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Correlation Between the Neutrophil-to-Lymphocyte Ratio, Monocyte-to-Lymphocyte Ratio, and Platelet-to-Lymphocyte Ratio with Troponin T Levels in Patients with ST-Elevation Acute Myocardial Infarction

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Abstract: Background: Testing for cardiac-specific biomarkers is essential for the timely diagnosis of ST-elevation acute myocardial infarction (STEMI). However, these tests are not yet available in all healthcare facilities. Therefore, a simple test is needed to aid in diagnosis. **Objective:** To determine the correlation between the Neutrophil-to-Lymphocyte Ratio (NLR), Monocyte-to-Lymphocyte Ratio (MLR), and Platelet-to-Lymphocyte Ratio (PLR) with troponin T levels in STEMI patients. **Methods:** An observational analytical study with a cross-sectional design. A total of 58 patients who met the inclusion and exclusion criteria were selected using purposive sampling. Correlation analysis was performed using Spearman's correlation test. The cutoff value was determined using ROC analysis. **Results:** The majority of subjects in this study were male, totaling 48 individuals (82.8%), and were over 50 years old (60.3%). A moderate positive correlation was found between NLR and MLR with troponin T levels ($r = 0.422$; $p < 0.001$ and $r = 0.429$; $p < 0.001$, respectively), while a weak positive correlation was observed between PLR with troponin T levels ($r = 0.334$; $p = 0.01$). **Conclusions:** There is a moderate positive correlation between NLR and MLR and troponin T levels in STEMI patients, and a weak positive correlation between PLR and troponin T levels in STEMI patients.

Keywords: STEMI, NLR, MLR, PLR, Troponin T

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Introduction

ST-elevation acute myocardial infarction (STEMI) is included in the clinical scope of acute coronary syndrome, along with non-ST-elevation myocardial infarction and unstable angina. This condition is characterized by damage to the heart muscle caused by a prolonged lack of blood flow, leading to irreversible death of heart muscle cells. This subsequently results in a significant increase in cardiac markers and ST-segment elevation on the electrocardiogram (PERKI, 2018). Data

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from the World Health Organization in 2019 indicate that coronary heart disease, which includes STEMI, is the leading cause of death globally, accounting for nearly 9 million deaths, or approximately 16% of all deaths worldwide. (WHO, 2020)

Biomarker testing is an important part of the diagnostic process for STEMI. The most commonly used biomarker today is troponin T because it has relatively high sensitivity and specificity (PERKI, 2018). A previous study demonstrated satisfactory sensitivity (67.3%) and specificity (73.8%) of troponin T in detecting cardiomyocyte injury (Sharma et al., 2017). However, this test is not yet widely available across all healthcare facilities. Additionally, the cost of cardiac biomarker testing remains relatively high (Nathaniel *et al*., 2022; Wikananda & Diartoputra, 2023).

Recently, several studies have indicated that hematological profiles, particularly inflammatory markers, demonstrate significant diagnostic potential in coronary heart disease. The neutrophil-to-lymphocyte ratio (NLR), monocyte-to-lymphocyte ratio (MLR), and platelet-to-lymphocyte ratio (PLR) are inflammatory markers that have been extensively studied for their role as markers of thrombosis and inflammation in STEMI. (Nathaniel et al., 2022; Wikananda & Diartoputra, 2023) Neutrophils, monocytes, and platelets are nonspecific inflammatory markers in myocardial necrosis (Fang et al., 2015), whereas lymphocytes act as regulatory cells that control the inflammatory response and prevent apoptosis of cardiac muscle cells. (Evelyn et al., 2022) Therefore, the NLR, MLR, and PLR values respectively reflect the ratio of neutrophils, monocytes, and platelets as pro-inflammatory components and lymphocytes as protective and anti-inflammatory components in cardiac muscle cells. Elevated NLR, MLR, and PLR values reflect the severity of inflammation and are associated with increased troponin T levels and a larger area of necrosis. Routine laboratory tests to obtain NLR, MLR, and PLR values are easily accessible and do not incur significant costs (Nathaniel et al., 2022; Wikananda & Diartoputra, 2023).

