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Systemic Coagulation Inflammation Index Associated With Bleeding in Acute Coronary Syndrome

Aim	Assessment of the inflammatory component of acute coronary syndrome (ACS) and the degree of activation of the coagulation cascade may provide prognostic information. The systemic coagulation-inflammation index (SCI) assesses both inflammation and the coagulation system, and it has also been found to be associated with clinical outcomes. We investigated the relationship between SCI and in-hospital clinical events (acute kidney injury, cardiogenic shock, life-threatening arrhythmia, bleeding) and mortality.
Material and methods	The study included 396 patients aged ≥18 yrs who were hospitalized with a diagnosis of ACS. The SCI was calculated using the formula: platelet count (103/μl) X fibrinogen (g/l)/white blood cell (WBC) count (103/μl). Patients were divided into two groups according to whether their SCI score was >100 or <100, and the relationship between clinical and laboratory characteristics was analyzed accordingly.
Results	The mean age of the patients was 61.4±12.2 years and 78.3% (n=310) were male. The type of ACS was NSTEMI in 56.1% (n=222). The responsible vessel was the left anterior descending artery (LAD) in 42.4% of the patients (n=168). The mean SCI score was 97.5±47.1. WBC, neutrophil, and lymphocyte counts were higher in the SCI <100 group, whereas fibrinogen, C-reactive protein, and platelet count were higher in the SCI >100 group. Bleeding from any cause as an in-hospital complication was significantly higher in patients with SCI >100 ($p<0.05$). Other in-hospital events were not significantly associated with SCI ($p>0.05$).
Conclusions	Bleeding in ACS patients was significantly more common in the group with SCI >100. Thus, SCI may be a useful parameter for predicting in-hospital bleeding complications in ACS. On the other hand, SCI was not associated with mortality and other in-hospital clinical events.

Keywords	Systemic coagulation inflammation index; acute coronary syndrome; ST segment elevated myocardial infarction; non-ST segment elevated myocardial infarction; bleeding
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Introduction

Acute coronary syndrome (ACS) is a clinical entity with various complications and a certain mortality rate. Therefore, data to predict events associated with ACS are of great importance for patient management. The pathophysiology of ACS is characterized by a cascade of inflammation leading to plaque rupture [1]. In addition, platelet activation and activation of the coagulation cascade are important components of the ACS process [2]. The prognosis of ACS is, to some extent, determined by the degree of activation of the inflammatory process and the coagulation system.

Assessment of the inflammatory component of ACS and the degree of activation of the coagulation cascade may provide prognostic information. Previously, high-sensitivity C-reactive protein (hs-CRP) concentrations have been associated with cardiovascular outcomes after ACS [3]. It has been reported that the inflammatory biomarkers, the neutrophil to lymphocyte ratio (NLR) and the platelet to lymphocyte ratio (PLR), can also be used to determine prognosis in ACS [4, 5]. It was also found that a high systemic immune inflammation index (SII) was associated with an increase in cardiovascular death [6]. Increased fibrinogen levels are also associated with increased major adverse cardiovascular events in ACS [7].

The systemic coagulation-inflammation index (SCI), a cost saving, easily calculated parameter that assesses both inflammation and the coagulation system, has also been found to be associated with clinical outcomes in aortic dissection [8]. Due to the presence of common pathophysiological mechanisms, there is limited data on the relationship of this index with clinical outcomes and its applicability in ACS. In the present study, we aimed to investigate the relationship between SCI and in-hospital clinical events and mortality.

Material and methods

Study Population

This study was designed as an observational, cross-sectional study. The study included 396 patients aged ≥18 yrs who were hospitalized with a diagnosis of ACS, i.e., ST-elevation myocardial infarction (STEMI) or non-ST-elevation

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myocardial infarction (NSTMI), and underwent coronary angiography (CAG) at Bursa City Hospital or Bursa Yüksek İhtisas Training and Research Hospital, Bursa, Turkey, between January 2023 and April 2023. Patients with severe liver or kidney disease, active malignancy, severe valvular disease, severe pulmonary disease, bleeding diathesis, or sepsis/septic shock were excluded. Demographic and clinical characteristics, echocardiography, electrocardiography, CAG, and percutaneous coronary intervention (PCI) results were recorded. The required written informed consent was obtained from the patients participating in the study. The study was conducted in accordance with the tenets of the Declaration of Helsinki and with the approval of the Bursa City Hospital Clinical Ethics Research Committee (Decision Number 2023–6/6, 12.04.2023).

