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Evaluation of Inflammatory Response in Patients Receiving Clopidogrel, Ticagrelor and Prasugrel Due to Acute Coronary Syndrome

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ABSTRACT

SII was calculated as the ratio of the total neutrophil count to the lymphocyte count (neutrophil/lymphocyte ratio; NLR) and the product of the total platelet count. (SII = NLR × total platelet count in the peripheral blood (per mm3)).Prasugrel and ticagrelor are known to cause rapid onset and more significant platelet inhibition than clopidogrel in patients with acute coronary syndrome. SII This study examined the role of prasugrel, ticagrelor, and clopidogrel in treating acute coronary syndrome.

Patients who underwent coronary angiography and received dual antiplatelet therapy for acute coronary syndrome were retrospectively screened from the hospital information system.

A total of 173 patients were excluded and who met the inclusion criteria (62 patients receiving clopidogrel, 46 receiving ticagrelor, and 65 receiving prasugrel) were examined. The inflammatory responses of these patients were compared by examining their complete blood counts at the time of admission and at the 3rd and 6th months, and a statistically significant difference was observed in the 6th month inflammatory responses ($p < 0.001$).

Although the current findings showed no significant difference between the three drugs in the early period, the inflammatory response to prasugrel and ticagrelor was better than that to clopidogrel in the 6th month.

Keywords; Clopidogrel, Prasugrel,Ticagrelor, Inflammation

Introduction

Cardiovascular diseases are the most common cause of mortality and morbidity worldwide(1). Many complex inflammatory processes play a role in acute myocardial infarction. In the inflammatory response, an increase in neutrophil levels occurs first, peaking within 1-3 days (2). Later, monocyte and platelet levels increase due to increased adrenaline and steroid levels, and lymphocyte levels decrease. The systemic immune inflammatory index (SII) has been used as a sensitive method for examining inflammation in recent years (3), and in some studies, it has even been shown to be superior to many inflammatory biomarkers, such as the neutrophil/lymphocyte ratio (NLR) and platelet/lymphocyte ratio (PLR) (4). Despite the improved treatment and stent technology for acute myocardial infarction, adverse cardiovascular events continue to occur. Approximately one-quarter of patients have residual ischemia after stent implantation, and approximately 10.7% have a history of rehospitalization within 30 days after the procedure (5). Therefore, risk assessment is important, particularly in high-risk patients. This study aimed to examine the relationship between antiplatelet therapy and the inflammatory response using commonly used hematological markers based on cost-effective blood cell counts and subtypes (neutrophils, monocytes, and lymphocytes).

Material and Method

Patients admitted to our hospital between 01.01.2023 and 01.01.2024 due with acute coronary syndrome were retrospectively screened. In our study, 289 patients were scanned, but only 173 were included in the study (Figure 1). A total of 49 patients with ST elevation myocardial infarction and 124 patients with non-ST elevation myocardial infarction diagnosed in accordance with the Universal Myocardial Infarction

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definition were included in the study. Patients with cardiogenic shock, severe active and chronic infection, active bleeding, autoimmune diseases, pregnancy, aortic dissection, acute pulmonary edema, hypertrophic cardiomyopathy, endocarditis, pericarditis, major surgery, and oncology who received active treatment were excluded from the study. The hemogram parameters of the patients who began to be administered dual antiplatelet therapy after the detection of ischemia in coronary angiography were retrospectively scanned through the hospital information system at the time of admission, at the 3rd month, and at the 6th month. SII was calculated as the ratio of the total neutrophil count to the lymphocyte count (neutrophil/lymphocyte ratio; NLR) and the product of the total platelet count. (SII = NLR × total platelet count in the peripheral blood (per mm3)).

