

The Predictive Value of Neutrophil/Lymphocyte Ratio for CK-MB Elevation in Myocardial Infarction: A Study in Syrian ACS Patients

Issa Yusuf^{1*}, Rama Ibrahim^{1*}

¹ Department of Biochemistry and Microbiology, Faculty of Pharmacy, Tishreen University, Latakia Syria

ABSTRACT

Acute coronary syndrome (ACS) is a serious cardiovascular condition associated with a high mortality rate. It typically arises from the rupture of an atherosclerotic plaque leading to thrombus formation and encompasses unstable angina (UA) and myocardial infarction (MI). The latter results in myocardial necrosis, which triggers an acute inflammatory response that contributes to disease progression. Few studies have explored the diagnostic utility of the neutrophil-to-lymphocyte ratio (NLR) as a complementary, inexpensive, and easily performed test for diagnosing MI, either alone or in conjunction with creatine kinase-MB (CK-MB). Therefore, this study aimed to evaluate the diagnostic accuracy of admission NLR as a biomarker for MI in ACS patients. This cross-sectional study included 89 patients with ACS who were admitted to the emergency department of Al-Basel Hospital between March 2023 and January 2024. The patients were categorized into two groups: MI (n = 41) and UA (n = 48). Baseline characteristics and specific inflammatory markers (WBC, neutrophils, lymphocytes, and NLR) were assessed and compared between the two groups. Our findings revealed that admission NLR values were significantly higher in the MI group compared to the UA group (4.62 vs. 2.56, $P < 0.01$). Moreover, NLR was significantly correlated with CK-MB activity in the MI group ($r = 0.45$, $P < 0.01$). A cutoff value of 2.78 for admission NLR yielded a sensitivity of 73% and a specificity of 62% for predicting MI in ACS patients. Notably, combining CK-MB and NLR measurements improved diagnostic performance, with a sensitivity of 88% and specificity of 93%. These findings suggest that a simple biomarker such as NLR could serve an adjunctive role in facilitating the diagnosis of MI in patients with ACS.

Keywords: neutrophil-to-lymphocyte ratio, CK-MB, acute coronary syndrome, myocardial infarction, inflammation.

1- INTRODUCTION

Cardiovascular diseases (CVDs) stand as a major global health concern, responsible for an estimated 17.9 million deaths worldwide [1, 2]. This high mortality rate is due to coronary artery disease (CAD), which is the first leading cause of global mortalities [3].

The main hallmark of CAD is atherosclerosis, a

chronic inflammatory condition that affects the endothelial cells of arteries and causes a lipid-rich plaque that could enlarge over time, leading to the stenosis of the coronary arteries [4, 5]. Some plaques remain stable, others could rupture, leading to the formation of coronary thrombosis [6]. Thrombosis could cause partial or total occlusion of the lumen resulting in the development of acute coronary syndrome (ACS). It may present as unstable angina (UA), in semi-blockage of the coronary arteries, or myocardial infarction (MI), generally corresponding to complete occlusion of the coronary arteries and necrosis of myocardial cells [7].

*Corresponding author:

Issa Yusuf: issa.yusuf@tishreen.edu.sy

Rama Ibrahim: ramaibrahim@tishreen.edu.sy

Received: 13/6/2024 Accepted: 21/9/2024.

DOI: <https://doi.org/10.35516/jjps.v18i2.2793>

Plaque rupture leads to the activation of systemic inflammatory response, as evidenced by elevated levels of inflammatory biomarkers such as high-sensitive C-reactive protein (hs-CRP) and tumor necrosis factor α (TNF- α) in the peripheral blood of ACS patients [8, 9]. This inflammatory response is aggravated by the ischemic cellular injury following MI [10]. Studies have shown that inflammatory biomarkers, including CRP, IL-1 β , ESR, and fibrinogen, are elevated in patients with MI compared to those in the healthy group [11, 12].

The neutrophil-to-lymphocyte ratio (NLR) is also used as an indicative biomarker of inflammatory status, it serves as an inexpensive, routinely available biomarker derived from the absolute neutrophil and lymphocyte counts [13, 14]. Various studies have emphasized the diagnostic and prognostic role of NLR in many diseases, including cancer, rheumatoid arthritis, Alzheimer's disease, and cardiovascular diseases [15-17]. However, there is a lack of research focusing on the utility of NLR as an adjunctive tool in the diagnosis of MI, particularly in combination with creatine kinase myocardial band isoform (CK-MB).