Hypertension (HT) was defined as use of antihypertensive medication or systolic blood pressure ≥140 mm/Hg and diastolic blood pressure ≥90 mm/Hg. Diabetes mellitus was defined as use of diabetes medication or fasting plasma glucose ≥126 mg/dl or glycated hemoglobin (HgbA1c) ≥6.5%. Smoking was defined as currently smoking or having stopped smoking in the past year. Hyperlipidemia was defined as use of medication for hypercholesterolemia or low-density lipoprotein (LDL) ≥130 mg/dl (≥3.4 mmol/l) and/or non-high-density lipoprotein (non-HDL) ≥160 mg/dl (≥4.1 mmol/l) [9]. Chronic kidney disease (CKD) was defined as the presence of an estimated glomerular filtration rate (eGFR) of less than 60 ml/min/1.73 m² for 3 mos or longer [10]. Previous heart failure (HF) was identified as a left ventricular ejection fraction (LVEF) <50% with clinical symptoms and signs of heart failure and/or being treated for HF. Chronic obstructive pulmonary disease (COPD) was defined as the requirement for a bi-level positive airway pressure (BiPAP) device at home due to obstructive airway disease or the use of bronchodilators and/or steroids. A previous cerebrovascular event (CVE) was defined as either an ischemic or hemorrhagic event. The SYNTAX I score was determined by totaling the scores of vessels >1.5 cm in diameter and anatomically >50% stenosis on CAG using an online diagnostic tool (http://syntaxscore.org/calculator/syntaxscore/frameset.htm).

Access site complications were defined as any hematoma, pseudoaneurysm, or fistula that occurred at the access site after the procedure. Bleeding was defined as any bleeding that resulted in a decrease in hemoglobin of at least 2 g/dl and/or required blood transfusion. No-reflow was defined as the presence of inadequate myocardial blood flow in an arterial segment in the absence of angiographic evidence of permanent physical obstruction of an epicardial vessel [11]. Acute kidney injury (AKI) was characterized as an increase in serum creatinine >50% within 7 days, an increase in serum creatinine of 0.3 mg/dl (26.5 μmol/l) within 2 days, or oliguria for ≥6 hrs [12]. The diagnosis of cardiogenic shock was based on a systolic blood pressure <90 mmHg for ≥30 min or the need for mechanical/pressure support to maintain this value [13]. Ventricular tachycardia/fibrillation (VT-VF), complete atrioventricular block (AVB), and new atrial fibrillation (AF) were considered arrhythmic complications if observed at any time during hospitalization, i.e., before, during or after the invasive coronary procedures. New HF was described as the development of symptoms and signs of HF after the procedure, without clinical evidence of HF before the acute coronary event. Stent thrombosis was defined as an in-stent thrombosis in association with ACS in the hyperacute, acute or subacute period after stent implantation.

Determination of Systemic Coagulation Inflammation Index

The first peripheral venous blood sample collected during ACS was used for evaluation. Hemogram parameters were measured by fluorescence flow cytometry method using a Sysmex XN instrument. Fibrinogen was measured by the Clauss method using a Roche Cobas T711 instrument. The concentration of CRP in the serum were determined by the immunoturbidimetric method using a Roche Cobas C702 instrument. SCI was calculated using the formula: platelet count (103/μl) X fibrinogen (g/l)/white blood cell (WBC) count (103/μl) [8]. The patients were divided into two different groups according to whether their SCI score was >100 or <100. With reference to the predictive value determined by 90-day mortality in a previous study [8], and to ensure sufficient statistical power with regard to the number of patients, the relationship between clinical and laboratory characteristics was analyzed according to this value. In addition, SII was calculated as: neutrophil (N) (10–3/μl) X platelet (P) (10–3/μl)/lymphocyte (L) (10–3/μl). LCR was calculated by dividing lymphocyte count (10–3/μl) by CRP (mg/l). NLRb was calculated as neutrophil (10–3/μl)/lymphocyte count (10–3/μl), and PLR as platelet (10–3/μl)/lymphocyte count (10–3/μl).

