DOI: https://doi.org/10.48305/arya.2024.42494.2942

Eosinophil percentage and platelet counts: Association with in-hospital mortality in ST-segment elevated myocardial infarction

Mahtab Mashayekhi[(1)], Mahdokht Rezaei[(1)], Abbas Allami[(1)], Narges Bazgir[(2)], Monirsadat Mirzadeh[(3)], Shahin Aliakbari[(1)], Kimia Rahimi Ardali[(1)]

Original Article

Abstract

BACKGROUND: ST-segment elevation myocardial infarction (STEMI) results from coronary artery blockage due to ruptured atherosclerotic plaque. Eosinophils play a dual role in STEMI, contributing to thrombus formation and tissue repair. This study investigates the association between eosinophil percentage, platelet counts, and in-hospital prognosis in STEMI patients.

METHODS: A cross-sectional study was conducted from September 2019 to February 2020, including patients aged 18 and above with a STEMI diagnosis. In-hospital mortality, arrhythmia, and left ventricular ejection fraction (LVEF) were recorded. Demographic data, clinical manifestations, and laboratory investigations were collected. Data were analyzed using SPSS (version 25.0), with a P value of <0.05 considered significant.

RESULTS: The study included 100 STEMI patients with a mean age of 65±13.26 years; 75% were male. The mortality rate was 13%. A significant relationship was found between eosinophil percentage and mortality (p=0.032), and platelet count also correlated significantly with mortality (p=0.008). The association between eosinophil percentage and EF was significant (p<0.001). The area under the ROC curve was 0.705 (95% CI 0.605 - 0.792) for platelet counts and 0.679 (95% CI 0.577 - 0.770) for eosinophil percentage in differentiating live and expired patients.

CONCLUSION: Platelet count could be a significant prognostic indicator for in-hospital outcomes in STEMI patients, suggesting an increased risk of mortality. Additionally, there is a notable relationship between eosinophil percentage and ejection fraction (EF).

Keywords: Acute Myocardial Infarction; Platelet Counts; Eosinophil Percentage, Ejection Fraction

Date of submission: 2024-02-05, ***Date of acceptance:*** 2024-11-11

Introduction

ST-segment elevation myocardial infarction (STEMI), primarily triggered by the blockage of the coronary artery due to the rupture of an atherosclerotic plaque, is the prominent cause of death worldwide[1, 2]. The economic burden of hospitalizations due to STEMI is substantial, with an estimated $12.1 billion spent on related hospital stays in the US in 2013 alone, underscoring its significant morbidity and impact on the healthcare system[3].

Eosinophils, although present in low quantities in the bloodstream, play a crucial role in the body's

1- Boali Hospital, Qazvin University of Medical Sciences, Qazvin, Iran
2- Hearing Disorders Research Center, Shahid Beheshti University of Medical Sciences, Tehran, Iran
3- Department of Community Medicine, Metabolic Diseases Research Center, Research Institute for Prevention of Non-Communicable Diseases, Qazvin University of Medical Sciences, Qazvin, Iran
Address for correspondence: Mahdokht Rezaei; Boali Hospital, Qazvin University of Medical Sciences, Qazvin, Iran; Email: rezaiemahdokht@yahoo.com.
Abbas Allami; Boali Hospital, Qazvin University of Medical Sciences, Qazvin, Iran; Email: allami9@yahoo.com

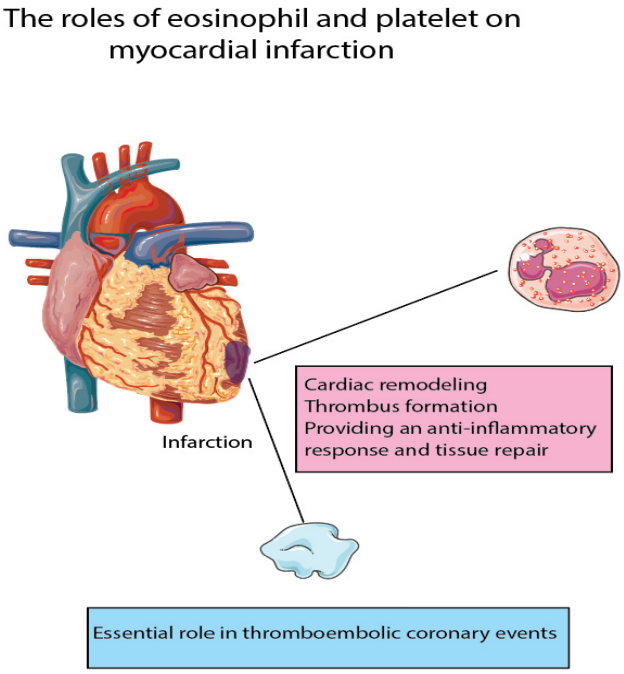


Figure 1. summarizes the function of eosinophil and platelets in acute myocardial infarction.

response to parasitic infections and allergies. They produce a variety of substances, including eosinophil peroxidase (EPO), eosinophil-derived neurotoxins (EDN), eosinophil cationic protein (ECR), galactin 10, and major basic protein (MBP)[4]. Furthermore, studies have shown that eosinophils play a significant role in coronary artery diseases, including STEMI, where they serve a dual function.