Research conducted by Wikananda & Diartoputra (Wikananda & Diartoputra, 2023) found an association between NLR and PLR values and high-sensitivity troponin T. Evelyn et al. (2022) in their investigation also confirmed a moderate correlation between the PLR score and troponin levels recorded within 24 hours of symptom onset in STEMI. Other studies have shown that patients with high MLR scores exhibit elevated troponin T levels (Chen et al., 2019). This indicates that these parameters can serve as diagnostic aids in detecting myocardial infarction. However, previous studies have generally examined these inflammatory marker ratios individually or in limited combinations and a comprehensive evaluation of NLR, MLR, and PLR together in relation to troponin T levels, particularly among STEMI patients in Indonesia remains limited. Based on these findings, the researchers aim to investigate the relationship between NLR, MLR, and PLR, particularly in the context of ST-elevation conditions accompanied by a significant increase in troponin T levels.

Methods

This study is a cross-sectional study conducted at the clinical pathology laboratory and medical records department of Abdoel Wahab Sjahranie General Hospital in Samarinda in June 2023. Sampling in this study was performed using non-probability sampling, specifically purposive sampling. Inclusion criteria included STEMI patients who were hospitalized between January and December 2023, were recorded in medical records, and had complete research variables. Additionally, exclusion criteria included patients with acute or chronic infections, patients with a history of malignancy, and patients suffering from kidney disorders, including both chronic kidney disease and acute kidney injury (AKI). Based on the calculation of the minimum sample size formula for correlational tests, a minimum sample size of 38 was determined.

The study subjects were identified by reviewing the medical records of patients who met the inclusion criteria. The collected data comprised demographic characteristics, complete blood count parameters (neutrophils, lymphocytes, monocytes, and platelets), and serum troponin T levels. NLR, MLR, and PLR were calculated by dividing the absolute neutrophil count, absolute monocyte count, and the absolute platelet count, respectively, by the absolute lymphocyte count. Correlation analysis of the study variables was performed using Spearman's correlation test in SPSS Statistics 27 software. The determination of cut-off values for NLR, MLR, and PLR, along with the sensitivity and specificity of troponin T at levels >30 pg/mL was conducted via Receiver Operating Characteristic (ROC) curve analysis. This study has obtained ethical approval from the Health Research Ethics Committee (KEPK) of Abdoel Wahab Sjahranie General Hospital, Samarinda, number 451/KEPK-AWS/VI/2024**.**

Results and Discussion

A total of 88 subjects with acute ST-segment elevation myocardial infarction (STEMI) requiring hospitalization were included in the study. Upon further review, incomplete data were found in 30 subjects, resulting in 58 subjects with complete data. The mean age of the subjects in this study was 54.10 years, and the majority were male (82.8%). Correlation tests between

the NLR, MLR, and PLR parameters and troponin T levels revealed a significant correlation with moderate positive correlation strength for NLR and MLR and weak positive correlation strength for PLR with troponin T levels. Table 5 shows the results of the ROC curve analysis, as well as the sensitivity and specificity values of NLR, MLR, and PLR for troponin T levels. The NLR cutoff value was 2.75, with a sensitivity of 72.1% and a specificity of 73.3%. MLR, with a cutoff of 0.25, has a sensitivity of 67.4% and a specificity of 66.7%, and PLR, with a cutoff of 109.66, has a sensitivity of 69.8% and a specificity of 73.3%.

The results show that the average age of the subjects was 54.10 years, with the youngest and oldest being 29 and 79 years, respectively (Table 1). This study reports that the majority of patients with STEMI were over 50 years of age (60.3%). These results are consistent with a study conducted by Amrullah et al., which reported that the age group with the highest frequency among MI patients was 53–62 years, accounting for 30.6%.

Table 1. Laboratory Result Characteristics

Characteristics	N= 58
Age (years), mean (SD)	54,10 (10,31)
≤40 years, n (%)	6 (10,3)
41–50 years, n (%)	17 (29,3)
>50 years, n (%)	35 (60,3)
Gender, n (%)	
Male	48 (82,8)
Female	10 (17,2)
Platelets, median (range)	250 (3,95-500)
Leukocyte, median (range)	10,11 (5,74-25,14)
Neutrophils, median (range)	6,80 (2,90-21,80)
Monocytes, median (range)	0,68 (0,07-2)
Lymphocytes, median (range)	2.15 (0,63-6,73)
Hemoglobin, median (range)	13,85 (8,6-18,20)
NLR, median (range)	3,09 (0,77-14,90)
MLR, median (range)	0,29 (0,07-1,32)
PLR, median (range)	119,13 (2,11-351,52)
HPR, median (range)	0,06 (0,02-2,58)
LPR, median (range)	0,04 (0,02-2,10)
Troponin T, median (range)	99 (6-7089)

