Original Article

Platelet-to-Lymphocyte Ratio as an Independent Predictor of In-Hospital Mortality in ST-Elevation Myocardial Infarction: A Prospective Study

Naman Jain[1], Jigar M. Haria[2], V. K. Singh[3]

[1]Post Graduate Student, Department of Medicine, Teerthanker Mahaveer Medical College and Research Centre, Moradabad, India. [2]Professor, Department of Medicine, Teerthanker Mahaveer Medical College and Research Centre, Moradabad, India. [3]Professor & Head, Department of Medicine, Teerthanker Mahaveer Medical College and Research Centre, Moradabad, India.

Abstract

Background: The platelet-to-lymphocyte ratio (PLR) is a relatively new hematological marker of inflammation and thrombus activity. The question regarding PLR and its use in prospectively predicting in-house mortality in STEMI remains unanswered. The present study evaluated the predictive capacity of admission PLR for in-house mortality for patients with STEMI and compared its predictive capacity with the GRACE score. **Material and Methods:** This is a prospective, observational study of sixty consecutive patients with STEMI who presented to a tertiary care center between January 2023 and December 2024. At admission, the PLR status was tested on three occasions: admission, Day 1, and Day 3 of hospitalization. The GRACE score was calculated at the time of admission. The primary outcome of interest was in-hospital cardiovascular mortality. The predictive capacity was calculated using receiver operating characteristic (ROC) curve analysis and logistic regression. **Results:** The admission PLR of patients who died was significantly higher ($p < 0.001$) than the admission PLR of patients who survived. Admission PLR for patients who died was statistically higher than that of patients who survived (25.5 ± 3.2 vs 12.8 ± 3.6), $p<0.001$. In addition, mortality was only seen in the highest PLR tertile (≥23; 60% mortality) and high GRACE risk (>140; 52% mortality) group only, with no mortality seen in lower tertiles or lower GRACE groups. The admission PLR was a significant independent predictor of mortality with an area under the curve (AUC) of 0.990 (95% CI: 0.961—1.000), slightly less than the GRACE AUC (0.992; p=0.31). In the multivariable analysis, the independent predictors of mortality were both PLR (adjusted OR 2.17; p=0.001) and GRACE score (adjusted OR 1.24 increase per 10; p=0.003). **Conclusion:** Overall, admission PLR appears accessible, low-cost, and a strongly predictor of in-hospital mortality in STEMI patients. It is potentially as useful as the GRACE score risk stratification tool. The simplicity of use, calculation, and accessibility is especially advantageous for rapid risk stratification in hospital STEMI patients, particularly when capacity is scarce.

Keywords: STEMI, platelet-to-lymphocyte ratio, GRACE score, in-hospital mortality, prognostic marker.

Received: 01 September2025 **Revised: 20 September 2025** **Accepted: 07 October 2025** **Published: 05 February 2026**

Introduction

Known to be a leading threat to world health, cardiovascular disease (CVD) currently accounts for nearly 17.9 million deaths annually, roughly 32% of all global deaths.[1] Acute Myocardial Infarction (AMI) is a leading cause of serious illness requiring extensive resources, even with diagnosis protocols and treatment.[2] This situation is particularly serious in countries with low- and middle-income economies, such as India, where the increasing incidence of AMI occurs in younger populations because of "lifestyle [changes] and epidemiological transition" (Harrison et al.), and the related disease burden and socio-economic burden associated with AMI is considerable.

AMI occurs primarily as the result of atherogenesis, newly considered to be a chronic inflammatory disease, involving the progressive development of plaque in coronary artery locations.[3,4] Plaque rupture or erosion can lead to the formation of a thrombus, and this results in the sudden occlusion of the coronary arteries.[5] Inflammation is a prominent part of this process, from endothelial dysfunction to destabilization and rupture of plaque.[6,7] Inflammation is also associated with the ectopic remodeling of the left ventricle after AMI and a subsequent risk for heart failure, an important yet often overlooked issue.

