtar kelimeler: Nötrofil lenfosit oranı, Syntax skoru, STEMI

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INTRODUCTION

Coronary artery disease (CAD) and acute myocardial infarction (MI) are a major cause of death and morbidity worldwide. Atherosclerosis is the major cause of cardiovascular disease (CVD) [1-3]. Multiple pathophysiological factors influence this atherosclerotic process and one of the most important factors is inflammation [4]. Inflammation plays a significant role in initiating atherosclerosis and facilitating its progression [5].

White blood cell (WBC) count and the neutrophil and lymphocyte have been investigated as inflammatory markers to predict cardiovascular outcomes in patients with CAD [6,7] The neutrophil to lymphocyte ratio (NLR) is calculated from the WBC-c and is a novel prognostic marker in patients with CVD [8,9]. High NLR was reported to be associated with increased cardiac mortality in clinically stable patients with CAD [6-7]. Increased NLR was also reported to be associated with the presence and long-term mortality of ST elevation myocardial infarction (STEMI) [10].

The SYNTAX score (SS) is a comprehensive anatomic scoring system based on the coronary angiogram. The SS quantifies the properties of lesion including complexity, morphology, and location in coronary tree and predicts outcome after percutaneous coronary intervention (PCI) in patients with CAD who are undergoing revascularization [11-13]. Also, the SS reflects the pattern of atheroma and the technical difficulty of PCI [12]. It is able to aid revascularization decisions and predicts mortality and morbidity in patients with CAD [11,12]. The relationship between NLR and STEMI has been shown in several studies, but there is little data available about the association of NLR levels with SS. In this study, we aimed to evaluate relationship between the severity of coronary atherosclerosis assessed by SS and NLR in patients with STEMI.

METHODS

Study population

A total of 600 patients that retrospective presented with STEMIs and underwent primary PCIs within 12 hours of symptom onset between January 2012 and March 2013 were included in the study. STEMI was defined based criteria created by the American College of Cardiology and the European Society of Cardiology [14]: an increase in troponin I >1 ng/mL; a new ST elevation as measured from the J-point in 2 or more contiguous leads with leads V1, V2, and V3 measuring at least 0.2 mV or at least 0.1 mV in the remaining leads; during the first 12 hours after symptom onset.

Exclusion criteria from the study were patients with severe liver disease, autoimmune diseases, cancer, hematological disorders, severe valvular disease, inflammatory or infectious diseases, and a history of bleeding diathesis. Patients on the following medications were excluded from the study: corticosteroids, cytotoxic drugs, thrombolytic therapy, glycoprotein IIb/IIIa inhibitors, diuretics, if during the study the patient was not treated with primary PCI, did not follow-up for blood work, and had poor echocardiographic windows, then they were also eliminated from the investigation. After accounting for all of these exclusion criteria, a total of 291 patients remained in study sample. All patients received a complete physical examination, assessment of coronary risk factors, and medical histories and presenting clinical symptoms were also recorded. Patients were evaluated for heart failure prognosis according to Killip clinical examination guidelines [15].

Monitoring for major adverse cardiac events (MACEs) was performed during the in hospital follow-up period. Examples of MACEs were cardiogenic shock, new advanced heart failure, pulmonary edema, complete atrioventricular block (AVB) requiring a temporary pacemaker, severe ventricular arrhythmia and in-hospital mortality during the post-PCI follow-up period. An in-hospital mortality was only considered a MACE if the death was due to myocardial infarction, cardiac arrest, or due to some other cardiac-related cause. Cardiogenic shock was defined as marked and persistent hypotension lasting more than 30 minutes with a systolic arterial pressure less than 80 mmHg with signs of hypoperfusion due to left ventricular dysfunction, right ventricular infarction, or cardiac mechanical complications. New-onset advanced heart failure was diagnosed if the patient qualified for a New York Heart Association functional classification of III or greater. In order for a severe ventricular arrhythmia to be considered a MACE, it needed to occur within 48 hours of admission and the rhythm must have been ventricular fibrillation, ventricular tachycardia, or asystole.

