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ASSOCIATION BETWEEN NEUTROPHIL-TO-LYMPHOCYTE RATIO AND TROPONIN LEVELS IN PATIENTS WITH ACUTE CORONARY SYNDROME

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Abstract

Background: Acute coronary syndrome is strongly associated with myocardial injury and inflammatory response. In this study, we sought to examine the relationship between NLR and troponin levels in patients with acute coronary syndrome.
Methods: In this retrospective study, we analyzed clinical data obtained from the records of 410 patients with acute myocardial infarction, who had been hospitalized in our institution during January 1, 2025 and December 31, 2025. Demographic information as well as data regarding neutrophil count, lymphocyte count, NLR, and troponin were extracted from patient files. Logarithmic transformation was done because troponin values were not normally distributed. For correlation analysis, both Pearson and Spearman correlation tests were used for examining the relationship between NLR and troponin.
Results: The mean age of patients was 63.3 ± 10.5 years, and 75.1% of the sample was males. The prevalence of STEMI and NSTEMI were 58.8% and 41.2%, respectively. The mean NLR and troponin value was 6.1 ± 3.8 and 9099.9 ± 9948.0, respectively. A significant positive relationship was noted between NLR and troponin, according to Pearson correlation analysis ($r = 0.63$, $P < .001$) and Spearman correlation analysis ($\rho = 0.63$, $P < .001$).
Conclusion: The association between increased NLR levels and increased troponin levels was statistically significant among the participants suffering from acute coronary syndrome. The findings further demonstrate the association between systemic inflammation and myocardial damage, providing evidence for using NLR as a useful and cost-effective auxiliary marker for the early diagnosis of acute myocardial infarction.

Keywords: Acute coronary syndrome, neutrophil-to-lymphocyte ratio, troponin, myocardial infarction, inflammation

1. Introduction

Acute Coronary Syndrome (ACS) is currently one of the major diseases that lead to morbidity and mortality all around the world. However, even with the development of new diagnostic and therapeutic options, ACS still continues to present a serious medical challenge in contemporary practice[1]. ACS includes a variety of conditions, which are caused by acute myocardial ischemia and include STEMI, NSTEMI, and unstable angina[2]. Effective and timely diagnostic and treatment measures are vital for minimizing tissue damage and improving patient outcomes.
From this point of view, the role of the serum biomarkers in the detection of myocardial injury becomes crucial. However, such markers as aspartate aminotransferase (AST), creatine kinase (CK), creatine kinase-MB (CK-MB), and lactate dehydrogenase (LDH) became obsolete because of their low sensitivity and specificity. On the other hand, cardiac troponins are currently considered the ideal diagnostic biomarkers for myocardial infarctions due to their higher sensitivity and specificity[3,4]. Troponins that exist under normal conditions are primarily bound to the cellular contractile proteins of cardiomyocytes; a limited number of troponins can be found freely dissolved in the intracellular fluid. Troponin release from cardiomyocytes

occurs in case of myocardial injuries; thus, increased troponin levels in the blood indicate the presence of myocardial injury in such conditions as myocardial infarctions, myocarditis, cardiac trauma, and myocardial surgeries. Troponins typically start rising about 4-6 hours following myocardial injury, reach their peak values around 24 hours, and persist for 7-10 days.

Apart from the myocardial necrosis markers, there has been considerable focus on the contribution of inflammation to the pathogenesis of ACS. The current consensus is that atherosclerosis is an inflammatory condition involving endothelial dysfunction, lipid deposition, vessel remodeling, and the presence of inflammatory cells in the walls of the arteries. It has become clear that inflammation is not only involved in atherosclerotic plaque development and progression but in addition acts as a mechanism for plaque destabilization and plaque rupture that cause ACSs. Inflammation acts via several mediators released from neutrophils including reactive oxygen species, cytokines, matrix metalloproteinases, and neutrophil extracellular traps. All of these factors are responsible for endothelial damage and thrombosis, leading to plaque instability. There are studies indicating that neutrophil activation and its subsequent inflammation are directly responsible for the development of atherosclerotic cardiovascular disease[6,7,8].