Statistical Analysis

All data were recorded and analyzed using the Statistical Package for Social Sciences (SPSS) for Windows 22. Data from the study were described using descriptive statistics such as mean and standard deviation for numerical variables and frequency and percentage analyses for categorical variables. The first step in the analysis was to test the assumptions that had to be met for deciding which tests, i.e., parametric or non-parametric, to use. Kolmogorov-Smirnov, kurtosis, and skewness values were examined to determine the normality of the distribution and to determine which analyses could be performed with parametric tests. The independent sample t-test and Mann–Whitney U test were used for two group comparisons. The relationship between categorical variables was analyzed using chi-square and Fisher's exact tests. A significance value (p value) of 0.05 was used as the criterion

for interpreting whether the values obtained from statistical analyzes were statistically significant.

Results

Characteristics and laboratory values of the patients are shown in Table 1. The mean age of the patients was 61.4±12.2 yrs and 78.3 (n=310) were male. DM was present in 38.4% (n=152), HT in 50.5% (n=200), HL in 15.7% (n=62), and 38.6% (n=153) were smokers. The type of ACS was NSTEMI in 56.1% (n=222) of the patients, the responsible vessel was the left anterior descending artery (LAD) in 42.4% (n=168), and 65.2% (n=258) were multivessel patients. Previous AF was present in 3.3% (n=13) of patients. The mean left ventricular ejection fraction (LVEF) was 46.3±10.8 and the SYNTAX-1 score was 14.6±7.8. The mean SCI score was 97.5±47.1.

WBC, neutrophil and lymphocyte counts were higher in the SCI <100 group, whereas fibrinogen and CRP were higher in the SCI >100 group. NLR and LCR were higher in the SCI <100 group, whereas PLR and platelet count were higher in the SCI >100 group ($p<0.05$). No statistically significant relationship was found between SCI and the other parameters ($p>0.05$, Table 1).

DM, HT, previous HF, CKD were significantly higher in the group with SCI >100. Bleeding from any cause was considered an in-hospital complication and was significantly higher in patients with SCI >100 ($p<0.05$). Other in-hospital events were not significantly associated with SCI ($p>0.05$, Table 1).

Discussion

This study found that bleeding was a significantly more common in-hospital event in ACS patients with SCI >100, whereas other in-hospital events, including access site complications, no-reflow, AKI, cardiogenic shock, new heart failure, VT-VF, new AF, AVB, stent thrombosis, and mortality, were not significantly associated with the SCI value.

Table 1. Clinical and demographic characteristics of the study population and comparison of these characteristics with in-hospital complications according to the SCI

Characteristic	All Patients	SCI <100 (n = 237)	SCI >100 (n = 159)	p*
Age, yrs	61.4±12.2	60.2±12.3	63.3±11.8	0.013
Gender, male	310 (78.3)	200 (84.4)	110 (69.2)	<0.001
DM	152 (38.4)	80 (33.4)	72 (45.3)	0.02
HT	200 (50.5)	102 (43)	98 (61.6)	0.01
HL	62 (15.7)	40 (16.9)	22 (13.8)	0.41
Smoking	153 (38.6)	103 (43.5)	50 (31.4)	0.016
Previous HF	21 (5.3)	6 (2.5)	15 (9.4)	0.01
Previous CAD				0.36
Stent	79 (20)	45 (19)	34 (21.4)	-
CABG	33 (8.3)	16 (6.8)	17 (10.7)	-
Non-critical	20 (5)	14 (5.9)	6 (3.8)	-
None	264 (66.7)	162 (68.3)	102 (64.1)	-
COPD	15 (3.8)	8 (3.4)	7 (4.4)	0.6
Previous CVE	23 (5.8)	10 (4.2)	13 (8.2)	0.099
CKD	43 (10.9)	17 (7.1)	26 (16.3)	0.01
ACS Type				0.01
Anterior	72 (18.2)	53 (22.4)	19 (12)	-
Inferior	102 (25.8)	66 (27.9)	36 (22.6)	-
NSTMI	222 (56)	118 (49.8)	104 (65.4)	-
IRA				0.054
LAD	168 (42.4)	106 (44.7)	62 (39)	-
CX	96 (24.2)	50 (21.1)	46 (29)	-
RCA	114 (28.8)	74 (31.2)	40 (25.2)	-
Saphenous	18 (4.6)	7 (3)	11 (6.9)	-
Number of Vessels				0.025
Single Vessel	138 (34.9)	93 (39.2)	45 (28.3)	-
Multivessel	258 (65.2)	144 (60.8)	114 (71.7)	-
Drugs				
ASA	381 (96.2)	228 (96.2)	152 (95.6)	0.625
P2y12 inh.	388 (98)	235 (99.2)	153 (96.2)	0.042
RAS blocker	304 (76.8)	187 (78.9)	117 (73.6)	0.219
BB	348 (87.9)	216 (91.1)	132 (83)	0.015