Conventional inflammatory biomarkers, including C-reactive protein (CRP) and interleukin-6 (IL-6), have been linked to poor outcomes after AMI; however, their generalizability is limited due to cost, accessibility, and variability in testing.[5] This has increased interest in simpler prognostic indices obtained from routine analysis of complete blood count (CBC). The platelet-to-lymphocyte ratio (PLR) received attention as a potential inflammatory-thrombotic marker.[8]

The PLR considers the relationship between two cell types: platelets, which promote thrombus formation and vascular

Address for correspondence: Dr. Naman Jain, Post Graduate Student, Department of Medicine, Teerthanker Mahaveer Medical College and Research Centre, Moradabad, India
E-mail: namanjain1080@gmail.com

DOI:
10.21276/amit.2026.v13.i1.333

How to cite this article: Jain N, Haria JM, Singh VK. Platelet-to-Lymphocyte Ratio as an Independent Predictor of In-Hospital Mortality in ST-Elevation Myocardial Infarction: A Prospective Study. Acta Med Int. 2026;13(1):251-255.

inflammation, and lymphocytes, whose decrease indicates reduced immune regulation.[8] An increased PLR means a pro-inflammatory, pro-thrombotic multi-cellular state known to be associated with inferior cardiovascular outcomes. In support of its prognostic value, clinical studies have identified increased admission PLR predicting an increased risk of in-hospital mortality and major adverse cardiovascular events (MACE) in AMI patients receiving primary percutaneous coronary intervention (PCI).[9] A systematic review of literature found that an increase in PLR was also related to adverse outcomes in acute coronary syndrome (ACS).[10]

Mechanistic studies indicate that an elevated PLR is associated with endothelial dysfunction, impaired coronary perfusion, adverse ventricular remodeling, and increased heart failure risk, following AMI.[11] Yang et al. also reported that increased PLR values could independently predict in-hospital mortality among AMI patients.[12] While other hematologic indices have been explored, such as the systemic immune-inflammation index (SII) and systemic inflammation response index (SIRI), PLR retains appeal through simplicity and availability.[11]

While a growing body of literature exists, PLR has not yet been deeply integrated into routine clinical practice. There have been challenges to implementation, such as cut-off values, population variability, and a lack of standardized validation in different ethnic groups and geographic groups. Most prior studies were retrospective, highlighting the importance of prospective evaluation.[12]

Considering its low cost, accessibility, and rapid risk stratification, this study will evaluate the prognostic relevance of admission PLR as a predictor of in-hospital mortality in patients with STEMI. If a predictive role for PLR score can be established, the future of medical work in patient triage could improve, clinical decision-making could be guided, and outcomes might improve in these patients at high risk for morbidity and mortality.[13]

Materials and Methods

Sample Size: The sample size was estimated based on an assumed in-hospital mortality rate of 10-15% for ST-Elevation Myocardial Infarction patients, a 95% confidence interval, and a 10% margin of error. The minimum sample size needed for the study was 55 patients. To account for attrition, we enrolled 60 patients.

Data Collection: All patients had a complete clinical assessment, ECG, echocardiogram, and routine laboratory investigations performed at admission. The Platelet-Lymphocyte Ratio (PLR) was calculated from the complete blood count (CBC) as absolute platelet count divided by absolute lymphocyte count. PLR readings were recorded for:

- Day 0 (admission)
- Day 1
- Day 3 of hospitalization

The Global Registry of Acute Coronary Events (GRACE) score was determined at admission for each patient using discrete variables: age, heart rate, systolic blood pressure, serum creatinine, Killip class, ST segment changes, presentation in arrest, and elevation in cardiac enzymes.

Outcome measures: The principal outcome measure was in-hospital death from cardiovascular causes. Patients were monitored for clinical adverse events during the clinical admission.

Statistical Analysis: Continuous outcomes will be reported as mean ± standard deviation (SD), and the two means will be compared with a Student's t-test or Mann-Whitney U test as appropriate. Categorical outcomes will be presented as counts and proportions, and the two groups in the categorical variables were compared using chi-squared tests or Fisher's exact test. Receiver operating characteristic (ROC) curves will be used to assess the discriminatory performance of PLR and GRACE score to predict in-hospital mortality; areas under the curve (AUC) and 95% confidence intervals (CI) will also be reported. The Youden index will be used to derive optimal cut-off values. Univariate and multivariate logistic regression analyses were conducted to determine independent predictors of mortality. A p-value < 0.05 was considered statistically significant.

All statistical analyses were completed using SPSS version 26.0 (IBM Corp., Armonk, NY, USA).

Results

Patient Enrolment and Baseline Characteristics

Sixty-five eligible patients were screened for STEMI, although five were eliminated for not having laboratory data, leaving a sample of 60. The cohort was primarily male (71%) and had an average age of 57 ± 10 years. Baseline characteristics are illustrated in Table 1. Most patients were intermediate to high risk based on their GRACE scores on admission (mean: 141 ± 41), and more than half of the cohort appeared to have left ventricular dysfunction.