From the aforementioned background, some straightforward hematologic parameters have been studied as potential tools to evaluate cardiovascular risk and prognosis. Among these, the neutrophil-to-lymphocyte ratio (NLR) is one example of a feasible, cheap, and accessible inflammatory marker obtained from complete blood count results. This index represents the relationship between neutrophil-related inflammation and lymphocyte-mediated immune response. High neutrophil count has been implicated in the development of increased inflammatory and thrombotic tendencies, while low lymphocyte counts have been correlated with poor immune modulation and worse cardiovascular outcome. As an inflammatory marker, NLR might provide more clinically relevant information than neutrophil and lymphocyte counts individually. Some research reports showed the link between high NLR levels and the degree of coronary artery disease and cardiovascular morbidity and mortality among ACS patients. Moreover, it has also exhibited significant correlation with known cardiovascular risk stratification scales such as SYNTAX, GRACE, and TIMI scores[9].

Despite the extensive studies on the role of NLR in different medical disciplines such as infectious diseases, inflammation, and cancers, NLR is still an evolving entity within the field of cardiology. The specific effect of systemic inflammation burden denoted by NLR on markers of myocardial damage like troponin has not yet been properly studied, particularly in clinical practice settings of patients with ACS.

Hence, the purpose of this study is to assess the link between NLR and troponin in the context of ACS.

2. Methods

This retrospective observational study was performed at a tertiary cardiology center and comprised subjects admitted to the hospital during the period from January 1, 2025, through December 31, 2025, with a definitive diagnosis of myocardial infarction, including ST-elevation myocardial infarction (STEMI) and non-ST-elevation myocardial infarction (NSTEMI). The diagnosis of acute coronary syndrome was confirmed based on clinical, electrocardiographic, and biochemical findings according to existing diagnostic standards.

Subjects, whose referral was made from another medical facility and lacked complete baseline laboratory data, were excluded from the study. Furthermore, subjects undergoing immediate percutaneous coronary intervention before their initial laboratory tests were taken were also excluded from the analysis. Subjects with elective admission for coronary angiography or any elective cardiovascular procedure were also excluded. To avoid possible confounding influence

on inflammation parameters, individuals with current infection, chronic inflammation, blood disorders, cancer, or autoimmune conditions were excluded from analysis.
The demographic and clinical characteristics such as age, gender, and final diagnosis were recorded from electronic health record systems and database files. Laboratory characteristics involved the absolute number of neutrophils, absolute number of lymphocytes, and troponin levels assessed in the early stage of hospital stay. Based on the clinical scenario, and depending on hospital procedures, blood samples were drawn either on admission or just after coronary interventions.
The neutrophil-to-lymphocyte ratio (NLR) was estimated based on the absolute neutrophil count and absolute lymphocyte count derived from peripheral venous blood samples. The assessment of troponin levels was conducted using laboratory procedures normally used in hospitals in the assessment of heart damage.
As troponin levels showed significant skewness, the values were transformed using logarithm prior to analysis for better normalization of the values. The continuous variables were reported as mean ± standard deviation, while categorical variables were reported as frequencies and percentages.
The correlation between the NLR and the troponin levels was tested by employing the Pearson and Spearman correlation tests. The Pearson correlation test was used for the determination of the linearity of the relationship between continuous variables after log transformation while the Spearman correlation test was used in addition to establish the consistency of the correlation irrespective of the distribution of the data. A P value of less than 0.05 in the 2-tailed test was taken as significant.
This study was approved by the Ethics Committee of the Faculty of Medical Sciences 12.05.2026, decision number 22-1432/1. The study was conducted in accordance with the principles of the Declaration of Helsinki. Because of the retrospective design of the study, the requirement for informed consent was waived by the Ethics Committee.