The SII was calculated as the ratio of the total neutrophil count to the lymphocyte count (neutrophil/lymphocyte ratio; NLR) multiplied by the total platelet count. (SII = NLR × total platelet count in the peripheral blood (per mm3).The Systemic inflammatory response index (neutrophil rophil× monocyte/lymphocyte), PLR (Platelet/lymphocyte ratio (PLR), and monocyte-to-lymphocyte ratio (MLR) were also Our study was approved by the local ethics committee in accordance with the Declaration of Helsinki(date of ethics committee 05.03.2024 no: 2024/3-9)

Results

The demographic and clinical findings of the study groups are presented in Table 1. Between the groups, it was found out that the patients receiving Clopidogrel were older than the patients receiving ticagrelor and Prasugrel (p<0.001). Again, it was observed that the patients receiving clopidogrel had better Ejection Fractions (EF), which is considered non-invasive (p<0.001). Hypertension (HT) was observed in 72.6% of patients receiving clopidogrel, 50% of those receiving ticagrelor, and 70.8% of those receiving prasugrel (p=0.029). Hyperlipidemia (HPL) was observed in 69.4% of patients receiving clopidogrel, 43.5% of those receiving ticagrelor, and 61.5% of those receiving prasugrel (p=0.023). 14.5% of patients receiving clopidogrel had STEMI (ST elevation Myocardial Infarction) and 85.5% had NSTEMI (Non-ST Myocardial Infarction); 37% of patients receiving ticagrelor had STEMI and 63% had NSTEMI; among the patients recceving prasugrel, 34.4% had STEMI and 64.6% had NSTEMI (p=0.011)

The laboratory findings of the study groups are presented in Tables 2 and 3. A statistically significant difference was observed in BUN and albumin levels between the groups (p = 0.006, p = 0.007).

While no statistical difference was observed in the monocyte counts of patients receiving clopidogrel, ticagrelor, and prasugrel at the beginning and 3rd month controls, a statistically significant difference was observed in favor of ticagrelor and prasugrel at the 6-month controls. (p=0.360, p=0.611, p=0.019) While no statistical difference was observed in neutrophil counts in the initial controls, a statistically significant difference was observed in favor of ticagrelor and prasugrel in the 3rd and 6th month controls (p = 0.423, p = 0.001, p < 0.001). While no statistical difference was observed in the lymphocyte counts of patients receiving clopidogrel, ticagrelor, and prasugrel at the initial controls, a statistically significant difference was observed in favor of ticagrelor and prasugrel at the 3rd and 6th month controls (p=0.281, p=0.007, and p<0.001, respectively). No statistically significant difference was observed in the platelet counts of patients receiving clopidogrel, ticagrelor and prasugrel at the initial 3rd and 6th month controls. (p=0.856, p=0.278, p=0.132)

While no statistical difference was observed in the SII responses of patients receiving clopidogrel, ticagrelor, and prasugrel at the initial controls, a statistically significant difference was observed in favor of ticagrelor and prasugrel at the 3rd and 6th month controls (p = 0.521, p = 0.006, p < 0.001, respectively) (Figure 2.3).

While no statistical difference was observed in the SIRI responses of patients taking clopidogrel, ticagrelor, and prasugrel at the initial controls, a statistically significant difference was observed in favor of ticagrelor and prasugrel at the 3rd and 6th month controls (p = 0.464, p = 0.017, p < 0.001). While no statistical difference was observed in NLR responses in the initial controls, a statistically significant difference was observed in favor of ticagrelor and prasugrel in the 3rd and 6th month controls (p = 0.461, p = 0.005, p < 0.001). No statistical difference was observed in the PLR responses of patients receiving clopidogrel, ticagrelor, and prasugrel in the initial controls. A statistically significant difference was observed in favor of ticagrelor and prasugrel at the 3rd and 6th month controls (p=0.576, p=0.026, and p<0.001, respectively). While no