PLR = Platelet-to-Lymphocyte Ratio; NLR = Neutrophil-to-Lymphocyte Ratio; MLR = Monocyte-to-Lymphocyte Ratio; HPR = Hemoglobin-to-Platelet Ratio; LPR = Leukocyte-to-Platelet Ratio

Table 2. Correlation between PLR, NLR, MLR, HPR, and LPR parameters and troponin T levels

Correlation	Troponin T Levels
NLR (r;p)	0,422 ; <0,001
MLR (r;p)	0,429 ; <0,001
PLR (r;p)	0,334 ; 0,01
HPR (r;p)	-0,126 ; 0,346
LPR (r;p)	0,098 ; 0,466

Subgroup analysis based on age and sex revealed a moderate positive correlation between NLR and troponin T in the 41–50 age group and a moderate positive correlation between PLR and MLR and troponin T in subjects aged >50 years. Subgroup analysis based on gender revealed a strong positive correlation between NLR and troponin T in female subjects and a positive correlation between NLR and MLR with troponin T in male subjects with a weak correlation strength. Additional results also included a correlation test between the HPR and LPR variables, and no significant correlation was found either in the analysis of the subjects as a whole or in the subgroup analysis based on age and gender.

Table 3. Correlation of PLR, NLR, MLR, HPR, and LPR parameters with troponin T levels by age

	≤40 years	41 – 50 years	>50 years
	Troponin T	Troponin T	Troponin T
NLR (r;p)	0,771 ; 0,072	0,559 ; 0,020	0,324 ; 0,058
MLR (r;p)	0,087 ; 0,870	0,455 ; 0,066	0,436 ; 0,009
PLR (r;p)	0,314 ; 0,544	0,270 ; 0,295	0,418 ; 0,012
HPR (r;p)	0,058 ; 0,913	0,106 ; 0,686	-0,305 ; 0,075
LPR (r;p)	0,143 ; 0,787	0,357 ; 0,160	-0,088 ; 0,613

Table 4. Correlation of PLR, NLR, MLR, HPR, and LPR parameters and troponin T levels by gender

Correlation	Male	Female
	Troponin T	Troponin T
NLR (r;p)	0,391; 0,006	0,648 ; 0,043
MLR (r;p)	0,398 ; 0,005	0,614 ; 0,059
PLR (r;p)	0,270 ; 0,063	0,430 ; 0,214
HPR (r;p)	-0,125 ; 0,397	0,052 ; 0,887
LPR (r;p)	0,179 ; 0,223	-0,093 ; 0,799

Table 5. Cut-off Values, AUC, Sensitivity, and Specificity of NLR, MLR, and PLR with Troponin T

Variable	Cut-off	AUC	Sensitivity	Specificity
NLR	2,75	0,791	72,1%	73,3%
MLR	0,25	0,764	67,4%	66,7%
PLR	109,66	0,712	69,8%	73,3%

