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Original Article

Correlation of Platelet-To-Lymphocyte Ratio and Troponin I Levels in Patients with ST-Elevation Myocardial Infarction

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Abstract

Introduction : Platelet and lymphocyte are crucial in acute myocardial infarction (AMI) pathogenesis. Platelet-lymphocyte ratio (PLR) is a systemic biomarker which indicates thrombosis and inflammation. The diagnosis of AMI can be made by detecting elevated blood troponin I levels. The correlation between PLR and troponin I levels in ST-segment elevation myocardial infarction (STEMI) patients has not been widely known. The objective of this study is to determine the correlation between PLR and troponin I levels in STEMI patients.
Methods : This study was a cross-sectional study which data were obtained from the medical records of patients with STEMI admitted to Dr. Kariadi General Hospital Semarang from 2019 to 2020. Serum Troponin I levels were measured with Enzyme-Linked Fluorescent Assay (ELFA) method in less than 24 hours from symptoms onset. Complete blood counts were performed immediately on hospital admission. Correlations between variables were obtained using Spearman's test.
Results : A total of 28 STEMI patients were included, 82.1% were male, mean age was 55.81 ± 2.13 years old. Mean symptoms onset was 7.52 ± 0.81 hours prior to admission. Mean PLR and troponin I levels were 233.32 ± 25.54 and 14.44 ± 3.16 µg/l, respectively. A moderately significant correlation was detected between troponin I levels and PLR in STEMI patients (r=0.333; p=0.042). Symptoms onset in STEMI patients had significant correlation to troponin I levels (r=0.596; p=0.001).
Conclusion : A moderate positive correlation between PLR and troponin I levels is observed within 24 hours from symptoms onset in STEMI patients.

Keywords : Platelet-to-lymphocyte ratio; ST-segment elevation myocardial infarction; troponin I

INTRODUCTION

In Indonesia, acute myocardial infarction (AMI) ranked in the top 10 mortality rate caused by non-communicable diseases within hospitalized patients with a mortality rate of 6.18% in 2009.[1] Based on the Semarang City Health Profile published by the Central Java Provincial Health Office, there was an increase in the number of AMI cases in hospitals from 2015 to 2019 with a total of 12.069 cases.[2] Approximately 25–40% of patients diagnosed with AMI experience ST-segment elevation myocardial infarction (STEMI).[3] The development of acute myocardial infarction has a close relationship with coronary artery occlusion by atherosclerotic plaque rupture which results in ischemia and necrosis of the myocardium.[4] Platelets have important roles in atherogenesis and atherothrombosis, thus platelets can be used as a marker to determine the risk and prognosis of myocardial infarction.[5] Lymphocyte also plays a significant part in modulating the inflammatory response in the atherothrombosis. Lymphocytopenia in AMI is caused by an increase in apoptosis of the lymphocyte in response to uncontrolled immune system activation.[6] Platelet-to-lymphocyte ratio (PLR) is a useful systemic biomarker which may determine the occurrence of thrombosis and inflammation.[5] Previous studies have found that PLR is a promising biomarker to establish the prognosis of various cardiovascular diseases.[7–11] Troponin is a cardiac marker that can be used to detect myocardial damage. The two troponin subtypes, troponin I and troponin T, are not detectable in significant amounts in normal individuals but their levels are elevated in AMI patients as a result of myocardial destruction.[12,13] The ability of troponin I to establish the diagnosis of AMI is more specific than troponin T.[14] It is known that troponin I levels within 24 hours after symptoms onset positively correlated with infarct size in AMI patients which was measured by several imaging modality.[15,16]

The relationship between PLR and prognosis of various cardiovascular diseases has been demonstrated in previous studies, but the correlation of PLR and troponin I levels in STEMI patients has not been widely known. Considering the potential and superiority of PLR as an excellent prognostic marker, the correlation of PLR and troponin I levels in STEMI patients is enticing to be studied further.