Table 1: Demographic Features

	Clopidogrel (n=62)	Ticagrelor (n=46)	Prasugrel (n=65)	p value
Gender (Female), n(%)	24(38.7)	12(26.1)	17(26.2)	0.227
Age, (years)	66.6±13.7	59.0±11.3	58.7±9.5	<0.001
Body mass index, (kg/m2)	26.2±2.8	25.8±2.9	23.8±4.5	0.001
Heart Rate (minute)	85.8±16.5	82.4±14.2	85.4±15.8	0.489
Systolic Blood Pressure (mmHg)	128.8±14.0	133.5±16.6	128.9±15.8	0.221
Diastolic Blood Pressure(mmHg)	78.8±7.7	80.9±10.7	81.8±9.6	0.183
EF, %(IQR)	55(50-60)	55(45-60)	50(40-55)	<0.001
CRF, n(%)	12(19.4)	2(4.3)	7(10.8)	0.056
HT, n(%)	45(72.6)	23(50)	46(70.8)	0.029
DM, n(%)	24(38.7)	9(19.6)	22(33.8)	0.097
HPL, n(%)	43(69.4)	20(43.5)	40(61.5)	0.023
SMOKING, n(%)	18(29)	17(37.0)	19(29.2)	0.618
COPD, n(%)	6(9.7)	2(4.3)	1(1.5)	0.113
Previous PCI, n%	31(50)	15(32.6)	35(53.8)	0.072
Previous CABGO, n%	2(3.2)	1(2.2)	4(6.2)	0.531
Hospitalization day, IQR	3.2±1.1	3.2±1.0	3.0±1.1	0.443
Myocardial infarction type STEMI, n(%)	9(14.5)	17(37.0)	23(35.4)	0.011
Myocardial infarction type NSTEMI, n(%)	53(85.5)	29(63.0)	42(64.6)	0.011

EF: Ejection Fraction; CRF: Chronic Renal Failure; HT: Hypertension; DM: Diabetes Mellitus HPL: Hyperlipidemia; COPD: Chronic Obstrictive Pulmonary Disease; PCI: Percutaneous Coronary Intervention; CABGO: Coronary Artery Bypass Graft Operation; STEMI: ST Elevation Myocard Infarction; NSTEMI: Non-ST Elevation Myocardial Infarction

statistical difference was observed in the MLR responses of patients receiving clopidogrel, ticagrelor and prasugrel at the initial controls, a statistically significant difference was observed in favor of ticagrelor and prasugrel at the 3rd and 6th month controls. (p=0.661, p=0.021, p<0.001)

Statistical Analysis: During the statistical analysis, continuous variables were expressed as mean ± standard deviation, and categorical variables were expressed as numbers and percentages. Normally distributed variables are expressed as mean ± standard deviation. The normal distribution of numerical variables was evaluated using subjective methods, such as histogram and probability curves (Q-Q and P-P plots), as well as objective methods, such as the Lilliefors and Shapiro-Wilk tests. Normally distributed numerical variables were evaluated using the parametric Student's t-test. The chi-square test was used to determine how the categorical variables changed between the groups. Specificity and sensitivity values were calculated using the receiver operating characteristic (ROC) curve to predict interatrial conduction characteristics. Statistical significance was set at p <0.05 was considered important.

Discussion

To the best of our knowledge, no study has examined the relationship between the inflammatory responses of these three drugs (clopidogrel, prasugrel, and ticagrelor) initiated for acute coronary syndrome. In our study, we examined the SII, SIRI, NLR, PLR, and MLR responses in patients receiving clopidogrel, prasugrel, and ticagrelor due to acute coronary syndrome. No statistically significant difference was observed in the initial and 3-month evaluations in patients receiving prasugrel and ticagrelor; however, a statistically significant difference was observed in the 6-month inflammation responses compared to those in patients receiving clopidogrel.