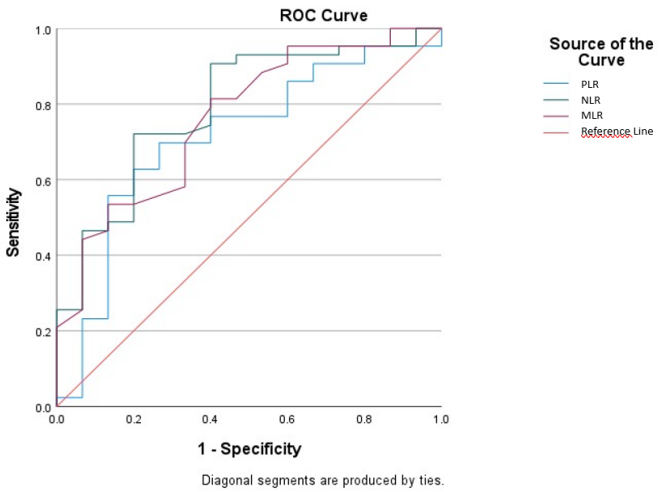


Figure 1. ROC Curve Analysis of NLR, MLR, and PLR with Troponin T Levels

Age and Gender Characteristics

As part of the aging process, arterial blood vessels experience increased stiffness caused by a decrease in elastin fiber levels and the accumulation of collagen fibers. Enzymatic activities, such as matrix metalloproteinases (MMPs), contribute to the degradation of elastin fibers, which are subsequently replaced by collagen fibers with stiffer biomechanical properties. Based on research findings, aging correlates with the development of atherosclerotic plaques through intrinsic and extrinsic mechanisms. Intrinsic. factors include mitochondrial damage and dysfunction in vascular cells resulting from aging, wherein damaged mitochondria release their internal components into the cytosol, thereby triggering an inflammatory response. Meanwhile, extrinsic factors are associated with increased production of interleukin-6 (IL-6) with advancing age, which subsequently promotes myeloid cell differentiation in the bone marrow and the recruitment of inflammatory cells, thereby facilitating the formation of atherosclerotic plaques on the blood vessel walls (Lilly, 2021).

The majority of subjects in this study were male (82.8%) (Table 1). This finding is consistent with the results of other studies, such as the study by Simanungkalit & Supit (2022), which reported that males were more prevalent in cases of STEMI, accounting for 55.35%. This condition is associated with smoking habits among men, which is a predisposing factor for atherosclerotic disease and can be explained through several mechanisms, including lipoprotein modification, reduced HDL levels, endothelial dysfunction, and increased oxidative stress (Lilly, 2021). Additionally, levels of androgen hormones, which play a protective role against the atherosclerotic process, tend to decline after peaking at ages 20–24. This decline may increase the tendency for atherosclerotic plaque formation and contribute to the risk of cardiovascular disease, including STEMI. Meanwhile, female subjects accounted for only 17.2% of the total sample in this study. This lower proportion relative to men can be explained by the atheroprotective effect of estrogen. In premenopausal women, physiologic estrogen levels raise high-density lipoprotein (HDL) and lower low-density lipoprotein (LDL) levels. Estrogen also exerts antioxidant and antiplatelet effects and improves endothelium-dependent vasodilation, all of which contribute to a lower incidence of coronary events compared with men of the same age. However, this protective effect declines sharply after menopause, at which point the incidence of coronary events in women approaches that seen in men (Lilly, 2021).

The Relationship Between NLR and Troponin T

A correlation analysis was conducted to evaluate the relationship between the neutrophil-to-lymphocyte ratio (NLR) and troponin T levels. The results showed a moderate positive correlation between the two parameters. Furthermore, a Receiver Operating Characteristic (ROC) curve analysis was performed for the troponin T variable. The NLF cutoff value identified via the ROC curve was 2.75, with a sensitivity of 72.1% and a specificity of 73.3%. These findings align with a study by Korkmaz et al. (2015), which demonstrated the potential of the neutrophil-to-lymphocyte ratio as an early predictor of ischemic cardiomyopathy, given the role of these two cellular populations in modulating the acute inflammatory response. Furthermore, the study reported that subjects experiencing persistent angina with an elevated neutrophil-to-lymphocyte ratio (NLR) at initial presentation at a healthcare facility showed a tendency toward elevated troponin levels.

Prolonged ischemia triggers necrosis in myocardial cells. This cell death then triggers an acute inflammatory response through increased production of pro-inflammatory cytokines. These cytokines play a role in recruiting polymorphonuclear cells, particularly neutrophils, as well as macrophages to the damaged area. This inflammatory process is also accompanied by a decrease in the number of lymphocytes, which have anti-inflammatory and protective functions for heart muscle cells. Consequently, the neutrophil-to-lymphocyte ratio (NLR) will increase significantly and peak approximately 12–14 hours after the onset of the infarction (Hartopo et al., 2015).