Table 1: Baseline characteristics of the study population

Variable	Mean ± SD / n (%)
Age (years)	57.35 ± 9.72
Male sex	44 (71.0%)
Female sex	16 (26.7%)
Heart rate (bpm)	78.13 ± 13.04
Systolic BP (mmHg)	109.37 ± 21.00
Diastolic BP (mmHg)	67.00 ± 11.86
Serum creatinine (mg/dL)	2.06 ± 1.02
Blood urea nitrogen (mg/dL)	80.67 ± 45.44
Total cholesterol (mg/dL)	208.42 ± 67.01
LDL cholesterol (mg/dL)	118.33 ± 39.20
HDL cholesterol (mg/dL)	51.42 ± 13.80
Triglycerides (mg/dL)	187.05 ± 54.20

LV dysfunction – Fair (>50%)	31 (51.7%)
LV dysfunction – Mild (45–50%)	15 (25.0%)
LV dysfunction – Moderate (30–44%)	11 (18.3%)
LV dysfunction – Severe (<30%)	3 (5.0%)
GRACE score	140.97 ± 41.33
PLR Day 0	15.35 ± 6.22

Serial PLR Trends and Outcomes: Admission PLR (Day 0) was significantly higher in non-survivors (25.5 ± 3.2) compared to survivors (12.8 ± 3.6, $p < 0.001$). This difference persisted on Day 1 and Day 3 without significant intra-group change. Figure 1 shows the divergence in PLR between survivors and non-survivors during hospitalization.

Table 2: PLR trend analysis by outcome

PLR Day	Non-survivors (mean ± SD)	Survivors (mean ± SD)	p-value
Day 0	25.50 ± 3.19	12.81 ± 3.64	<0.001
Day 1	25.80 ± 3.25	13.07 ± 3.63	<0.001
Day 3	26.05 ± 3.21	13.28 ± 3.63	<0.001

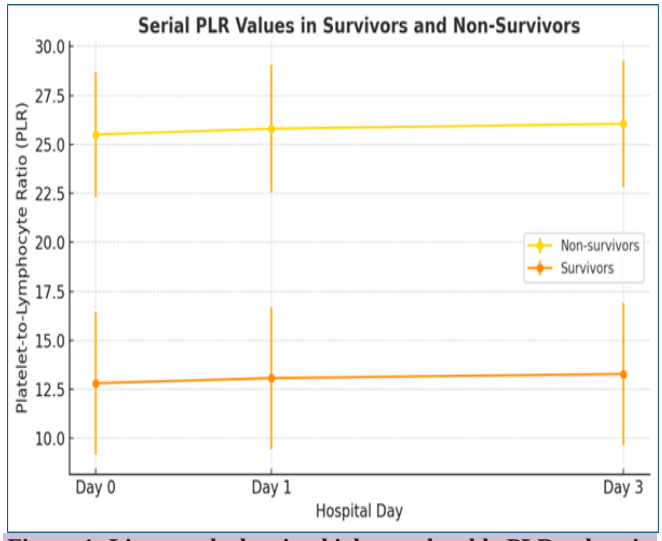

Figure 1: Line graph showing higher and stable PLR values in non-survivors across all three days.

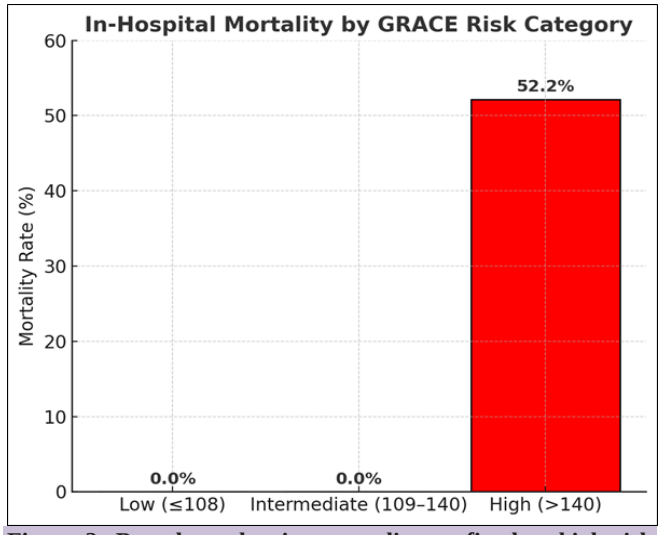

Figure 2: Bar chart showing mortality confined to high-risk GRACE group.