Table 2: Laboratory Findings

	Clopidogrel (n=62)	Ticagrelor (n=46)	Prasugrel (n=65)	p value
WBC(x103/uL)	9.9±2.8	10.2±3.2	10.8±3.1	0.239
BUN, (mg/dL)	42.6±17.0	32.4±9.1	37.9±18.6	0.006
Creatinine, (mg/dL)	0.99±0.34	0.87±0.22	0.94±0.29	0.145
CRP(mg/dL)	0.42(0.24-1.18)	0.56(0.15-1.20)	0.45(0.18-0.93)	0.807
Total cholesterol (mg/dl)	176.8±51.4	187.8±48.3	183.0±58.2	0.561
HDL cholesterol (mg/dl)	42.2±14.4	39.7±11.0	38.7±8.3	0.224
LDL cholesterol (mg/dl)	104.2±42.8	112.2±39.3	107.9±46.0	0.647
Triglyceride(mg/dl)	125(88-179)	140(102-221)	155(106-202)	0.289
Hgb(g/L)	14.0±1.8	14.5±1.5	14.4±1.6	0.172
Albumin (g/L)	3.9±0.5	4.1±0.3	4.1±0.3	0.007
Monocyteat admission(103/mm3)	0.66±0.28	0.70±0.22	0.72±0.23	0.360
3rd monthmonocyte(103/mm3)	0.63±0.23	0.61±0.16	0.59±0.15	0.611
6th monthmonocyte (103/mm3)	0.61±0.18	0.56±0.15	0.52±0.19	0.019
Neutrophil at admission (103/mm3)	7.06±3.08	7.11±3.36	7.75±3.30	0.423
3rd month neutrophil(103/mm3)	6.70±2.82	5.74±1.54	5.30±1.59	0.001
6th month neutrophil(103/mm3)	6.39±2.43	5.33±1.61	4.78±1.42	<0.001
Lymphocyte at admission (103/mm3)	1.9±0.7	2.2±1.1	2.1±0.9	0.281
3rd month lymphocyte(103/mm3)	2.0±0.7	2.5±1.0	2.2±0.7	0.007
6th month lymphocyte(103/mm3)	1.9±0.9	2.6±0.9	2.4±0.6	<0.001
Platelet at admission (103/mm3)	298.4±90.1	295.7±74.2	290.7±70.3	0.856
3rd month platelet(103/mm3)	261.5±65.5	257.5±58.5	245.2±54.0	0.278
6th month platelet(103/mm3)	244.4±56.1	235.1±49.4	225.8±48.8	0.132

WBC: White Blood Cell; BUN: Blood-Urea-Nitrogen CRP:C-Reactive Protein; HDL: High Density Lipoprotein; LDL: Low density Lipoprotein; Hgb: Hemoglobine

Table 3: SII, SIRI, NLR and MLR Responses At The Begining, 3rd and 6th Months

	Clopidogrel (n=62)	Ticagrelor (n=46)	Prasugrel (n=65)	p value
SII at admission	946(652-1577)	932(539-1528)	944(590-1692)	0.521
3rd month SII	785(516-1441)	646(359-805)	544(385-878)	0.006
6th month SII	759(475-1421)	508(278-643)	418(297-588)	<0.001
SIRI at admission	2.09(1.15-3.53)	1.76(1.25-3.69)	2.11(1.40-4.66)	0.464
3rd month SIRI	2.06(1.02-3.52)	1.31(0.95-2.08)	1.34(0.89-2.02)	0.017
6th month SIRI	1.82(1.25-3.55)	1.03(0.74-1.72)	0.94(0.71-1.35)	<0.001
NLRat admission	3.35(2.15-5.00)	2.61(1.86-4.68)	2.95(2.28-7.11)	0.461
3rd month NLR	3.08(2.13-4.78)	2.47(1.63-3.17)	2.26(1.73-3.28)	0.005
6th month NLR	3.26(1.99-6.25)	1.92(1.27-2.91)	1.90(1.44-2.64)	<0.001
PLRat admission	165(118-208)	144(100-208)	133(110-198)	0.576
3rd month PLR	128(100-188)	110(74-143)	106(82-145)	0.026
6th month PLR	131(89-210)	89(68-121)	91(73-123)	<0.001
MLRat admission	0.33(0.23-0.48)	0.32(021-0.51)	0.35(0.24-0.52)	0.661
3rd month MLR	0.30(0.21-0.44)	0.23(0.20-0.33)	0.26(0.22-0.35)	0.021
6th month MLR	0.35(0.22-0.54)	0.22(0.16-0.28)	0.21(0.17-0.27)	<0.001