METHODS

Medical records of STEMI patients who are treated and hospitalized in Dr. Kariadi General Hospital Semarang between 2019 to 2020 were observed with a cross-sectional study design. Patients diagnosed with STEMI and are older than 19 years old were included in this study. Patients with infective diseases, hematological disorders, and malignancy were excluded from this study. After a consecutive evaluation, a total of 28 patients with complete medical records and laboratory examination results were enrolled in this study.

Demographic information, patients' diagnosis, cardiovascular disease history, laboratory examination results, comorbidities, and the treatments given to the patients were obtained from the medical records. Laboratory examination results were evaluated from blood samples obtained immediately after patients' arrival in less than 24 hours from symptoms onset. Troponin I levels examination were performed with Enzyme-Linked Fluorescent Assay (ELFA) method. PLR was calculated by dividing platelet count to absolute lymphocyte count (ALC) from routine blood examination.

Ethical clearance was attained from the Ethical Commission for Medical and Health Research, Faculty of Medicine, Universitas Diponegoro and study permit was obtained from Dr. Kariadi General Hospital Semarang with document number DP.02.01/I.II/3774/2021.

Statistical analysis in this study included descriptive and bivariate analysis using the SPSS (Statistical Product and Service Solution) version 26.0 program. Numeric variables were expressed as mean ± standard deviation (SD), median, and minimum-maximum value in the descriptive statistics. Categorical variables were expressed in frequency and percentage. All data obtained were tested for normality with the Shapiro-Wilk test. As most of the data were not normally distributed, bivariate analysis was performed using Spearman's nonparametric test to determine the correlation coefficient, the direction of the correlation, and the p-value.

RESULTS

A total of 28 patients were included as the subjects of this study through a consecutive sampling method. The mean age of this study subjects was 55.81 ± 2.13 years old and most of the subjects were males (82.1%). The mean of symptoms onset was 7.52 ± 0.81 hours before hospital admission. Laboratory studies were completed within 3 to 23 hours after symptoms onset with a mean of 9.06 ± 1.11 hours after symptoms onset. The location of AMI in each patient varies with the most common infarct location was on the anterior side (35.7%) and inferior side (25%). A total of 39.3% of the samples experienced AMI on the other side such as anteroseptal, inferoposterior, and others. The mean of PLR and troponin I levels in this study subjects were 233.32 ± 25.54 and 14.44 ± 3.16, respectively. Half of this study subjects had hypertension. Only 17.9% of the subjects had type 2 diabetes, 7.1% had renal insufficiency, and 3.6% had asthma. All of the patients in this study were treated with antiplatelet and statin. Most of the patients were given ACE-inhibitor (92.9%), beta-blocker (82.1%),

anticoagulant (71.4%), and nitrate (67.9%). Mineralocorticoid receptor agonist (MRA) drugs were given to 4 (14.3%) patients and diuretics were given to only 2 (7.1%) patients. Of 28 patients, 25 (89.3%) patients are survivors (Table 1).

Based on the results of bivariate analysis, a moderate positive correlation was detected between PLR and troponin I levels (r = 0.333; p = 0.042; R2 = 0.146) in STEMI patients (Table 2). There was no significant correlation between platelet count and absolute lymphocyte count (ALC) on troponin I levels in the subjects of this study with the correlation coefficients 0.307 (p = 0.056) and -0.111 (p = 0.287), respectively. Onset of chest pain symptoms in STEMI patients was