CK-MB is an enzyme that is expressed predominately in myocardial cells[18]. It persists as a biomarker for MI diagnosis especially when cardiac troponin assay is not available [19, 20]. This study aimed to investigate the individual or combined diagnostic accuracy of admission NLR as a biomarker of MI in patients presenting to the emergency department with anginal complaints consistent with MI. Those complaints are described as acute chest pain or discomfort, dyspnoea, or pain in the left or right arm or neck/jaw.

2- MATERIALS AND METHODS

2-1- Study population

We conducted a cross-sectional study involving a total of 89 patients diagnosed with ACS (48 with UA and 41 with MI) who were admitted to the emergency department of AL-Basel Hospital between March 2023 and January 2024.

The study was conducted according to the Declaration

of Helsinki and all procedures undertaken in this study were approved by the Institutional Board of Tishreen University, confirmed by Ethical Approval No.123 dated 20/09/2022.

Patients presenting with ACS were initially diagnosed based on the presence of persistent and characteristic chest pain experienced at rest. Thereafter, the diagnosis was confirmed based on electrocardiography (ECG) and CK-MB measurements. Patients who presented with chest pain and subsequently showed no changes in either ECG or CK-MB were defined as having UA. Diagnosis of MI was established when an elevation of CK-MB was noted or when characteristic ECG changes, e.g. ST segment elevation or T wave inversion was recorded along with the presence of stenosis by cardiac coronary catheterization.

Patients diagnosed with ACS who had either acute or chronic infections, autoimmune diseases like rheumatoid arthritis or asthma, malignant tumors, or were undergoing steroid therapy were excluded from the study. Additionally, patients with hyperlipidemia who were receiving statin therapy were also excluded.

2-2- Demographic data

Demographic information was obtained using a standardized questionnaire. Information collected encompassed age, sex, current smoking and patient's medical history including diabetes and/or hypertension. Systolic blood pressure (SBP) and diastolic blood pressure (DBP) were also measured.

2-3- Laboratory analysis

Initial blood samples were drained from patients upon admission to the emergency unit, and a complete blood count (CBC) was performed using EDTA tubes on the QUINTUS 5-Part Hematology analyzer. We calculated NLR and platelet-to-lymphocyte (PLR) ratios by dividing the absolute neutrophil count and platelets count, respectively, by the absolute lymphocyte count. Biochemical tests for urea, creatinine, CK, and CK-MB were performed on BIOSYSTEMS BTS 350 Biochemistry analyzer using serum tubes.

2-4- Statistical analysis

The Shapiro-Wilk test was used to check the normality distribution of continuous variables. Categorical variables were presented as counts and percentages, while continuous variables were reported as mean $\pm$ standard deviation or median and interquartile range (25-75 percentile). The independent sample t-test and Mann-Whitney tests were conducted for continuous variables (according to their normal distribution), while chi-square test was used for categorical variables. Correlations were evaluated using Spearman's correlation test. Variables of interest were further analyzed by multivariate regression, and results were shown as odds ratio (OR) with 95% confidence intervals (CIs). Receiver Operating Characteristic (ROC) curve analysis was performed to determine the optimal NLR cut-off value with the best sensitivity and specificity for predicting MI. Results were considered statistically significant when $P < 0.05$. The

statistical analysis was done using R statistical programming language.

3- RESULTS

The study included 89 patients with ACS, subdivided into 41 patients with MI (mean age: 55.9 ± 11.5 years; 73% male) and 48 patients with UA (mean age: 56.4 ± 11.2 years; 62.5% male). No significant differences were observed between the two groups in terms of gender distribution, smoking status, hypertension, or diabetes. Similarly, there were no statistically significant differences in mean age, diastolic blood pressure (DBP), systolic blood pressure (SBP), serum creatinine, urea, hemoglobin, absolute lymphocyte count, platelet count, or platelet-to-lymphocyte ratio. In contrast, the MI group exhibited significantly higher levels of WBC, absolute and relative neutrophil counts, relative lymphocyte count, NLR, CK, and CK-MB ($P < 0.05$), as presented in Table 1.

Table 1. Patients' baseline characteristics and laboratory findings of MI vs. UA groups.