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Table 1. Continuation. Clinical and demographic characteristics of the study population and comparison of these characteristics with in-hospital complications according to the SCI

Characteristic	All Patients	SCI <100 (n = 237)	SCI >100 (n = 159)	p*
Statin	382 (96.5)	234 (98.7)	148 (93.1)	0.003
MRA	75 (18.9)	40 (16.9)	35 (22)	0.2
Nitrate	25 (6.3)	15 (6.3)	10 (6.3)	0.987
Furosemide	52 (13.1)	25 (10.5)	27 (17)	0.063
Inotrope	14 (3.5)	8 (3.4)	6 (3.8)	0.833
SGLT-2i	14 (3.5)	10 (4.2)	4 (2.5)	0.368
Amiodarone	15 (3.8)	8 (3.4)	7 (4.4)	0.6
Ivabradine	6 (1.5)	4 (1.7)	2 (1.3)	0.731
OAC	15 (3.8)	9 (3.8)	6 (3.8)	0.984
SYNTAX I score	14.6±7.8	14.6±8.1	14.7±7.3	0.88
LVEDD, mm	48.4±5.7	48±5.6	48.8±5.8	0.219
LVEF, %	46.3±10.8	46.4±10.2	46.1±11.6	0.80
Killip Class	1,4±0.8	1.4±0.8	1.4±0.8	0.35
Rhythm				0.48
SR	383 (96.7)	228 (96.2)	155 (97.5)	
AF	13 (3.9)	9 (3.8)	4 (2.5)	
Creatinine, mg/dl	1.1±0.9	1±0.7	1.2±1.2	0.011
eGFR, ml/dk	80.9±26.7	85.6±24.3	73.8±28.4	<0.001
Hs-Troponin1, ng/l	647.2±1282.4	672.7±1425.1	609.1±1037.1	0.63
Hs-Troponin2, ng/l	3210.5±469	3623.4±5241.6	2595±3603.7	0.03
NT-ProBNP, ng/L	1819.3±5469.9	1276.6±4600.9	2628.3±6487	0.02
D-dimer, ug FEU/ml	0.9±1.8	0.9±2.10	0.9±1.2	0.98
Fibrinogen, g/l	4±1.2	3.5±0.9	4.7±1.3	0.01
CRP, mg/l	20.5±36.2	15.1±27.6	28.6±45	0.01
WBC, $x10^3$	11.1±4	12±4.3	9.7±3	0.01
NEU, $x10^3$	8 ±3.7	8.7±4	6.9±2.9	0.01
LYM, $x10^3$	2.2±1.4	2.4±1.6	2±0.9	0.01
PLT, $x10^3$	250.9±77.7	152.5±59	256±75.7	0.01
SCI	97.5±47	68.8±18	140,1±44.6	-
SII	1224.3±1121.9	1181.4±1113.7	1288.3±1134.6	0.35
NLR	6.3±3.9	6.5±4	6±3.6	0.16
PLR	142.5±80.5	121.7±65.7	173.6±90.3	0.01
LCR	0.7±1.2	0.9±1.3	0.5±0.9	0.01
Complications				
Access Site	19 (4.8)	15 (6.3)	4 (2.5)	0.08
No-reflow	10 (2.5)	6 (2.5)	4 (2.5)	1.00
AKI	43 (11)	24 (10.1)	19 (12)	0.56
Cardiogenic Shock	24 (6.1)	15 (6.3)	9 (5.7)	0.78
Bleeding	28 (7.1)	9 (3.8)	19 (12)	0.02
New HF	44 (11.1)	26 (11)	18 (11.3)	0.91
VT-VF	17 (4.3)	13 (5.5)	4 (2.5)	0.15
New AF	14 (3.5)	9 (3.8)	5 (3.1)	0.73
AVB	8 (2)	2 (0.8)	6 (3.8)	0.06
Stent Thrombosis	19 (4.8)	9 (3.8)	10 (6.3)	0.25
Mortality	17 (4.3)	8 (3.4)	9 (5.7)	0.27