TABLE 1
Characteristics of research subjects

Variable		n (%)	Mean ± SD	Median (min–max)
Gender	Male	23 (82.1)		
	Female	5 (17.9)		
Age (years)			55.81 ± 2.13	56.00 (31–83)
Occupation	Private employees	11 (39.3)		
	State officials	4 (14.3)		
	Self employed	4 (14.3)		
	Other	9 (32.1)		
Onset of symptoms (hours)			7.52 ± 0.81	7.00 (1.5–19)
Time of laboratory examination (hours)			9.06 ±1.11	9 (3–23)
Location of AMI	Anterior	10 (35.7)		
	Inferior	7 (25)		
	Other	11 (39.3)		
Laboratory findings	Platelet (103/µl)		300.04 ± 12.92	294 (165–461)
	WBC (103/µl)		14.34 ± 0.76	14.2 (6.4–26.2)
	RLC (%)		11.19 ± 0.96	10 (3–25)
	ALC (103/µl)		1.53 ± 0.13	1.41 (0.45–3.5)
	PLR		233.32 ± 25.54	181.82 (114–718.75)
	Troponin I (µg/l)		14.44 ± 3.16	6.86 (0.08–50)
	CK-MB (µg/l)		143.33 ± 35.15	78 (15–945)
	Blood glucose (mg/dl)		156.11 ±15.89	126 (58–363)
	Creatinine (mg/dl)		1.22 ± 0.09	1.1 (0.8–2.9)
	Hb (g/dl)		13.46 ± 0.41	13.7 (8–16.3)
Comorbidities	Hypertension	14 (50)		
	Type 2 DM	5 (17.9)		
	Renal insufficiency	2 (7.1)		
	Asthma	1 (3.6)		
Treatments	Antiplatelet	28 (100)		
	Statin	28 (100)		
	ACEI	26 (92.9)		
	Beta-blocker	23 (82.1)		

Variable		n (%)	Mean ± SD	Median (min–max)
	Anticoagulant	20 (71.4)		
	Nitrate	19 (67.9)		
	MRA	4 (14.3)		
	Diuretic	2 (7.1)		
Mortality	Survivor	25 (89.3)		
	Non-survivor	3 (10.7)		

Abbreviations: N = frequency, WBC = White Blood Cells; RLC = Relative Lymphocyte Count; ALC = Absolute Lymphocyte Count; PLR = Platelet-to-Lymphocyte Ratio; CK-MB = Creatine Kinase-Myocardial Band; Hb = Hemoglobin; DM = Diabetes Mellitus; ACEI = Angiotensin Converting Enzyme Inhibitor; MRA = Mineralocorticoid Receptor Agonist

TABLE 2
Correlation coefficients between PLR, platelet count, lymphocyte count, onset of symptoms, and troponin I levels

Variables	Troponin I		
	r	*p*	R^2
PLR	0.333	0.042†*	0.146
Platelet	0.307	0.056†	
ALC	-0.111	0.287†	
Symptoms onset	0.596	0.000†*	0.256

* Significant (p<0.05); †Spearman
Abbreviations: PLR = Platelet-to-Lymphocyte Ratio; ALC = Absolute Lymphocyte Count

moderately correlated with troponin I levels (r = 0.596; p = 0.000; R2 = 0.256).

DISCUSSION

This study demonstrated that there was a significant positive correlation between PLR and troponin I levels measured in less than 24 hours after symptoms onset. Similar result was also found in a study by Shawky et al which shows a strong correlation between PLR with troponin I levels in non-ST-segment elevation myocardial infarction (NSTEMI) patients.[11] The pathogenesis of STEMI and NSTEMI is more or less similar, starting with the formation of atherosclerotic plaques, plaque destabilization due to the inflammatory process, and thrombosis in the coronary arteries. The cause of thrombosis in STEMI is plaque rupture, but plaque erosion is the more common cause of thrombosis in NSTEMI.[17] This statement is supported by previous studies conducted by Harun et al and Setianingrum et al which revealed that there was no significant difference between the PLR values in STEMI patients and NSTEMI patients.[18,19] The moderate correlation found between PLR values and troponin I levels in STEMI patients may be due to the difference in the inflammatory response degree in each individual. The inflammatory response depends on the characteristics and complexity degree (vulnerability, erosion, and rupture) of the atherosclerotic plaque. The results of previous studies revealed that less complicated and less susceptible atherosclerotic plaques have lower inflammatory responses than the more complicated plaques.[20] In addition, Ritschel et al stated that inflammatory response was associated with the extent of infarct in STEMI patients that was measured by peak troponin T levels.[21] This indicates that smaller infarct area may cause milder inflammatory response, so that it can affect the correlation between PLR values and troponin I levels in STEMI patients.