	MI N=41	UA N=48	P-value
Males count	30 (73%)	30 (62.5%)	0.284
Smokers count	29 (70.7%)	28 (58.3%)	0.22
Hypertension	16 (39%)	23 (48%)	0.399
Diabetes	13 (31.7%)	15 (31.25%)	0.96
Hyperlipidemia	11 (26.83%)	17 (35.41%)	0.521
Age (years)	55.9 $\pm$ 11.5	56.4 $\pm$ 11.2	0.85
DBP (mmHg)	78.97 $\pm$ 21.49	74.75 $\pm$ 13.77	0.328
SBP (mmHg)	122.79 $\pm$ 28.95	125.25 $\pm$ 17.68	0.662
Urea (mg/dL)	37.01 $\pm$ 13.61	36.73 $\pm$ 12.11	0.92
Creatinine (mg/dL)	1.08 $\pm$ 0.41	1.02 $\pm$ 0.22	0.484
Hemoglobin (g/dL)	13.82 $\pm$ 1.72	13.11 $\pm$ 1.81	0.072
Platelets (*10³/μL)	249.95 $\pm$ 72.49	235.04 $\pm$ 56.63	0.311
WBC (*10⁹/L)⁺	11.95(9.8-15.78)	7.7(7-9.8)	<0.001*
Neutrophils (*10⁹/L)⁺	9.02(6.3-12.5)	5.2(4.2-7.1)	<0.001*
Neutrophils %	74.87 $\pm$ 10.96	66.96 $\pm$ 6.91	<0.001*
Lymphocytes (*10⁹/L)⁺	2.05(1.4-2.8)	2.14(1.8-2.6)	0.91
Lymphocytes%	19.29 $\pm$ 9.31	25.83 $\pm$ 5.57	<0.001*
NLR⁺	4.62(2.46-6.38)	2.56(1.9-3.4)	<0.001*
PLR⁺	116(91.25-148.9)	110.36 $\pm$ (90.6-146.7)	0.546
CK (U/L)⁺	315.5(130.75-520)	87.5(72-121.75)	<0.001*
CK-MB (U/L)⁺	32.5(28-49.75)	11(8.8-16.3)	<0.001*

⁺ Variable are not normally distributed, and expressed as median (interquartile range).
^{*} The difference is considered statistically significant.

Admission NLR showed a significant positive correlation with serum CK-MB activity, whereas

lymphocyte counts demonstrated a negative correlation with CK-MB in MI patients (Table 2).

Table 2. Spearman's correlation coefficients between CK-MB and inflammatory parameters in MI patients.

	CRP	WBC	Neutrophils	Lymphocytes*	NLR*
CK-MB					
<i>rho</i>	0.243	0.214	0.274	-0.412*	0.456*
P-value	0.302	0.204	0.101	<0.05*	<0.01*
* Variables are significantly correlated with CK-MB.					

Using multivariate logistic regression analysis, higher admission NLR values independently predicted MI among ACS patients (OR: 2.36, 95% CI: 1.4-4.5, $P < .01$; Table 3). In ROC analysis, a cut-off level of NLR > 2.78 had a sensitivity of 73% and a specificity of 62% for predicting MI [Area under the ROC curve (AUC) = 0.737, 95% CI: 0.620-0.852, $P < .001$; Figure 1]. The sensitivity and specificity of CK-MB were calculated to compare results

with those of NLR, and the threshold value of 25 IU/L was selected based on the assay kit used for analysis (Biosystems immunoinhibition CK-MB kit "COD 11792"). As shown in Table 4, adding admission NLR measurement to CK-MB analysis demonstrated better sensitivity and specificity (0.88 and 0.93, respectively) for predicting MI than CK-MB alone (0.78 and 0.87, respectively).

Table 3. Multivariate logistic regression analysis for predictors of MI in ACS patients.

Predictors	Odds Ratio (95%CI)	P-value
NLR	2.35 (1.4-4.5)	<0.01*
CRP	0.95 (0.86-1.02)	0.21
Age	1 (0.9-1.08)	0.78
Male gender	2.68 (0.45-19.49)	0.29
Smoking	3.72 (0.58-36.76)	0.19
Hyperlipidemia	0.63(0.1-3.37)	0.59

Table 4. Sensitivity, specificity, Positive predictive value (PPV), negative predictive value (NPV) of CK-MB, NLR, and CK-MB+NLR for predicting MI in ACS patients.