Data are n (percentage) or mean±SD. *p<0.05 indicates statistical significance between SCI groups. Critical stenosis was defined as >50% stenosis on CAG, whereas <50% stenosis was considered non-critical. Those with no previous evidence of coronary artery disease were classified as "None". DM, diabetes mellitus; HT, hypertension; HL, hyperlipidemia; HF, heart failure; CAD, coronary artery disease; CABG, coronary artery bypass grafting; COPD, chronic obstructive pulmonary disease; CVE, cerebrovascular event; CKD, chronic kidney disease; AKI, acute kidney injury; ACS, acute coronary syndrome; IRA, infarct related artery; NSTMI, non-ST segment elevated myocardial infarction; LAD, left anterior descending artery; CX, circumflex artery; RCA, right coronary artery; SR, sinus rhythm; AF, atrial fibrillation; ASA, acetylsalicylic acid; BB, beta blocker; RAS, renin angiotensin aldosterone system; MRA, mineralocorticoid receptor antagonist; SGLT-2i, sodium-glucose co-transporter 2 inhibitor; OAC, oral anticoagulant; LVEDD, left ventricular end diastolic diameter; LVEF, left ventricular ejection fraction; SCI, systemic coagulation inflammation index; SII, systemic immune inflammation index; NLR, neutrophil lymphocyte ratio; eGFR, estimated glomerular filtration rate; WBC, white blood cell; NEU, neutrophil; LYM, lymphocyte; hs, high sensitive; NT-proBNP, n-terminus pro-B-type natriuretic peptide; CRP, C-reactive protein; PLR, platelet lymphocyte ratio; LCR, lymphocyte to CRP ratio; 1, hospital admission; 2, highest; VT, ventricular tachycardia; VF, ventricular fibrillation; AVB, atrioventricular block.

ACS results from the rupture of a vulnerable atherosclerotic plaque that has been developing for years, and which occurs against a background of inflammation. The inflammation-induced plaque rupture exposes the necrotic plaque contents to circulating blood and activates the coagulation cascade and the thrombogenic system. Leukocytes, platelets, and fibrinogen are important components of this process. Elevated WBC concentrations contribute to the development of the prothrombotic state by modulating platelet activity, by direct endothelial damage, by activation of the extrinsic pathway, and by generating prothrombotic tissue factors [14]. There is a known association between WBC count and cardiovascular outcomes in patients with STEMI and NSTMI. In ACS, the degree of leukocyte migration after plaque rupture and its relationship to the extent of infarct area suggest that high leukocyte counts are associated with clinical outcomes [15]. On the other hand, fibrinogen, an acute phase reactant and coagulation factor, causes platelet aggregation, affects blood viscosity and erythrocyte aggregation, and causes endothelial cell damage during ACS [16]. Fibrinogen is converted to fibrin in the final step of the coagulation cascade, and fibrin is one of the molecules that forms thrombi. Elevated concentrations of fibrinogen have been associated with the risk of recurrent, unstable angina and myocardial infarction [17–19].