Blood work and echocardiography

Venous blood samples were collected when the patient initially presented to the emergency department or intensive coronary care unit (ICCU) before primary PCI. Hematologic indices were measured by an automated hematology analyzer system (Ab-

bott Cell-Dyn 3700; Abbott Laboratory, Abbott Park, Illinois). Absolute cell counts were utilized to perform subsequent analyses. Baseline NLR was measured by dividing the neutrophil count by the lymphocyte count. Total cholesterol, high-density lipoprotein (HDL), low-density lipoprotein (LDL), triglycerides, glucose and creatinine levels were measured with the Abbott Architect C16000 autoanalyzer (Abbott Laboratory). Creatinine kinase-MB (CK-MB) levels were measured with a Beckman Access analyzer (U.S.A). Troponin I levels were measured with a Beckman Image 800 analyzer (U.S.A).

Transthoracic two-dimensional echocardiography was performed upon admission to the ICCU to determine left ventricular ejection fraction (LVEF), left ventricular systolic diameter (LVSD), left ventricular diastolic diameter (LVDD), and left atrial diameter (Vivid S6, GE Medical Systems, USA).

Syntax Score and Angiographic Analysis

All patients underwent selective coronary angiography using the Judkins technique. Coronary lesions leading to ≥50% diameter stenosis in vessels ≥1.5 mm was scored separately and added together to provide the cumulative SS which was prospectively calculated using the SS algorithm on the baseline diagnostic angiogram [16]. Two experienced interventional cardiologists analyzed the SS; the opinion of a third analyst was obtained and the final judgment was made by consensus in cases of disagreement. The final score was calculated from the individual lesion scores by analysis who were blinded to procedural data and clinical outcome. The Gensini scoring system was used to determine the severity of CAD [17].

Statistical analysis

All analyses were performed with SPSS for Windows version 18.0 (SPSS Inc. Chicago, Illinois, USA). Continuous variables were expressed as the mean plus minus standard deviation (SD), and categorical variables were expressed as percentages. The two sample Kolmogorov-Smirnov test assessed whether continuous variables followed a normal distribution. Comparisons between categorical variables between the SS groups were performed using the $\chi 2$ or Fischer's exact test. Analysis of variance (ANOVA) and Kruskal Wallis test were used in the analysis of continuous variables. A stratified analysis of clinical and laboratory variables was performed according to the tertiles of the SS. The correlation between SS and clinical, laboratory parameters was assessed by the Pearson correlation test. Statistical significance was defined as a p-value less than 0.05.

The study protocol was reviewed and approved by the Ethics Committee in accordance with the Declaration of Helsinki.

RESULTS

The SS classified as tertiles are SSlow <12 (n=84), SSmid >12 and <22 (n=102), and SShigh ≥22 (n=105). The baseline characteristics of the 3 groups are summarized in Table 1. Mean age of SShigh group was higher than SSlow and SSmid groups (P=0.003 for all). Frequency of HT, DM smoking, hyperlipidemia positive family history and previous medical history were similar between the groups. Killip class III-IV were more common in SShigh group patients (p<0.001 for all). Admission heart rate was high in SShigh group patients (p=0.021 for all). Initial laboratory findings of patient groups were compared in Table 2. The SShigh group WBC count (p=0.009 for all), neutrophil count (p=0.008 for all), NLR (p=0.048 for all), peak troponin levels (p<0.001 for all), peak CK-MB levels (p=0.001 for all), lactate dehydrogenase (p=0.005 for all), aspartate aminotransferase (p=0.004 for all) values were significantly higher compared with SSlow and SSmid groups. Echocardiographic and angiographic characteristics are listed in Table 3. SS was increased, LVEF was signifficantly decrease (p<0.001 for all) and LVSD was increased (p=0.007 for all). The Gensini score was higher in the high SS group as compared to the other groups (p<0.001). The in-hospital adverse outcomes are listed in Table 4. The in-hospital death rate was greater in the high SS group than in the other groups (p<0.001). MACEs were more frequent in the high SS group than in the other groups (p<0.001). Similarly, in hospital advanced pulmonary edeme (p=0.032), in-hospital cardiogenic shock (p=0.008), severe ventricular arrhythmia (p=0.002), cardiopulmonary resuscitations (p<0.001), were higher in the high SS group than in the other groups patients. Pearson's correlation analysis revealed significant associations between a high SS and age (r=0.206, p<0.001), NLR (r=0.148, p=0.012) (Fig. 1), peak troponin (r=0.248, p<0.001), peak CK-MB (r=0.200, p=0.001), lactate dehydrogenase (r=0.221, p<0.001), aspartate aminotransferase (r=0.179, p=0.003), killip on admission (r=0.245, p<0.001), number of coronary arteries narrowed (r=0.489, p<0.001), In-hospital mortality (r=0.246, p=0.001) and In-hospital MACE (r=0.258, p<0.001), Gensini score (r=0.661, p<0.001) (Table 5).