The researchers then conducted subgroup analyses of the studied variables based on age and sex categories. In the age-based analysis, a strong correlation was found between the NLR and troponin T levels in female subjects ($r = 0.648$; $p = 0.043$) and a weak correlation in male subjects ($r = 0.391$; $p = 0.006$). This finding is associated with smoking habits in men. Smoking is known to increase the number of peripheral blood neutrophils compared to other cell types. Smoking is known to have various immunological effects, including reduced activity of killer T cells and increased numbers of T4 and T8 lymphocytes. This mechanism is linked to airway inflammation caused by exposure to cigarette smoke, which is associated with the release of various chemotactic factors for blood cells and elevated cytokine levels, particularly IL-6 and TNFα. This leads to instability in neutrophil and lymphocyte counts in men, resulting in a tendency toward a specific direction, so that the statistical analysis results do not show as strong a correlation as in female subjects, whose complete blood counts tend to be more stable (Korkmaz et al., 2015).

Relationship Between MLR and Troponin T

The monocyte-to-lymphocyte ratio was found to have a moderate positive correlation with troponin T levels. Receiver operating characteristic (ROC) curve analysis for troponin T indicated that the cutoff value for the monocyte-to-lymphocyte ratio (MLR) is 0.25, with a sensitivity of 67.4% and a specificity of 66.7%. Monocytes play a fundamental role in the innate immune response and also contribute to endogenous inflammatory processes. Monocytes and their derivatives play a significant role in the initiation and progression of atherosclerotic plaques. The migration of monocytes from the bloodstream into tissues occurs in response to inflammatory stimuli, after which they differentiate into dendritic cells, macrophages, and foam cells. Subsequently, these cells induce the production of pro-inflammatory cytokines, matrix metalloproteinases, and reactive oxygen species (ROS), all of which contribute to the progression of atherosclerotic lesions, ultimately leading to plaque rupture and the formation of intracoronary thrombi (Chen et al., 2019).

In cases of STEMI, proinflammatory monocytes increase immediately after neutrophil infiltration and peak 3–5 days after the infarction, which is simultaneously accompanied by a decrease in lymphocyte count due to increased apoptosis of these cells. Therefore, the combination of increased monocytes and decreased lymphocytes occurring concurrently with troponin T extravasation may serve as a biomarker reflecting the elevated troponin T levels themselves (Chen et al., 2019).

Relationship Between PLR and Troponin T

This study identified a weak positive correlation between the platelet-to-lymphocyte ratio (PLR) and troponin T levels. Receiver Operating Characteristic (ROC) curve analysis for troponin T yielded a PLR cut-off value of 109.66, with a sensitivity of 69.8% and a specificity of 73.3%. These findings aligns in direction with the investigation conducted by Evelyn et al. (2022), which indicated that the PLR can serve as a specific predictor of acute myocardial necrosis. That previous study reported a moderate positive correlation ($p=0.042$; $r=0.333$) between PLR and troponin levels in patients with STEMI monitored during the first 24 hours after symptom onset. Furthermore, these results align with the study by Wikananda & Diartoputra (2023), which reported an association between PLR and high-sensitivity troponin T ($p=0.004$; $r=0.284$).

Myocardial necrosis causes damage that triggers an inflammatory response and activates platelets through the release of inflammatory mediators such as IL-1 and IL-3, which stimulate megakaryocyte proliferation and ultimately increase the number of platelets in circulation (Wikananda & Diartoputra, 2023). Subsequently, platelets play a role in the inflammatory response through two mechanisms: the release of inflammatory mediators and platelet-lymphocyte interactions. The inflammatory mediators released include serotonin, histamine, chemokines, cytokines, interleukins, and matrix metalloproteinases (MMPs). Meanwhile, platelet-lymphocyte interactions contribute to the activation and infiltration of leukocytes into inflamed tissues (Fang et al., 2015; Lilly, 2021; Wikananda & Diartoputra, 2023). In turn, lymphocytes play a role in regulating the inflammatory response and suppressing excessive inflammatory processes (Evelyn et al., 2022). Additionally, lymphocytes act as protectors of the infarct region by preventing the recruitment of inflammatory cells and cytokines, and play a role in post-infarction myocardial healing (Nathaniel et al., 2022). When an inflammatory response occurs, there is an increase in the secretion of cortisol and catecholamine hormones. Elevated levels of these hormones lead to increased apoptosis of lymphocytes, resulting in a decrease in lymphocyte count. This explains why the reduction in lymphocyte count during post-myocardial infarction inflammation is associated with the severity and extent of the infarct in the heart muscle (Shawky & Radwan, 2018; Wikananda & Diartoputra, 2023).