	CK-MB	NLR	NLR+CK-MB
Sensitivity	0.78	0.73	0.88
Specificity	0.87	0.622	0.93
PPV	0.84	0.57	0.92
NPV	0.82	0.70	0.9

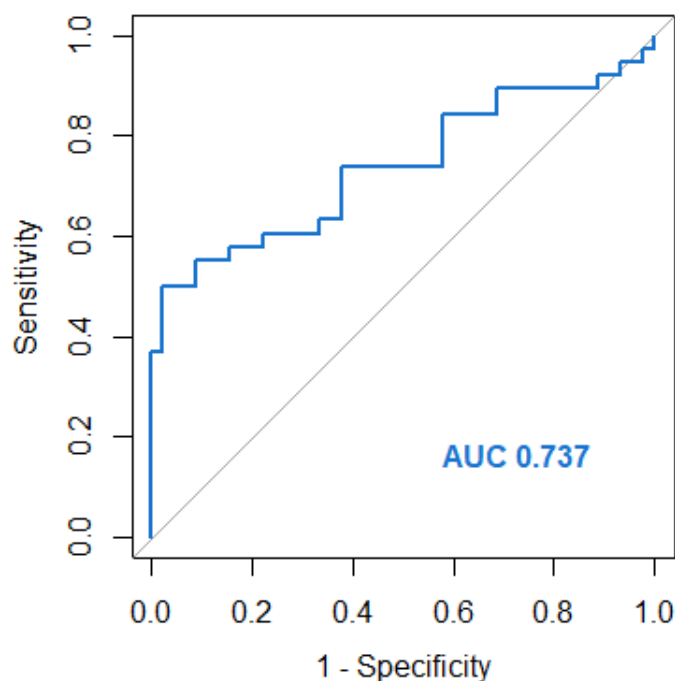


Figure1: The receiver–operating characteristic curve analysis of NLR for predicting MI.

4- DISCUSSION

Our study aimed to investigate the significance of NLR as an adjunctive biomarker in the diagnosis of MI in patients with ACS, in relation to the diagnostic performance of CK-MB within the first six hours of emergency admission.

This study showed that white blood cell and neutrophil counts were significantly higher in the MI group than in the UA group ($P < 0.01$). These results are consistent with the research of Marechal et al., who reported a significant difference in white blood cell count between patients with MI and those with stable and unstable angina. This difference was primarily attributed to variations in neutrophil count [21]. Likewise, Al-Fartosi et al., found that neutrophils were significantly higher in MI and UA groups compared to healthy controls, and also elevated in the MI group compared to the UA group [22]. Neutrophils

are thought to participate in the pro-inflammatory response that follows the MI [23]. The cellular necrosis of cardiac muscle cells observed during MI results in the release of a variety of pro- and anti-inflammatory molecules. Of these, DAMPs (Damage-Associated Molecular Patterns) are nominated to play a crucial role in the inflammatory process. DAMPs bind to their receptors on the surface of innate immune cells, PRRs (Pattern Recognition Receptors), resulting in the induction of pro-inflammatory cytokines such as $\text{TNF-}\alpha$ and IL-1, which stimulate chemotaxis and recruitment of white blood cells to the site of necrosis, with neutrophils playing a primary role in the inflammatory response [24, 25]. Cellular necrosis is absent in UA, which explains the decreased neutrophil count compared to the MI. No significant difference was found in the number of lymphocytes between the MI and UA groups ($P > 0.05$), although the value was higher in the MI

group. This result was also reported by Tahto et al., and Marechal et al., who did not find any significant difference in absolute lymphocyte counts between the MI and UA groups [21, 26]. However, in our study, the difference in the relative lymphocyte count between the two groups was also investigated, because the higher count of WBC in the MI group could mislead the interpretation of the true difference in the lymphocyte count. Indeed, the mean relative lymphocyte count in the MI group was significantly lower than in the UA group ($P < 0.01$). Additionally, the relative lymphocyte count was below the reference limit in the MI group (19.3%) and within normal limits in the UA group (25.8%).

The role of lymphocytes in the pathogenesis of atherosclerosis remains ambiguous.

Mustafic et al., showed significantly lower relative lymphocyte values in patients with ACS compared to stable angina but without significant difference in absolute lymphocyte count [27]. Another study comparing absolute and relative lymphocyte counts between patients with stable and unstable angina showed no differences in either parameter [28]. These results together with ours imply that MI is the main contributor to the downregulation of lymphocytes in patients with ACS. To determine the mechanism underlying the reduction in lymphocyte count in MI patients, *in-vivo* experiments were conducted on MI-induced mice. The study found that MI mice exhibited glandular hypertrophy on the first day along with increased glucocorticoid production due to activation of the hypothalamic-pituitary-adrenal axis [29]. Plasma levels of glucocorticoids reduce lymphocyte count in peripheral blood by inducing reverse migration of lymphocytes to the bone marrow, promoting lymphocyte apoptosis and inhibiting their proliferation [29, 30]. We excluded patients undergoing steroid therapy because it affects lymphocyte counts. Admission NLR has been thoroughly studied as an inflammatory biomarker in a wide variety of medical conditions, including cardiovascular diseases [31], owing to its low cost, ease of measurement, and laboratory availability.