Table 1. Demografic Characteristics of Patients in Syntax Score Groups

Variables	SS Low n=84	SS Mid n=102	SS High n=105	p value
Age, years	58.33±12.57	60.75±15.62	65.08±12.84	0.003*
Sex, male, n (%)	66 (31)	76 (35)	74(34)	0.448
Previous history				
Hypertension , n (%)	34 (31)	31 (28)	44 (41)	0.180
Diabetes mellitus, n (%)	20 (29)	26 (38)	23 (33)	0.832
Smoking, n (%)	58 (35)	58 (35)	49 (30)	0.090
Hyperlipidemia, n (%)	3 (21)	5 (36)	6 (43)	0.790
Family history of CAD, n (%)	16 (28)	20 (34)	22 (38)	0.943
Cerebrovascular event, n (%)	5 (33)	3 (20)	7 (47)	0.444
History of PCI, n %	3 (23)	3 (23)	7 (54)	0.386
History of CAD, n (%)	2 (15)	2 (15)	9 (70)	0.039
Previous medication				
Preadmission aspirin use, n (%)	66 (30)	71 (33)	79 (37)	0.364
Preadmission clopidogrel use, n (%)	51 (35)	48 (33)	46 (32)	0.055
Beta blocker, n (%)	7 (24)	9 (31)	13 (45)	0.583
ACE inhibitors, n (%)	8 (25)	11 (34)	13 (41)	0.820
Statin, n (%)	6 (27)	6 (27)	10 (46)	0.603
Preadmission enoxaparine use, n (%)	72 (27)	96 (37)	95 (36)	0.154
Killip on admission				
I-II	83 (99)	99 (97)	90 (86)	<0.001
III-IV	1 (1)	3 (3)	15 (14)	
Admission SBP (mmHg)	126.08±18.91	129.74±24.27	125.19±26.25	0.350
Admission heart rate (bpm)	79.30±14.79	83.83±16.87	86.09±18.06	0.021

*Kruskal Wallis test; Other statics ANOVA test and χ 2 test; Syntax score, SS; PCI,Percutaneous coronary intervention CAD, Coronary artery disease

Table 2. A Comparison of Initial Laboratory Values of Patients in Syntax Score Groups

Laboratory Findings	SS Low n=84	SS Mid n =102	SS High n=105	p value
WBC, K/uL	12.65±4.80	13.65±4.90	14.51±5.44	0.009*
RBC, M/uL	4.83±0.48	4.87±0.57	4.89±0.56	0.806
Hemoglobin, g/dL	13.87±1.50	14.13±1.67	13.94±1.60	0.498
RDW, %	15.75±1.39	16.20±1.71	16.01±1.63	0.166
PDW, NULL	17.87±1.27	17.85±1.01	17.92±1.20	0.542
Platelet count, K/uL	242.29±67.29	257.31±69.66	248.63±59.33	0.213
Lymphocyte count, NULL	1.92±0.97	1.99±1.05	1.96±1.48	0.923
Monocyte count, NULL	0.63±0.25	0.69±0.33	0.67±0.33	0.423
Basophil count, NULL	0.07±0.04	0.08±0.07	0.07±0.05	0.309
Neutrophil count, NULL	9.93±4.37	10.91±4.76	11.82±5.08	0.008*
NLR	6.40±3.66	6.92±4.16	8.20±5.60	0.048*
PLR	153.35±76.47	165.62±99.30	168.13±97.96	0.525
Glucose on admission, mg/dL	161.69±71.15	167.20±75.96	179.84±95.39	0.291
Creatinine on admission, mg/dL	0.86±0.28	0.89±0.21	0.94±0.53	0.320
Lactate dehydrogenase U/L	328.64±156.58	389.06±213.69	461.27±281.40	0.005*
Aspartate aminotransferase U/L	83.24±84.16	105.40±84.49	146.03±158.05	0.004*
Alanin aminotransferaz U/L	35.13±27.82	47.34±61.86	51.37±77.52	0.186
Peak Troponin I, ng/mL	40.34±36.87	62.11±34.15	68.48±35.46	<0.001*

(Önceki sayfadan devam)