Another explanation that may also elaborate the moderate correlation between PLR values and troponin I levels in this study is that the physiology of the cardiovascular system such as blood pressure, heart rate, cardiac output, and endothelial function are influenced by circadian rhythms. It is hypothesized that extracardiac factors, such as fluctuating levels of the hormones epinephrine and cortisol, prothrombotic factors, platelet aggregation, and coronary artery flow, influence these cardiovascular events.[22] The activity of the immune system is also known to be influenced by circadian

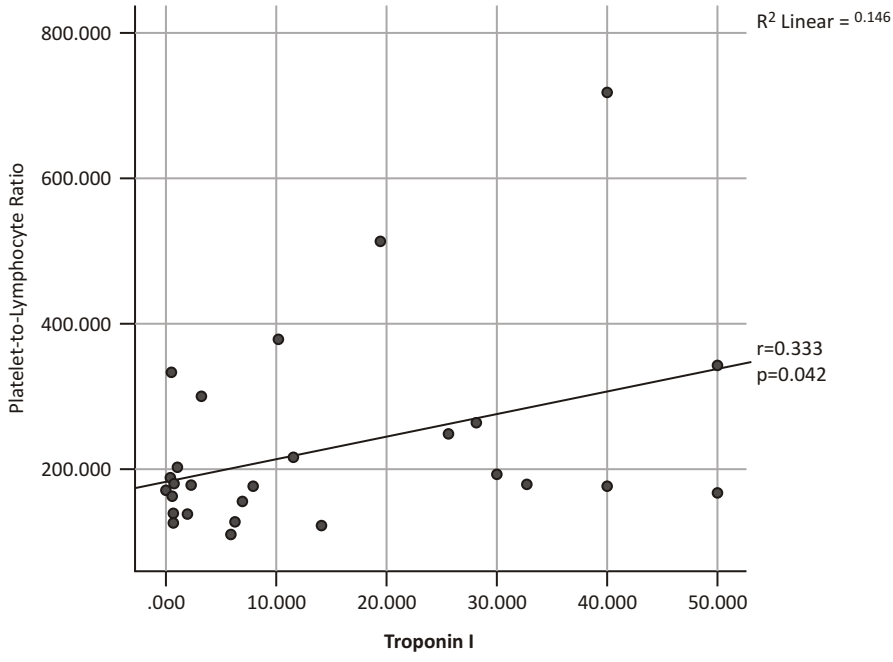

Figure 1. Scatter plot diagram of PLR and troponin I levels in STEMI patients

rhythms. The number of hematopoietic cells, hormones, and cytokines fluctuate depending on the resting and active times of the organism. Previous studies have observed that the number of T and B lymphocytes reached its peak in the resting phase of an organism, whereas levels of cortisol, adrenaline, noradrenaline, and proinflammatory cytokines such as TNF and interleukin-1ß reached their peak in the active phase of an individual.[23,24] A prospective study conducted by Albackr et al demonstrated that morning to noon was the most common symptoms onset time in STEMI patients.[25] Previous studies have revealed that the onset of infarction at the dark-to-light transition of the day is associated with a larger infarct area.[22,26] This phenomenon may occur because the increase in systolic blood pressure and heart rate in the morning can increase the energy and oxygen demand of the heart, whereas the vascular tone of the coronary arteries also increases in the morning. This may result in decreased blood flow and oxygen supply in the coronary arteries. Furthermore, there is also an increase in thrombus formation and platelet aggregation in the morning due to an increase in Plasminogen Activation Inhibitor-1 and various platelet activation markers such as Glycoprotein Ib and P-selectin.[25] In this study, research subjects were admitted to the hospital at various times of the day so that cardiovascular activity and the degree of inflammation that had occurred may be different in each individuals, depending on the circadian rhythm of each individual. Furthermore, the examination of troponin I levels in each sample of this study also varied and was not carried out at a uniform time, whereas troponin levels begin to increase 3-4 hours after the onset of infarction and peak levels are reached after 18-36 hours after the onset of AMI.4 As a result, the troponin I levels obtained for analysis in this study may be less than ideal.