Inflammation has a key role in ACS context, since atherosclerosis, which is the main cause of coronary heart disease, develops as a result of the chronic inflammatory process in damaged endothelial cells lining the arteries. Furthermore, plaque rupture and atherothrombosis trigger an acute local and systemic inflammation, that is exacerbated after myocardial cellular necrosis [32]. As atherothrombosis is an acute inflammatory response involving both innate and acquired immunity [33], a biomarker such as NLR which combines neutrophils and lymphocytes (representing the innate and acquired immunological response, respectively) could be considered as a biomarker with prognostic and diagnostic value in ACS and MI.

We found that NLR at admission was significantly increased in the MI group compared to the UA group ($P < 0.01$) and thus emerged as the biomarker with the strongest positive correlation with CK-MB values in patients with MI among other inflammatory indicators, i.e. WBC, lymphocytes, and neutrophils. In the study of Erturk et al., NLR was independently elevated in ACS patients compared to controls. Furthermore, the highest levels of NLR were observed in patients with STEMI followed by NSTEMI and UA. They also found that NLR was positively correlated with high-sensitivity troponin (hsTn) in ACS patients [34]. The results of our study support the above-mentioned conclusions and suggest that NLR could be used as an indicator to discriminate different types of ACS. Shumilah et al., investigated the diagnostic accuracy of NLR, mean platelet volume (MPV), PLR, and monocyte-to-lymphocyte ratio (MLR) for ACS, they found that NLR is the strongest predictive biomarker for ACS, while MLR was not significant [35]. This study highlights the diagnostic efficiency of NLR among other inflammatory biomarkers derived from complete blood count (CBC), thus supporting our choice of NLR as an inflammatory biomarker correlated with CK-MB during ACS. The mechanism that links MI to NLR involves the two phases of inflammation associated with MI: the inflammatory phase and the proliferative phase [36].

During the inflammatory phase, neutrophils play a crucial role by recruiting macrophages and removing cellular debris. Consequently, the neutrophil count in peripheral blood positively correlates with infarct size [37]. Lymphocytes are vital for myocardial remodeling after MI due to their anti-inflammatory properties. However, unlike neutrophils, lymphocyte levels decrease during the inflammatory phase, which contributes to the observed increase in NLR levels in the early stages of MI [38].

An NLR cutoff value of 2.78 showed a sensitivity of 0.73 and specificity of 0.62 for predicting ACS in patients with MI symptoms. Despite the low sensitivity and specificity of NLR compared to CK-MB, our study showed that integrating both biomarkers resulted in greater accuracy than using each biomarker individually. This suggests that NLR could help improve the accuracy of MI diagnosis when used in combination with CK-MB, a cardiac biomarker widely used in clinical practices. Similar conclusion was proposed by Korkmaz et al., who demonstrated that NLR was higher in the troponin-positive group and that a cut-off value of 2.80 had high sensitivity and specificity for predicting troponin elevation in ACS patients [39]. These findings indicate that NLR could serve as a useful biomarker in clinical practice. We recommend adding NLR to the routine hematorgram and considering it as an adjunctive diagnostic tool, as well as a biomarker for assessing the severity of inflammatory response.

5- CONCLUSION

Admission NLR is an adjunctive complementary tool in the diagnosis of MI in patients with ACS. It could also be a simple but potential biomarker of the inflammatory response that follows myocardial necrosis.

Admission NLR demonstrated a significant increase in MI compared to UA and a strong correlation with CK-MB levels, suggesting its relevance in discriminating ACS subtypes. While NLR alone has lower sensitivity and specificity than CK-MB, its use in combination with CK-MB could improve diagnostic accuracy in clinical practice.

These findings highlight the need for further research into the role of inflammatory biomarkers in the diagnosis and management of ACS to optimize patient care.