Peak CK-MB mass, ng/mL	144.27±114.14	207.61±103.29	210.48±109.46	0.001*
Total cholesterol, mg/dL	175.38±33.43	181.18±39.04	172.34±42.32	0.253
LDL, mg/dL	109.60±28.97	115.55±30.64	114.44±33.18	0.398
HDL, mg/dL	33.35±7.72	35.79±11.37	35.06±9.81	0.236
Triglycerides, mg/dL	163.02±81.15	150.34±99.10	141.47±97.14	0.291

Values are mean ±SD or n (%),*Kruskal Wallis test; Other statics ANOVA test; WBC, White blood cell count; RBC Red blood cell count; RDW, Red cell distribution width; PDW, Platelet distrubition width; NLR, Neutrophyl/lymphocyte ratio PLR, Platelet/lymphocyte ratio LDL, low-density lipoprotein; HDL, high-density lipoprotein; CK, Creatin kinase;

Table 3. A Comparison of Echocardiographic, Angiographic Findings of Patients in Syntax Score Groups

Variables	SS Low n=84	SS Mid n =102	SS High n=105	p value
STEMI location				
Anterior, n (%)	17 (20)	57 (56)	60 (46)	<0.001
Nonanterior, n (%)	67 (80)	45 (44)	45 (549	
Duration of chest pain (hour)	5.41±2.99	6.00±3.36	5.56±3.51	0.443
Admission LVEF (%)	50.07±7.67	43.11±8.96	38.63±9.60	<0.001*
Left ventricular diastolic diameter (mm)	45.96±5.17	46.43±5.35	46.75±5.60	0.644
Left ventricular systolic diameter (mm)	32.12±5.73	33.48±5.87	35.04±6.50	0.007*
Left atrial diameter (mm)	37.09±5.33	35.35±5.06	36.53±5.25	0.028
Culprit lesion				
LAD, n (%)	20 (24)	60 (59)	59 (56)	
RCA, n (%)	49 (58)	29 (28)	30 (29)	<0.001
CX, n (%)	15 (18)	13 (13)	16 (15)	
Number of coronary arteries narrowed				
1 vessel, n (%)	62 (74)	51 (50)	14 (13)	<0.001
>1 vessel n, (%)	22 (26)	51 (50)	91 (87)	
Gensini score	35.21±14.63	57.98±21.23	59.81±29.21	<0.001*
Syntax score	7.51±2.80	16.66±2.82	31.19±9.80	<0.001*

*Kruskal Wallis test; Other statics ANOVA test and χ 2 test; Syntax score, SS LVEF, left ventricular ejection fraction; LAD, left descendan coronary artery; RCA, right coronary artery; CX, circumflex coronary artery

Table 4. A Comparison of İn-hospital Major Adverse Cardiovascular Events of Patients in Syntax Score Groups

Variables	SS Low n=84	SS Mid n =102	SS High n=105	p value
In hospital MACEs, n (%)	9(14)	17(27)	38(59)	<0.001
Advanced heart failure, n (%)	3(18)	7(41)	7(41)	0.574
Advanced pulmonary edema, n (%)	0(0)	4(33)	8(67)	0.032
Cardiogenic shock, n (%)	2(10)	5(24)	14(66)	0.008
Complete AVB requiring transient pacemaker, n (%)	3(20)	4(27)	8(53)	0.358
Serious ventricular arrhythmia, n (%)	0(0)	4(25)	12(75)	0.002
In-hospital mortality, n (%)	1(4)	6(21)	21(75)	<0.001
Cardiopulmonary resusitation , n (%)	1(3)	10(32)	20(64)	<0.001
Time of hospital stay (days)	4.29±2.86	5.29±5.55	5.95±5.06	0.059

Statics ANOVA test and χ 2 test; MACE, major advers cardiovascular event; AVB, atrioventricular block,

Table 5. Pearson Correlations of Patients in Syntax Score

	r	p value
Age	0.206	<0.001
Admission heart rate (bpm)	0.147	0.012
Admission LVEF (%)	-0.392	<0.001
Left ventricular systolic diameter (mm)	0.171	0.006
Killip classification on presentation	0.245	<0.001
WBC K/uL	0.112	0.058
Neutrophil count, NULL	0.112	0.038
Peak Troponin I, ng/mL	0.248	<0.001
Peak CK-MB mass, ng/mL	0.200	0.001
Lactate dehydrogenase U/L	0.221	<0.001
Aspartate aminotransferase U/L	0.179	0.003
NLR	0.148	0.012
Number of coronary arteries narrowed	0.489	<0.001
In-hospital mortality	0.246	0.001
In-hospital MACE	0.258	<0.001
Gensini score	0.661	<0.001