In the subgroup analysis conducted by the researchers, no correlation was found between NLR, MLR, and PLR in patients aged ≤40 years. It remains unclear why these results were obtained, as literature studies suggest that younger individuals actually have more stable platelet, neutrophil, and lymphocyte counts compared to the elderly, due to immunosenescence—a process characterized by a decline in adaptive immune system function (Guo et al., 2018). This condition tends to cause changes in cell count and may lead to a specific statistical trend. However, the study results actually showed a correlation in elderly subjects (>50 years), which contradicts the researchers' hypothesis. The researchers suspect that the results obtained are related to the small number of young adult subjects (≤40 years) analyzed, which did not provide a statistically representative data set.

Relationship with Other Parameters

Additional findings from this study involve investigating correlations between inflammatory markers that are also hypothesized to have prognostic implications in the diagnosis of coronary heart disease, namely platelet, hemoglobin, and leukocyte components (hemoglobin-to-platelet ratio [HPR] and leukocyte-to-platelet ratio [LPR]).

Most studies indicate that the proliferation of pro-inflammatory cytokines as a consequence of the inflammatory response triggers a reduction in iron supply from the bone marrow for erythropoiesis, which

in turn leads to a decrease in hemoglobin levels. Furthermore, the release of pro-inflammatory cytokines such as IL-1, IL-6, IFNγ, TNFα, and TGFβ when acute coronary syndrome (ACS) manifests can cause various effects, including accelerated erythrocyte degradation and suppression of erythropoietin production.

It is known that STEMI is also associated with elevated platelet counts. During hematopoiesis, the transcription factor GATA-2, which is expressed in progenitor cells, is overexpressed in response to increased IL-6 levels. This upregulation of the transcription factor inhibits erythrocyte development but, conversely, enhances platelet production (Ikhsan et al., 2021).

Additionally, the leukocyte-to-platelet ratio (LPR) also reflects a key process in the pathophysiology of ACS. In the early stages, leukocytes present within the lipid core of atherosclerotic plaques undergo activation due to endothelial damage, which subsequently stimulates the release of pro-inflammatory cytokines and ultimately leads to thrombus formation. As previously described, it has been reported that platelets also contribute to the pathogenesis of acute coronary syndrome through inflammatory activation pathways. Platelet activation triggers leukocyte-platelet adhesion and interactions with various proinflammatory cytokines that contribute to the progression of the atherosclerotic process. Leukocyte-platelet adhesion also plays a role in attracting other leukocytes to the site of plaque rupture, which exacerbates ischemic conditions until necrosis occurs in the myocardial muscle. This ultimately affects the release of cardiac enzymes such as troponin T (Ikhsan et al., 2021; Wikananda & Diartoputra, 2023).

The hypothesis proposed by the researchers, based on a literature review, regarding the correlation between HPR and LPR values and elevated troponin T levels in patients with STEMI was not confirmed in this study. The findings apply to both the overall analysis of the subjects and the subgroup analysis based on age and gender categories. These observed differences are thought to be caused by variations in the time of symptom onset among study subjects with STEMI, resulting in highly variable kinetics of the hematological parameters measured upon arrival at the healthcare facility. Furthermore, the variable inflammatory response among individuals also contributed to differences in the rate of change in hematological parameters as the final outcome. Additionally, this study did not consider the patients' hydration status, even though this condition can affect hemoglobin and hematocrit concentrations. The possibility of certain hematological abnormalities that could affect hemoglobin, platelet, and leukocyte levels in patients was also not accounted for.

Limitations of this study include the small sample size and limited access to secondary data in the form of medical records with incomplete data. Additionally, the researchers did not present data that could be analyzed to examine correlations with other variables, such as BMI, classic risk factors, blood pressure, and so on.

Conclusion

This study concluded that there is a correlation between NLR, MLR, and PLR and troponin T levels in patients with ST-elevation myocardial infarction (STEMI). However, further investigation is still needed to confirm the causal relationship between NLR, MLR, and PLR and troponin T levels, particularly in younger and elderly STEMI patients. In addition, further research is also needed on the correlation between other inflammatory parameters that have been proven to have prognostic value in coronary heart disease and troponin T levels so that they can be considered in the use of simple parameters as diagnostic tools, especially in facilities with limited resources.

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