6- LIMITATIONS

We were unable to study the prognostic value of NLR because there was a lack of long-term follow-up of patients. A cohort study design would be preferable for this purpose. The sample size is relatively small, which may have affected the significance of some results. Troponin was performed for MI screening initially but was later replaced by CK-MB due to its intermittent availability during the study period. The relationship between NLR and other inflammatory biomarkers, such as hs-CRP and TNF- α , could provide more details about the inflammatory response following MI.

7- DECLARATION

7-1- Competing interests

The author declares that there is no interest in the publication of the manuscript. In addition, the ethical issues, including plagiarism, informed consent, misconduct, data fabrication and/or falsification, double publication and/or submission, and redundancies have been completely observed by the author.

7-2- Author contributions

All work relevant to this study, from data collection, analysis, and writing of the article, was carried out by the authors I.Y and R.I.

7-3- Data sharing statement

The authors confirm that the data supporting the findings of this article are available within this article.

7-4- Acknowledgements

We deeply appreciate the commitment and efforts of the medical staff and healthcare professionals at "Al-Basel Hospital for Cardiology & Heart Surgery".

7-5- Funding

This work funded by Tishreen University in Syria.

7-6- Informed Consent Statement

Written consent was acquired from patients to ensure their informed participation.

REFERENCES

1. Alrob O.A. Cardiac energy metabolism: a potential novel therapeutic target in the treatment of ischemic heart diseases. *Jordan Journal of Pharmaceutical Sciences*. 2017; 10(3).
2. World Health Organization. Health topics: Cardiovascular diseases. Fact sheet. Available online: http://www.who.int/cardiovascular_diseases/en/ (accessed on 11 December 2020). 2013.
3. Ralapanawa U., Sivakanesan R. Epidemiology and the magnitude of coronary artery disease and acute coronary syndrome: a narrative review. *Journal of Epidemiology and Global Health*. 2021; 11(2):169.
4. Al-Motassem Y., Bulatova N.L., AbuRuz S. Management of coronary artery disease in Jordan: cross-sectional comparative study. *Jordan Journal of Pharmaceutical Sciences*. 2008; 1(1).
5. Milutinović A., Šuput D., Zorc-Plesković R. Pathogenesis of atherosclerosis in the tunica intima, media, and adventitia of coronary arteries: an updated review. *Bosnian Journal of Basic Medical Sciences*. 2020; 20(1):21.
6. Bhatt D.L., Lopes R.D., Harrington R.A. Diagnosis and treatment of acute coronary syndromes: a review. *JAMA*. 2022; 327(7):662–675.
7. Komilovich E.B.Z. Coronary artery disease. *European Journal of Modern Medicine and Practice*. 2023; 3(12):81–87.
8. Sayfiddinovna S.A., et al. Comparative assessment of nonspecific inflammation markers and various key factors in patients with acute coronary syndrome. *European Science Review*. 2019; 2(1–2):210–213.
9. Tsioufis P., et al. The impact of cytokines in coronary atherosclerotic plaque: current therapeutic approaches. *International Journal of Molecular Sciences*. 2022; 23(24):15937.
10. Matter M.A., et al. Inflammation in acute myocardial infarction: the good, the bad and the ugly. *European Heart Journal*. 2024; 45(2):89–103.
11. Oprescu N., et al. Inflammatory markers in acute myocardial infarction and the correlation with the severity of coronary heart disease. *Annals of Medicine*. 2021; 53(1):1042–1048.
12. Wojtkowska A., et al. The inflammation link between periodontal disease and coronary atherosclerosis in patients with acute coronary syndromes: case–control study. *BMC Oral Health*. 2021; 21:1–17.
13. Guthrie G.J., et al. The systemic inflammation-based neutrophil–lymphocyte ratio: experience in patients with cancer. *Critical Reviews in Oncology/Hematology*. 2013; 88(1):218–230.
14. Han J.-B., et al. Predictive value of inflammation biomarkers in patients with portal vein thrombosis. *Journal of Clinical and Translational Hepatology*. 2021; 9(3):384.
15. Buonacera A., et al. Neutrophil to lymphocyte ratio: an emerging marker of the relationships between the immune system and diseases. *International Journal of Molecular Sciences*. 2022; 23(7):3636.
16. Li M., Xie L. Correlation between NLR, PLR, and LMR and disease activity, efficacy assessment in rheumatoid arthritis. *Evidence-Based Complementary and Alternative Medicine*. 2021; 2021:1–6.
17. Mosca M., et al. Neutrophil-to-lymphocyte ratio (NLR) in NSCLC, gastrointestinal, and other solid tumors: immunotherapy and beyond. *Biomolecules*. 2023; 13(12):1803.
18. Tilea I., Varga A., Serban R.C. Past, present, and future of blood biomarkers for the diagnosis of acute myocardial infarction—promises and challenges. *Diagnostics*. 2021; 11(5):881.
19. Byrne R.A., et al. 2023 ESC guidelines for the management of acute coronary syndromes: developed by the task force on the management of acute coronary syndromes of the European Society of Cardiology (ESC). *European Heart Journal: Acute Cardiovascular Care*. 2024; 13(1):55–161.