GRACE Score and Mortality: All in-hospital deaths occurred in the high-risk GRACE group (>140). Mortality in this group was 52%, whereas no deaths occurred in intermediate or low-risk groups ($p < 0.001$ for trend).

PLR Tertiles and Mortality: When stratified into tertiles, mortality clustered entirely within the highest PLR tertile (≥23), where 60% of patients died. No deaths occurred in the lower two tertiles.

Table 3: Mortality by GRACE risk category

GRACE category	n	Deaths	Mortality rate (%)
High (>140)	23	12	52.17
Intermediate (109–140)	29	0	0.0
Low (≤108)	8	0	0.0

Table 4: Mortality by PLR tertile

PLR tertile	n	Deaths	Mortality rate (%)
Low (≤14)	20	0	0.0
Intermediate (15–22)	20	0	0.0
High (≥23)	20	12	60.0

Table 5: ROC-derived cut-off values and diagnostic performance

Predictor	AUC (95% CI)	Optimal cut-off	Sensitivity (%)	Specificity (%)
PLR Day 0	0.990 (0.960–1.000)	22.25	100	98
GRACE score	0.992 (0.972–1.000)	158.0	100	92

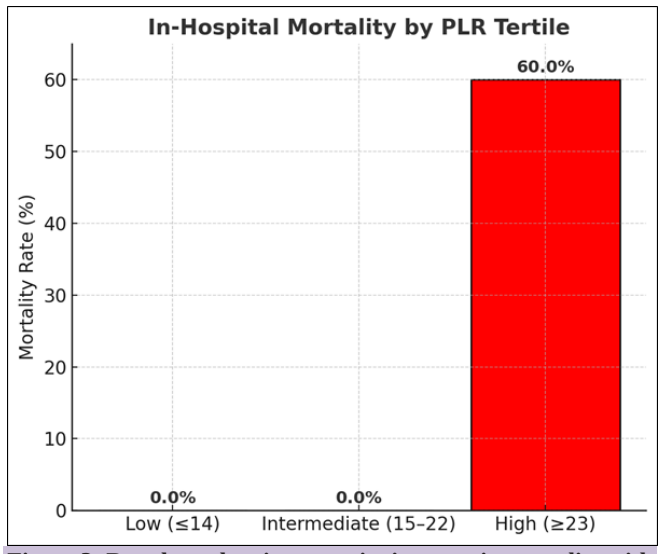

Figure 3: Bar chart showing stepwise increase in mortality with PLR tertiles.

ROC Curve Analysis: Admission PLR demonstrated excellent discrimination for in-hospital mortality (AUC = 0.990, 95% CI: 0.961–1.000), comparable to the GRACE score (AUC = 0.992, 95% CI: 0.972–1.000; p = 0.31 for difference).

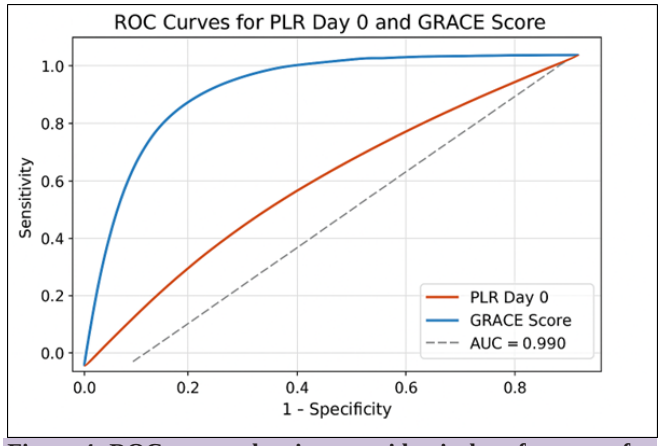

Figure 4: ROC curves showing near-identical performance for PLR and GRACE

Multivariate Analysis: On multivariate logistic regression, both GRACE score and PLR at admission were independent predictors of in-hospital mortality:

- GRACE score (per 10-point increase): Adjusted OR = 1.24 (95% CI: 1.16–1.53, p = 0.003)
- PLR Day 0 (per unit increase): Adjusted OR = 2.17 (95% CI: 1.18–2.44, p = 0.001)

Discussion

In this prognostic study, we demonstrated that the admission platelet-to-lymphocyte ratio (PLR) was a strong and independent predictor of in-hospital mortality in patients with STEMI. A PLR of 22.25 had a 100% sensitivity and a 98% specificity and, therefore, a diagnostic ability comparable to the GRACE score. Furthermore, we only observed mortality in the highest PLR tertile and the high-risk category of the GRACE score.