WBC, White blood cell count;LVEF, Left ventricular ejection fraction

NLR, Neutrophyl/lymphocyte ratio MACE, major advers cardiovascular event

DISCUSSION

We showed that NLR was significantly associated with SS in patients STEMI. Also, we demonstrated that on admission heart rate, LVEF, LVSD, killip class, peak troponin, peak CK-MB, lactate dehydrogenase, aspartate aminotransferase, multivessel disease, in-hospital mortality and in-hospital MACE were significantly associated with SS in STEMI patients. Moreover SS was signifficantly correlated with NLR on admission heart rate, LVEF, LVSD, killip class, peak troponin, peak CK-MB, lactate dehydrogenase, aspartate aminotransferase, multivessel disease, in-hospital mortality and in-hospital MACEs. Atherogenesis is an active inflammatory process [5] with vitally acting leukocytes [18]. Neutrophils play role either in adaptive infarct healing or in leukocyte platelet aggregate formation and in reperfusion injury in the setting of acute coronary syndromes (ACS) [19,20]. Several previous studies have assesed the relation of total and differential WBC count and presence and prognosis of CAD. White blood cell count, neutrophil count, and NLR were reported as an independent prognostic predictor both in acute MI [21,22] and in stable CAD [7]. Lymphocytes also play a important role in modulating the inflammatory response in atherosclerotic process [23] and comparative lymphopenia in MI was postulated as a stress response mediated by increased endogenous cortisol [24,25].

Recent studies have shown that high neutrophil counts are associated with poorer angiographic outcomes, larger infarct sizes, and worse prognosis in STEMI patients [10,22,26]. Several studies have demonstrated that NLR is a marker of CVD prognosis [27]. Previous studies centre on NLR and its association with adverse outcomes in patients with ACSs [28]. Arbel et al. found that increased NLR was associated with increased severity of CAD, thereby providing additive predictive value to conventional risk factors and commonly used biomarkers e.g. C-Reactive Protein and total WBC count [29]. Polat et al. show that increased NLR was signifficantly associated with increased severity of rheumatic mitral valvular disease [30]. Yıldız et al. show that increased NLR was signifficantly associated with ventricular premature contraction existence [31]. Acet et al. show that increased NLR was indepently and signifficantly associated with severity and duration of atrial fibrillation [9]. Kaya et al. show that increased NLR was signifficantly associated with severity of stable CAD with gensini score [8]. Yıldız et al demonstrate that NLR was signifficantly associated with the coronary no-reflow phenomenon in STEMI patients [32]. Oylumlu et al. show that NLR was new inflammatory markers in pre-eclampsia [33]. Yıldız et al. show that detected a significant association between the NLR and low funtional capacity, both of which has predictive and prognostic value in patients with heart failure [34]. Yıldız et al. show that myocardial bridge is associated with elevated NLR, which is used to assess inflammatory status of the body [35]. Çil et al. show that there was no association between NLR and coronary collateral circulation with diagnosis of CAD and detected significant stenosis or occlusion at least one of the coronary arteries [36]. Increase in the severity of CAD with respect to SS was associated with increased neutrophils and finally with increased NLR in our study. Similar to the current study, a relationship between NLR and severity of CAD was reported in patients with stable CAD [6,7]. However, a similar independent relationship was investigated a little in STEMI. To our knowledge, only one study by Şahin et al. has investigated the relationship between NLR and

SS [37]. They investigated the relationship between NLR and SS in STEMI patients. They showed that NLR was significantly increased with SS. Similarly, we have shown that NLR is significantly associated with SS. Multiple ACS studies now support the use of NLR as an admission biomarker, which can be used to determine prognosis [38]. NLR can be readily calculated at point of care, thereby facilitating short and long-term risk prediction for STEMI patients, even prior to revascularization.

Conclusion: We found that high NLR was significantly and correlated increased with SS in patients with STEMI. In addition, we found that high SS were significantly associated with increased in-hospital MACE and in-hospital death. Further prospective studies assessing the predictive role of both SS and NLR in conjunction for risk stratification might improve risk prediction in patients with STEMI.

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