20. Al-Shatnawi S.F., Khasawneh R.A. Emergency research status in the Middle Eastern region. *Jordan Journal of Pharmaceutical Sciences*. 2022; 15(2):214–226.
21. Maréchal P., et al. Neutrophil phenotypes in coronary artery disease. *Journal of Clinical Medicine*. 2020; 9(5):1602.
22. Al-Fartosi K., Al-Salih R., Batah S.J. Study of relationship between blood parameters and oxidant-antioxidant status of patients with unstable angina pectoris and myocardial infarctions. *Thi-Qar Medical Journal*. 2010; 4:47–64.
23. Puhl S.-L., Steffens S. Neutrophils in post-myocardial infarction inflammation: damage vs. resolution? *Frontiers in Cardiovascular Medicine*. 2019; 6:25.
24. Jiang K., Hwa J., Xiang Y. Novel strategies for targeting neutrophil against myocardial infarction. *Pharmacological Research*. 2024; 107256.
25. Ma Y., Yabluchanskiy A., Lindsey M.L. Neutrophil roles in left ventricular remodeling following myocardial infarction. *Fibrogenesis & Tissue Repair*. 2013; 6:1–10.
26. Tahto E., et al. Neutrophil-to-lymphocyte ratio and its relation with markers of inflammation and myocardial necrosis in patients with acute coronary syndrome. *Medical Archives*. 2017; 71(5):312.
27. Mustafic S., Ibralic A.M., Loncar D. Association of inflammatory and hemostatic parameters with values of high sensitive troponin in patients with acute coronary syndrome. *Medical Archives*. 2022; 76(2):84.
28. Zouridakis E.G., Garcia-Moll X., Kaski J.C. Usefulness of the blood lymphocyte count in predicting recurrent instability and death in patients with unstable angina pectoris. *American Journal of Cardiology*. 2000; 86(4):449–451.
29. Ma Y., et al. Circulating lymphocyte trafficking to the bone marrow contributes to lymphopenia in myocardial infarction. *American Journal of Physiology-Heart and Circulatory Physiology*. 2022; 322(4):H622–H635.
30. Jia W.-Y., Zhang J.-J. Effects of glucocorticoids on leukocytes: Genomic and non-genomic mechanisms. *World Journal of Clinical Cases*. 2022; 10(21):7187–7194.
31. Afari M.E., Bhat T. Neutrophil to lymphocyte ratio (NLR) and cardiovascular diseases: an update. *Expert Review of Cardiovascular Therapy*. 2016; 14(5):573–577.
32. Liuzzo G., et al. Inflammation and atherothrombosis. In: *Clinical Immunology*. Elsevier; 2019. p. 935–946.e1.
33. Cimmino G., et al. Evolving concepts in the pathophysiology of atherosclerosis: From endothelial dysfunction to thrombus formation through multiple shades of inflammation. *Journal of Cardiovascular Medicine*. 2023; 24(Suppl 2):e156–e167.
34. Özalp B., et al. Correlations between hematological indicators and other known markers in acute coronary syndromes. *EJCM*. 2017; 5(4):67–74.
35. Shumilah A.M., Othman A.M., Al-Madhagi A.K. Accuracy of neutrophil to lymphocyte and monocyte to lymphocyte ratios as new inflammatory markers in acute coronary syndrome. *BMC Cardiovascular Disorders*. 2021; 21:1–6.
36. Tudurachi B.-S., et al. Assessment of inflammatory hematological ratios (NLR, PLR, MLR, LMR and Monocyte/HDL–Cholesterol Ratio) in acute myocardial infarction and particularities in young patients. *International Journal of Molecular Sciences*. 2023; 24(18):14378.
37. Ma Y. Role of neutrophils in cardiac injury and repair following myocardial infarction. *Cells*. 2021; 10(7):1676.
38. Kologrivova I., et al. Cells of the immune system in cardiac remodeling: main players in resolution of inflammation and repair after myocardial infarction. *Frontiers in Immunology*. 2021; 12:664457.
39. Korkmaz A., et al. Utility of neutrophil–lymphocyte ratio in predicting troponin elevation in the emergency department setting. *Clinical and Applied Thrombosis/Hemostasis*. 2015; 21(7):667–671.