3. Results

A total of 410 patients with a diagnosis of acute myocardial infarction participated in the study. The average age of the study subjects was 63.3 ± 10.5 years. The majority of the participants were males (308 patients, 75.1%), and 102 patients (24.9%) were females. According to the clinical findings, 241 patients (58.8%) had STEMI, whereas 169 patients (41.2%) were found to have NSTEMI.
The mean value of neutrophil-to-lymphocyte ratio (NLR) for the entire group of participants was 6.1 ± 3.8. The average concentration of serum troponin was 9099.9 ± 9948.0. The higher the values of NLR, the higher were the levels of troponins, indicating that there may be an association between systemic inflammation and myocardial damage. The demographic and laboratory parameters of the study subjects are presented in Table 1.
Correlation analysis showed a statistically significant positive relationship between the NLR and troponin level. Pearson correlation analysis showed a strong positive relationship between the two ($r = 0.63$, $P < .001$). Likewise, a Spearman correlation analysis showed the same level of relationship ($\rho = 0.63$, $P < .001$), suggesting that the established relationship was consistent even with different statistical techniques. These results provide evidence on the stability of the relationship between inflammation burden and myocardial damage in patients with ACS. The relationship between NLR and log-transformed troponin levels is depicted in Figure 1 using scatter plot analysis.

Table 1. Baseline demographic and laboratory characteristics of the study population.

Variable	Total patients, n	Age	Male sex, n(%)	Female sex n(%)	STEMI, n(%)	NSTEMI, n(%)	NLR	Troponin
Value	410	63.3 ± 10.5	(308, 75.1%)	102, (24.9%)	241, (58.8%)	169, (41.2%)	6.1 ± 3.8	9099.9 ± 9948.0

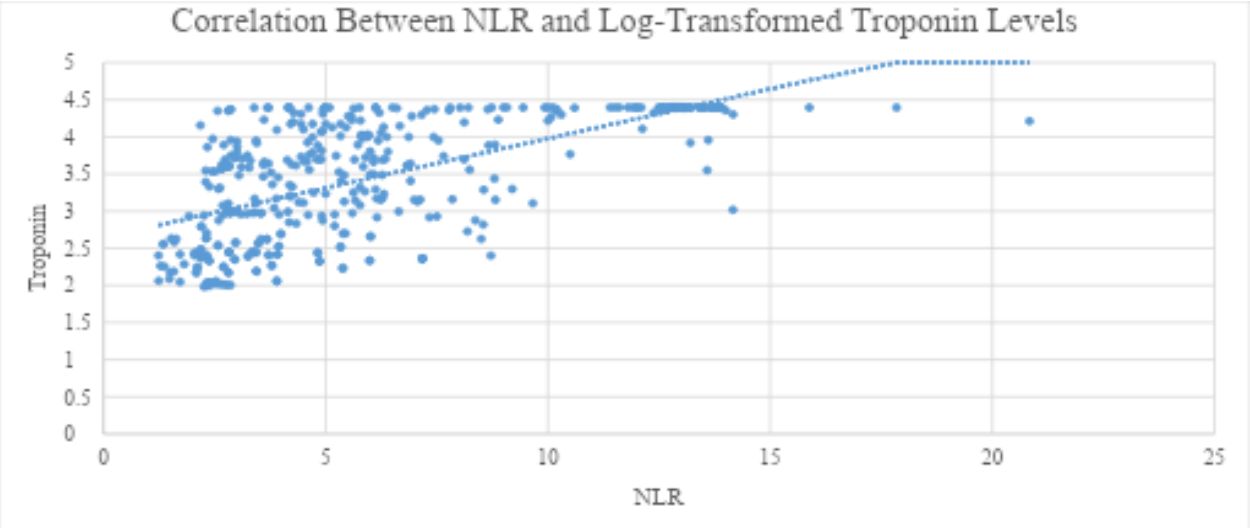

Figure 1. Scatter plot showing the correlation between NLR and log-transformed troponin levels.

4. Discussion

In the current study, it was noted that there exists a positive correlation between NLR and troponin concentration among acute myocardial infarction patients. Notably, the obtained association between the two parameters was consistent when applying both Pearson and Spearman methods of correlation analysis. In general, the findings indicate that increased inflammation in the body is linked to higher myocardial damage among acute coronary syndrome patients.