SCI was evaluated as a parameter reflecting the activity of the inflammatory and coagulation systems in type A aortic dissection [8]. In that study, the primary outcome was 90-day, out of hospital, all-cause death. The secondary outcomes were 30-day hospital and intensive care unit mortality, duration of mechanical ventilation, duration of stay in the intensive care unit, bleeding, and stroke. The patients were divided into three groups: SCI <40, SCI 40–100, SCI >100. 90-day survival was 96.4% in the SCI >100 group, 92.7% in the SCI 40–100 group, and 86.9% in the SCI <40 group ($p<0.001$). 30-day mortality, hospital mortality, ventilator support, and length of ICU stay were significantly different between the groups and were higher in the SCI <40 group ($p<0.05$) [8]. Based on the findings of in study, we evaluated SCI values in ACS where inflammation and coagulation are co-activated. We found no correlation between SCI values and other in-hospital clinical outcomes, except for bleeding in the two groups with SCI <100 and SCI >100. It can be concluded that the high WBC in the SCI <100 group was compensated by the high fibrinogen and platelets in the SCI >100 group and did not lead to a significant change in SCI. In aortic dissection, leukocytosis is seen as a reflection of inflammation, and fibrinogenopenia and thrombocytopenia are also present due to increased systemic fibrinolytic activity, regional thrombosis, and redistribution of peripheral platelets [8]. In our study, platelet and fibrinogen concentrations were within normal limits. In another study, fibrinogen concentrations were found to be higher in the American population than in the Japanese population [20]. Given that the above aortic dissection study was also conducted in China, racial and environmental differences may have led to these results.

In our study, bleeding as a clinical outcome was significantly more common in the group with SCI >100. Interestingly, fibrinogen and platelet counts were significantly higher in this group. On the contrary, in the aortic dissection study [8], bleeding was more common in the SCI <40 group, and fibrinogen and platelet concentrations were also lower in the SCI <40 group. The relationship between low fibrinogen concentration and bleeding is not clear [7]. In addition, the coagulation system has other components in addition to platelets and fibrinogen. In one study, WBC elevation was associated with 30-day major/minor bleeding [14]. In our data, the WBC count was lower in the SCI >100 group. On the other hand, the increase in hs-CRP, another inflammatory parameter, from admission to outpatient clinic was associated with major bleeding [21]. In that study, CRP was significantly higher in the SCI >100 group, which had more frequent bleeding. In the SCI >100 group, however, DM, HT, and CKD were also higher. Also, the risk of major bleeding was found to be higher in the presence of HT in NSTMI [22]. In another study, the presence of HT, renal insufficiency, and ACS (higher in STEMI) was associated with bleeding [23]. In contrast to that study, we found that the rate of STEMI was lower in the SCI >100 group in which bleeding was more common (50.1% STEMI in the SCI <100 group and 34.6% in the SCI >100 group, $p=0.01$). With platelet dysfunction in CKD, the use of antiplatelet agents during ACS will increase the risk of bleeding [24]. This may be one of the reasons that bleeding was more common in the SCI >100 group. However, one study found no significant increase in the risk of bleeding in patients with DM who took antiplatelet drugs for ACS [25].

Study Limitations

This study has several limitations. First, the study population was relatively small and represented only individuals in the location of our hospital. Second, it was an observational cross-sectional study. Prospective, randomized, controlled trials in larger and different populations may provide more informative data. Third, the time between blood sampling and symptom onset was not the same in all patients. Fourth, patients received antiplatelet and anticoagulant therapy, which may have affected the blood values. Fifth, the analyses were performed on a single blood sample taken at the hospital. Different and significant values might have been obtained if calculations were performed on follow-up blood samples taken during hospitalization. Sixth, values obtained by recalculating the SCI in ACS patients at medium- and long-term follow-up might have produced different results. Seventh, in the study in which we used our cut-off as a reference [8], SCI was divided into 3 groups as <40, 40–100, >100, whereas in our study we

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could not make such an evaluation because of the small number of patients <40. Different results may be found in studies that include the <40 group of patients.

Conclusion

In this study, bleeding in ACS patients was significantly more common in the group with SCI>100. There was no significant correlation between SCI and other clinical outcomes. According to these findings, SCI may be a useful parameter for predicting in-hospital bleeding complications in ACS. On the other hand, SCI was not associated with other in-hospital events.

No conflict of interest is reported.

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