The role of platelets and lymphocytes in the pathogenesis of STEMI is the fundamental of the positive correlation found between PLR and troponin I, where the myocardial damage due to the inflammatory process by platelets and lymphocytes will then be detected by troponin I. Platelets play important roles in atherogenesis and thrombus formation after plaque destabilization. This is supported by the results of previous studies which stated that patients with coronary heart disease had more circulating activated platelets, microparticles derived from platelets, and monocyte-platelet aggregates. The results of the same study also showed an increase in platelet reactivity in patients with coronary heart disease.[27,28] A study by Núñez et al informed that there was a significant decrease in the number of lymphocytes in the acute phase of STEMI, precisely at 6 to 24 hours after symptoms onset. Lymphocytopenia in the acute phase of STEMI may be due to increased apoptosis associated with uncontrolled activation of the immune system. In addition, nonoptimal phagocytosis of the cells undergoing apoptosis will result in secondary necrosis. This will trigger the release of proinflammatory cytokines such as tumoral necrosis factor-α (TNF-α) and interleukin-6 (IL-6) so that this inflammatory process will be further activated.[6]

Troponin regulates the interaction of actin and myosin which facilitate muscle contraction and

relaxation. Troponin consists of 3 subunits, namely troponin C, troponin I, and troponin T. Troponin I and cardiac troponin T are known as troponin subunits which amino acid sequences are typical for the heart muscle, so they are commonly used as cardiac markers that are sensitive and specific for the diagnosis of AMI. Most troponins are bound to myofibrils, but about 7% of troponin T and 3–5% of troponin I circulate freely in the cytoplasm. Therefore, there is an increase in serum troponin levels immediately after myocardial cells damage.[4,12]

Troponin I in less than 24 hours after symptoms onset can be used to predict infarct size in AMI patients. A study by Younger et al revealed that troponin I levels in 12 hours after the occurrence of myocardial infarction had a positive correlation with infarct size as measured using cardiac MRI.[15] This statement is supported by the results of a study conducted by Panteghini et al which stated that troponin I levels within 12 hours after hospital admission were positively correlated with infarct size as measured using Single-Photon Emission Computed Tomography (SPECT).[16] There was a positive correlation detected between PLR and troponin I levels in less than 24 hours after symptoms onset in this study, indicating that PLR may be used to estimate the extent of infarction in STEMI patients.

This study found that there is no significant correlation between platelet count and ALC on troponin I levels. Similar to our findings, previous studies conducted by Mailoa et al stated that there was no significant correlation between platelet count and troponin I levels in Acute Coronary Syndrome (ACS) patients.[29] The results of a study conducted by Badran et al also stated that there was no significant correlation between lymphocyte count and troponin I levels in STEMI patients.[30] PLR is considered better for use as a prognostic marker than platelet count or lymphocyte count alone.[31,32] PLR values are able to describe the occurrence of thrombotic and inflammatory processes at the same time so that it is more beneficial than using individual blood parameters. In addition, PLR is considered more stable than ALC or platelet count because ALC or platelet count alone can be influenced by various physiological and pathological conditions.[33]

There are some limitations in this study. Troponin I levels measured in 96 hours after symptoms onset could not be analyzed in this study because troponin I levels examination and white blood cells (WBC) differential count was not commonly performed within 96 hours after symptoms onset. Another limitation of this study was the differences in comorbidities and symptoms onset in each sample. The examination of troponin I levels was not carried out in a uniform time in this study. As a result of these matters, there were variations in troponin I levels in the study sample.

CONCLUSION

There is a moderate positive correlation between PLR values and troponin I levels observed in less than 24 hours from symptoms onset in STEMI patients.

Based on present study, first, a prospective study with uniform measurement of troponin I levels is needed to verify the usefulness of PLR in determining prognosis and predicting infarct area as represented by troponin I levels following the acute phase (24 hours to 96 hours post-infarction onset). Second, differential WBC count examination should be performed as a routine examination in STEMI patients since blood markers such as PLR may help in establishing patient's prognosis and severity of the patient's illness.

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