Our findings show that we align with and confirm the growing evidence regarding PLR as a prognostic marker in acute coronary syndromes (ACS). Wang et al. (2018) indicated that a higher PLR recorded on admission was associated with increased risk of in-hospital mortality and major adverse cardiovascular events (MACE) in patients with acute myocardial infarction (AMI) undergoing primary PCI. Yang et al. (2020) found that PLR was an independent predictor of in-hospital death in patients with AMI, whereby the worst outcomes correlated with an increase in calculated PLR. In our study, PLR remained a powerful predictor, even after controlling for the GRACE score, suggesting that the PLR may provide incremental prognostic information compared to conventional scoring systems.

The observed relationship between higher PLR and adverse outcomes may reflect the contribution of both thrombocytosis and lymphopenia in the context of STEMI, as both drive downstream pathophysiology. Specifically, platelets' role in thrombus formation, propagating inflammation, and lymphocyte depletion reflects stress-related immune suppression.[14] Indeed, this mechanism was supported in previous work from Sun et al,[15] who also demonstrated a correlation between PLR and markers of endothelial dysfunction, with an association between PLR and impaired microvascular reperfusion following PCI.

Our study also parallels the observations of Pruc et al,[16] where a systematic review investigated all subtypes of ACS and concluded that high PLR was consistently related to higher mortality and MACE. Interestingly, the diagnostic utility of PLR in our study (AUC=0.990) is among the highest reported. This is likely due to the homogeneous nature of our STEMI group and the exclusion of other inflammatory confounders.

From a clinical utility perspective, PLR is uniquely advantageous over more complex risk scores in various settings. First, it is quick and easy to calculate from routine blood counts at admission. Second, it is much easier to calculate in lower resource environments. This concept of hematological indices, including PLR, as early, cost-effective measures for stratifying patients with AMI was echoed in the conclusions of Xu et al.[17]

Nonetheless, our findings must be interpreted with some limitations in mind. The study was conducted at a single center with a small sample size, which may affect generalizability. The cut-off points for PLR are highly variable across studies,[9-12] and currently, there is no accepted cut-off. Furthermore, while we took measures to exclude patients with overt inflammatory or hematologic diseases, it is still possible that some aspects of subclinical inflammation influenced PLR.

Future studies should consider validating PLR cut-offs in larger multi-ethnic populations and the additional prognostic ability of PLR when combined with existing scoring systems, such as GRACE. Finally, longitudinal studies measuring PLR beyond the initial acute phase may help to clarify the role PLR has in predicting long-term outcomes.

Our results prove that admission PLR is a straightforward, inexpensive, and highly precise prognostic indicator for STEMI patients. In the case of STEMI patients, using PLR might lead to early identification of high-risk patients when used alongside the GRACE score and provide an opportunity for early intensive interventions that may reduce in-hospital mortality. While this

study was helpful, it is a single-center study with a small sample size and may limit the extrapolation of the results. While we believe we excluded patients with clinically obvious inflammatory or hematologic disorders, subclinical inflammatory and pertinent hematologic conditions may have altered PLR results. Additionally, variability in the optimal cut-offs for PLR among different populations means that the results should be interpreted cautiously until validated in larger, multicenter cohorts.

Conclusion

Admission PLR is a rapid, low-cost, and highly accurate predictor of in-hospital mortality, on par with established GRACE score benchmarks, and has the additional advantage of reflecting values from routine blood work at the same time without requiring extra effort or financial considerations. Admission PLR can be used with existing risk stratification models to improve early triage, especially when time is short or resources in the emergency department are scarce. Including admission PLR into clinical pathways has the potential to increase prognostic accuracy as well as optimize the quality of acute coronary care.

Financial support and sponsorship

Nil.

Conflicts of interest

There are no conflicts of interest.