SII: Systemic immune inflammatory index; SIRI: NLR: Neutrophil/Lymphocyte ratio; PLR: Platelet/Lymphocyte ratio MLR (Monocyte/Lymphocyte ratio

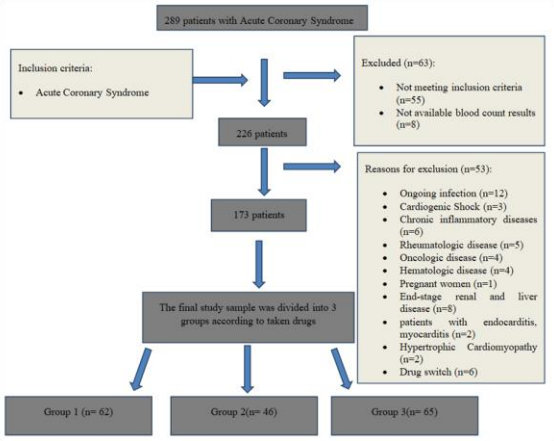

Fig.1. Flow Chart

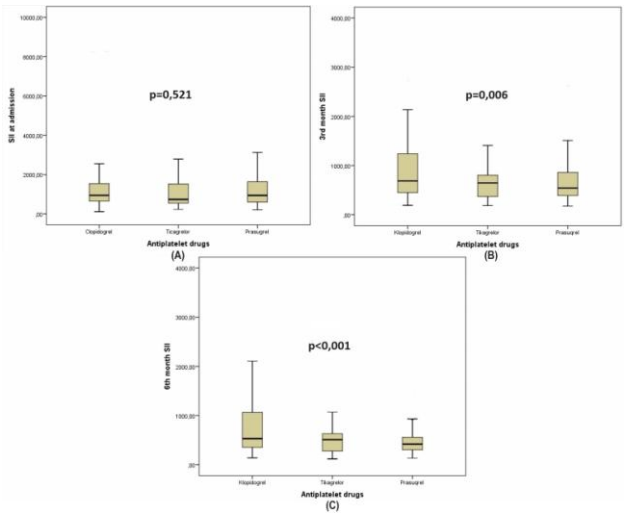

Fig.2. SII responses at admission, 3rd and 6th months

The immune system and inflammatory processes play a significant role in the pathogenesis of atherosclerosis. Inflammation is responsible for ischemic-reperfusion injury in ACS.Monocytes, a specific leukocyte subgroup, initiate atherosclerosis by releasing reactive oxygen radicals (ROS) and proteolytic enzymes and they differentiate into macrophages in the endothelium and turn into foam cells by phagocytosing lipids (6). The number of monocytes is considered predictive of mortality in the long and short terms, independent of other risk factors. In contrast, neutrophils cause muscle cell apoptosis and aggravate inflammation. In addition, a high neutrophil count has been associated with plaque rupture and increased risk of thrombosis. Additionally, lymphocytes prevent atherosclerosis progression. Platelets activate plaque formation, inflammation, and thrombosis (7).

The SII is thought to be an important indicator of systemic inflammation, in which neutrophils, platelets, and lymphocytes play a role. In a study

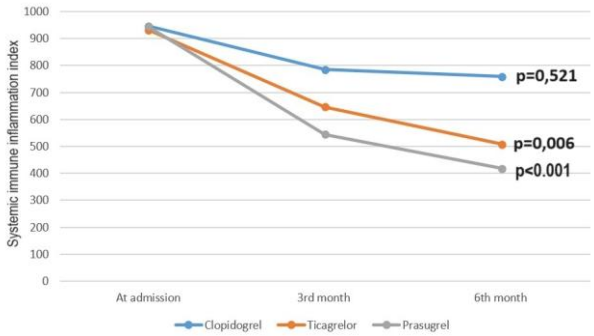

Fig.3. SII responses at admission, 3rd and 6th months

by Liu et al., SII was evaluated as an independent factor in the diagnosis and severity of CAD(8). The SII and NLR are independent determinants of major adverse cardiovascular cases) in patients with ACS (9). Yang et al. In 5602 patients with coronary artery disease, SII was a stronger predictor of major cardiovascular events than traditional risk factors (10).