These results seem biologically feasible and in accordance with the current knowledge on the pathogenesis of ACS. ACS is not regarded only as the result of coronary artery obstruction anymore, but as an intricate inflammatory process, which includes endothelial dysfunction, unstable plaques, and immune system activation. There is compelling evidence that neutrophil-driven inflammation contributes to the onset and progression of atherosclerosis in various clinical and genetic researches, suggesting that neutrophils play an active role in atherosclerosis, and are not just part of the body's secondary reaction to inflammation. In this regard, NLR can indicate the balance between inflammatory activation and immunological regulation and, hence, indirectly reflect the magnitude of inflammation accompanying myocardial damage[10].

Moreover, our results are also similar to earlier investigations of the correlation between NLR and signs of myocardial necrosis in ACS. Specifically, Altun et al. found out that a high concentration of high-sensitive troponin T, along with higher NLR levels, were highly correlated with increased complexity of ACS. In another study, Awan et al. concluded that a significant correlation existed between elevated levels of NLR and troponin-I in patients who were suspected of having non-ST-elevation ACS. The study conducted by Tahto et al. further confirmed the positive correlation between NLR and signs of myocardial necrosis among

patients suffering from ACS. Overall, these studies confirm the notion that NLR is not only an unspecific marker of inflammation but also a sign of myocardial injury[11,12,13].
One of the strengths of the current study is the consistency of the relationship even in spite of the variations seen in practice. The level of troponins is a dynamic biomarker which fluctuates considerably over time, especially after injury to the heart muscle. In our study, the level of troponins was assessed at different intervals depending on the patient's presentation. Some patients had their blood samples analyzed at the time of admission while others had theirs taken shortly after treatment. Though these variations could have affected the levels of troponins, the relationship between NLR and troponins was still significantly consistent.
From the point of view of a physician, these results indicate that NLR can contribute to the early evaluation of acute coronary syndrome patients. As NLR is a cheap test with immediate availability and routine determination as part of a complete blood count, it can be viewed as a convenient complementary inflammatory biomarker for clinical use in emergencies or cardiological departments. Though NLR cannot serve as a substitute for cardiac troponins, it can act as an additional indicator for assessing the inflammation process associated with heart damage.
There are several limitations in the current study that must be mentioned. First, the retrospective and single-center approach to conducting research can pose a threat to generalizability of results obtained during this project. Second, the sampling time was not completely controlled since the tests for troponin level were carried out following clinical guidelines within the hospital settings. This can contribute to some bias regarding the level of measured troponin. Additionally, other inflammatory markers and patients' future health conditions were not considered within the scope of this work. Regardless of the above limitations, the current paper managed to show a significant relationship between NLR and troponin level among patients with acute myocardial infarction.

5. Conclusion

The present study revealed the existence of a significant positive correlation between the neutrophil-to-lymphocyte ratio and troponin concentration in patients with ACS. In other words, higher values of NLR corresponded to an increase in troponin concentration, confirming the presence of a relation between systemic inflammation and the degree of myocardial damage. Consistency of results obtained through various statistics techniques adds additional credibility to this relationship.
Due to its cheapness, widespread availability, and fast access through blood analysis, NLR may be used as a supplemental marker for evaluating the degree of inflammation in the process of diagnosing ACS at the early stages of the disease. However, despite the advantages of using NLR, it is not suggested as a substitute for currently recognized markers such as troponin.
There is a need for additional prospective multicenter studies in order to further clarify the significance of NLR as a predictive tool in patients with ACS.

Human Ethics and Consent to Participate declarations: not applicable
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Highlights

- Neutrophil-to-lymphocyte ratio was significantly associated with troponin levels in patients with acute coronary syndrome.
- Both Pearson and Spearman correlation analyses showed a consistent positive relationship between NLR and troponin.

NLR may serve as a simple, inexpensive, and readily available auxiliary inflammatory marker in acute myocardial infarction.

The findings support the relationship between systemic inflammation and myocardial injury in acute coronary syndrome.

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Figure Legends

Figure 1. Scatter plot demonstrating the positive correlation between neutrophil-to-lymphocyte ratio (NLR) and log-transformed troponin levels in patients with acute coronary syndrome.