References

1. World Health Organization. CVDs. https://www.who.int/news-room/fact-sheets/detail/cardiovascular-diseases-(cvds)
2. World Heart Federation. World Heart Report 2023. https://world-heart-federation.org/wp-content/uploads/World-Heart-Report-2023.pdf
3. Canto, J. G., Rogers, W. J., Goldberg, R. J., et al. (2012). Association of age and sex with myocardial infarction symptom presentation and in-hospital mortality. JAMA, 307(8), 813–822. https://doi.org/10.1001/jama.2012.199
4. Libby, P. (2021). Inflammation in atherosclerosis: A key pathophysiological mechanism. Cardiovascular Research, 117(13), 2525–2540. https://academic.oup.com/cardiovascres/article/117/13/2525/6373891
5. Hansson, G. K. (2005). Inflammation, atherosclerosis, and coronary artery disease. New England Journal of Medicine, 352(16), 1685–1695. https://doi.org/10.1056/NEJMra043430
6. Mitsis A, Avraamides P, Lakoumentas J, Kyriakou M, Sokratous S, Karmioti G, Drakomathioulakis M, Theodoropoulos KC, Nasoufidou A, Evangeliou A, Vassilikos V, Fragakis N, Ziakas A, Tzikas S, Kassimis G. Role of inflammation following an acute myocardial infarction: design of INFINITY. Biomark Med. 2023 Dec;17(23):971-981. doi: 10.2217/bmm-2023-0491. Epub 2024 Jan 18. PMID: 38235565.
7. Balagopal PB, de Ferranti SD, Cook S, Daniels SR, Gidding SS, Hayman LL, et al. Nontraditional risk factors and biomarkers for cardiovascular disease: mechanistic, research, and clinical considerations for youth: a scientific statement from the American Heart Association. Circulation. 2011;123:2749–2769. doi: 10.1161/CIR.0b013e31821c7c64.
8. Martín-Ventura JL, Blanco-Colio LM, Tuñón J, Muñoz-García B, Madrigal-Matute J, Moreno JA, Vega de Céniga M, Egido J. Biomarkers in cardiovascular medicine. Rev Esp Cardiol. 2009 Jun;62(6):677-88. English, Spanish. doi: 10.1016/s1885-5857(09)72232-7. PMID: 19480764.
9. Yusuf, S., Rangarajan, S., Teo, K., et al. (2020). Cardiovascular risk and events in 17 low-, middle-, and high-income countries. New England Journal of Medicine, 382(8), 768–779. https://doi.org/10.1056/NEJMoa1913981
10. Li T, Yan Z, Fan Y, et al. Cardiac repair after myocardial infarction: A two-sided role of inflammation-mediated. Front Cardiovasc Med. 2023;9:1077290.
11. Acet, H., Ertaş, F., Bilik, M. Z., Akıl, M. A., Özyurtlu, F., Aydın, M., ... & Toprak, N. (2015). Original paper The relationship between neutrophil to lymphocyte ratio, platelet to lymphocyte ratio and thrombolysis in myocardial infarction risk score in pts with ST elevation acute myocardial infarction before primary coronary intervention. Advances in Interventional Cardiology/Postępy w Kardiologii Interwencyjnej, 11(2), 126-135.
12. Li, L., Ma, Y., Geng, X. B., Tan, Z., Wang, J. H., Cui, C., ... & Shang, X. M. (2021). Platelet-to-lymphocyte ratio relates to poor prognosis in elderly pts with acute myocardial infarction. Aging clinical and experimental research, 33, 619-624.
13. Wang, H., Li, L., & Ma, Y. (2023). Platelet-to-lymphocyte ratio: A potential prognosticator in acute myocardial infarction: A prospective longitudinal study. Clinical Cardiology, 46(5), e24002. https://doi.org/10.1002/clc.24002
14. Yang, Z., Qiao, Y., Wang, D., & Yan, G. (2021). Platelet-lymphocyte ratio as a new predictor of in-hospital mortality in pts with acute myocardial infarction. Scientific Reports, 11, 1–9. https://doi.org/10.1038/s41598-021-02686-1
15. Sun, H., Liu, H., Li, J., Kou, J., & Yang, C. (2024). Analysis of the clinical predictive value of the novel inflammatory indices SII, SIRI, MHR and NHR in pts with acute myocardial infarction and their extent of coronary artery disease. Journal of Inflammation Research, 17, 7325–7338. https://doi.org/10.2147/JIR.S479253
16. Pruc, M., Peacock, F. W., Rafique, Z., et al. (2023). The prognostic role of platelet-to-lymphocyte ratio in acute coronary syndromes: A systematic review and meta-analysis. Journal of Clinical Medicine, 12(21), 6903. https://doi.org/10.3390/jcm12216903
17. Xu, X., Zhang, D., & Liu, J. (2023). Prognostic biomarkers for acute myocardial infarction: Insights from molecular studies. International Journal of Molecular Sciences, 23(16), 9168. https://www.mdpi.com/1422-0067/23/16/9168.