القيمة التنبؤية لنسبة العدلات إلى اللمفاويات في التنبؤ بارتفاع مستويات CK-MB في احتشاء العضلة القلبية: دراسة لدى مرضى ACS في سوريا

عيسى يوسف¹، راما إبراهيم¹

¹قسم الكيمياء الحيوية والأحياء الدقيقة، كلية الصيدلة، جامعة تشرين، سوريا

ملخص

المتلازمة الإكليلية الحادة هي داء قلبي وعائي شديد مترافق مع معدل وفيات مرتفع. ينتج الداء عادةً من تمزق لويحة التصلب العصيدي مما يؤدي إلى تشكّل الخثرة العصيدية. تتضمن المتلازمة الإكليلية الحادة كل من الخناق غير المستقر واحتشاء العضلة القلبية. يسبّب احتشاء العضلة القلبية تنحراً للعضلة القلبية بالتالي تحريض استجابة التهابية حادة تساهم في تقدّم المرض. قلّة فقط من الدراسات قد تحرّرت القدرة التشخيصية لنسبة العدلات إلى اللمفاويات باعتباره مؤشر مساعد رخيص الثمن وسهل الإجراء لتشخيص احتشاء العضلة القلبية، وذلك لوحده أو بالمشاركة مع الكرياتين كيناز MB. بالتالي تهدف الدراسة إلى تحديد القدرة التشخيصية لنسبة العدلات إلى اللمفاويات عند القبول كمؤشر لاحتشاء العضلة القلبية عند مرضى المتلازمة الإكليلية الحادة. شارك 89 مريضاً بالمتلازمة الإكليلية الحادة في هذه الدراسة المقطعية والذين تم قبولهم في وحدة الإسعاف ضمن مشفى الباسل بين شهري آذار 2023 وكانون الثاني 2024. تم تقسيم المرضى إلى مجموعة احتشاء العضلة القلبية (عدد 41) ومجموعة الخناق غير المستقر (عدد 48). تم مقارنة وتقييم الخصائص الأساسية إضافةً لعدد من المشعرات الخلوية الالتهابية (تعداد الكريات البيضاء والعدلات واللمفاويات ونسبة العدلات إلى اللمفاويات) بين مجموعتي الدراسة. أظهرت نتائجنا أن قيم نسبة العدلات إلى اللمفاويات عند القبول كانت أعلى عند مجموعة الاحتشاء منها عند مجموعة الخناق غير المستقر (4.62 مقابل 2.56، $P < 0.01$)، كما كانت القيم مرتبطة مع فعالية الكرياتين كيناز في مجموعة الاحتشاء (معامل الارتباط = 0.45، $P < 0.01$). وجد أن القيمة الحدية 2.78 لنسبة العدلات إلى اللمفاويات عند القبول كان لها حساسية 73% ونوعية 62% في التنبؤ باحتشاء العضلة القلبية عند مرضى المتلازمة الإكليلية الحادة. كما كان من المثير للاهتمام أن مشاركة نسبة العدلات إلى اللمفاويات مع الكرياتين كيناز MB أدت إلى زيادة الحساسية والنوعية إلى (88% و 93% على التوالي) أي أفضل من استخدام المشعرين بشكل منفصل. يدل ذلك إلى أن مشعراً تشخيصياً بسيطاً كنسبة العدلات إلى اللمفاويات يمكن أن يكون له دور كعامل مساعد في تشخيص احتشاء العضلة القلبية عند المرضى المصابين بالمتلازمة الإكليلية الحادة.

الكلمات الدالة: نسبة العدلات إلى اللمفاويات، الكرياتين كيناز MB، المتلازمة الإكليلية الحادة، احتشاء العضلة القلبية، الالتهاب.

* المؤلف المراسل:

عيسى يوسف: issa.yusuf@tishreen.edu.sy

راما إبراهيم: ramaibrahim@tishreen.edu.sy

تاريخ استلام البحث 2024/6/13 وتاريخ قبوله للنشر 2024/9/21.