It has been shown that high WBC levels are associated with mortality in patients with ACS (11). In our study, we did not observe a statistically significant difference between WBC values and medication intake ($p = 0.239$). It has been shown that SII levels are an independent predictor of no-reflow in ACS patients (12).

SII is an independent predictor of contrast-induced nephropathy and coronary collateral formation in patients with NSTEMI. (13). In a study in which more than 5000 patients diagnosed with CAD treated with PCI were observed, it was found out that SII predicted the occurrence of MACE when compared with classical risk factors (10).

In an observational study involving 85 thousand people, a positive correlation was shown between SIRI and the frequency of ACS in patients under the age of 60 years; however, this could not be demonstrated with SII (14). In a study involving 4606 patients with heart failure, the SII was demonstrated to predict short-term mortality (15). All these studies show that SII levels are associated with poor outcomes. SII is a prognostic indicator, especially for malignancies rather than cardiovascular events (16).

SIRI includes the absolute count of neutrophils, monocytes, and lymphocytes and has been associated with the occurrence of CVD and the development of atrial fibrillation in patients with CVD (17). A high NLR after dual antiplatelet therapy still indicates weakness of platelet inhibition in patients with ACS, which increases

the risk of thrombosis and recurrent ischemia events (18).

In a study by Choi et al., NLR >2.8 was found to be a determinant of adverse cardiovascular events in coronary artery disease (19). XU et al. showed that NLR >3.39 was related with 2-year adverse cardiovascular events in patients with 3-vessel and left main vessel disease (20). The increase in neutrophils and the decrease in the number of lymphocytes may intensify oxidative stress and inflammatory damage, increasing the risk of coronary artery disease (21).

Megakaryocyte proliferation in ACS patients may be related to inflammatory activation and a prothrombotic state (2). Li et al showed that increased PLR was associated with poor prognosis in elderly patients. Trakarnwijitr et al. showed that PLR was associated with coronary artery disease in patients aged > 55 years (22).

Studies have shown that MLR can be used to predict the severity of coronary lesions in coronary artery disease (23), and monocytes play an important role in the development of atherosclerosis. Monocytes differentiate into macrophages in the endothelium and become foam cells by digesting oxidized lipoproteins in the subendothelium (23).

The SII was initially developed as a parameter useused in oncology patients. In recent years, it has been shown to be an important indicator for determining the severity of coronary artery disease. Compared to NLR, PLR, and MLR, SII SIRI aret is more comprehensive parameters that include three inflammatory cells; therefore, they can be said to be more powerful in showing cardiovascular outcomes. It should be noted that while the component in SII is platelets, the component in SIRI is monocytes. Monocytes are key cells in the formation of atherosclerosis, which can cause a significant difference in the results.

Limitation: In addition to the strengths of this study, there are some limitations.

For example, our study was designed as a single-center retrospective and cross-sectional study, and the relatively small sample size of our research highlights the need for further studies that can be carried out with this patient population and relatively broad exclusion criteria (cardiogenic shock, severe active and chronic infection, active bleeding, autoimmune diseases, pregnancy, aortic dissection, acute pulmonary edema, hypertrophic cardiomyopathy, endocarditis, percarditis, major surgery, and oncology patients receiving active treatment). In addition to patients who underwent coronary angiography, the issues requiring technical details such as the number of stents used in the procedure, contrast material, amount of radiation, and malopposition were not counted.

Many complex parameters with limited practical use are used to determine the long- and late-term prognosis in ACS patients (such as the Global Registry of Acute Coronary Events (GRACE) risk score and the SYNergy between PCI with TAXus and cardiac surgery (SYNTAX) score) and practical parameters such as SII. SII was calculated as the ratio of the total neutrophil count to the lymphocyte count (neutrophil/lymphocyte ratio; NLR) and the product of the total platelet count. (SII = NLR × total platelet count in the peripheral blood (per mm3). A low-cost and powerful parameter can be used as an important indicator for determining prognosis and monitoring treatment response.

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