They contribute to the formation of thrombus by activating platelets, promoting exposure of endothelial tissue factor, and exhibiting prothrombotic properties, which could potentially influence the severity of myocardial damage[5-7]. Additionally, eosinophils contain IL-4 within their cytoplasmic granules, which provides an anti-inflammatory response and aids in tissue repair[8]. Platelet aggregation at sites of vessel injury plays an essential role in thromboembolic coronary events[9] (Figure 1).

Patients with acute STEMI and higher platelet counts have been observed to have worse outcomes, such as heart failure, arrhythmia, infarction, and death[10–12]. Conversely, lower admission platelet counts result in higher diastolic velocity in the left anterior descending coronary artery and better myocardial perfusion in patients with anterior STEMI treated by primary percutaneous coronary intervention (PPCI). Higher platelet counts are associated with lower left ventricular systolic function[13].

To our knowledge, few studies have discussed the relationship between eosinophil range and in-hospital prognosis of STEMI, thus there is debate on it. This study investigated the association between eosinophil percentage, platelet counts, and in-hospital prognosis in patients with STEMI. We also evaluated the effects of eosinophil percentage and platelet counts on in-hospital arrhythmia, heart failure, and hospital stay.

Methods

Study design

A cross-sectional study was performed on patients with STEMI who were admitted from September 2019 to February 2020. Qazvin School of Medicine Ethical Committee approved this survey. The study followed the Declaration of Helsinki. All eligible participants were selected after explaining the purpose and the survey process, and then informed written consent was obtained.

Case definition, laboratory data collection

We included all patients aged older than 18 who were admitted to the emergency department with the primary diagnosis of STEMI. These patients were candidates for PPCI or fibrinolytic therapy within

24 hours from the onset of chest pain. STEMI was diagnosed by a cardiologist based on the third universal definition of AMI, which includes the following criteria:

1. Typical chest pain or discomfort in the chest area.
2. Changes in electrocardiogram (ECG) that can be recognized as ST-segment elevations or as left bundle branch blocks (LBBB).
3. Elevation in levels of cardiac-specific enzymes (CPK, troponins, etc.)[14].

Individuals with infections, cancer, asthma, or congenital diseases that alter platelet and eosinophil percentages were excluded. Additionally, patients taking treatments such as pyrimethamine and dapsone, which increase eosinophil levels, were excluded. After these exclusions, 100 individuals were available for analysis. The primary endpoints of the present study were in-hospital mortality, arrhythmia, and left ventricular ejection fraction (LVEF).

Each participant's demographic data, clinical manifestations, and laboratory investigation results were recorded. The laboratory tests included total cholesterol, complete blood cell count (CBC), and troponin levels, obtained 24 hours after the patient's admission.

The cardiac function of each participant was evaluated by echocardiography, and LVEF was calculated. Patients were followed until discharge, and in-hospital mortality and complications, such as arrhythmias, were recorded. A checklist containing relevant data from the patients was designed. The checklist consisted of 19 items: age, gender, systolic and diastolic pressures, diabetes, ejection fraction (EF), troponin level, chronic kidney disease, in-hospital mortality, and complications such as arrhythmia.

Statistical analysis

The data were analyzed using the Statistical Package for Social Sciences (SPSS) software (version 25.0; IBM et al.). The qualitative data were reported by frequency and percentage. The continuous variables were summarized using appropriate measures of central tendency and dispersion. Normally distributed variables were presented as means with standard deviations (mean ± SD), while non-normally distributed variables were reported as medians with interquartile ranges (median [IQR]). The normality of the distributions was assessed using the Kolmogorov–Smirnov (K-S) test, and for variables with small sample sizes (<50), the Shapiro-Wilk normality test was used instead of the K-S test. The independent t-test and the Mann-Whitney U test were used to compare the data between deceased and surviving patients. The associations of evaluated variables, eosinophil percentage and platelet count, were assessed using Pearson's and Spearman's correlation tests. A P value less than 0.05 was considered statistically significant.

Results

One hundred patients presenting with STEMI over six months met our inclusion criteria and entered the study. Patients' mean age (mean ± SD) was 65±13.26 years, and the majority (75%) were men. Additionally, half of the included individuals suffered from hypertension. Due to the frequency of hypertension in patients, the most consumed drugs were angiotensin-receptor blockers (ARBs) and aspirin. The mean systolic and diastolic pressure levels were 132±26 and 86±7, respectively. The mean EF was 38.11±11.27. Demographic, clinical, and laboratory investigations were conducted for STEMI patients; their results are demonstrated in Table 1.

As illustrated in Table 2, the median [IQR] eosinophil percentage was 2 [1-2]. The mortality rate was 13%, and 12% of the patients had life-threatening arrhythmias.

After conducting the Kolmogorov–Smirnov test, it was revealed that despite platelet count, eosinophil percentage distribution was not normal. As a result, to assess the association between study variables, the Spearman correlation statistical test was performed.

The results of the binary logistic regression analysis for predicting mortality in patients with STEMI are presented in Table 3. Age was identified as a significant predictor of mortality, while sex and DM showed potential associations. Other variables, including HTN, ACE/ARBs, statin, and ASA use, were not significantly associated with mortality outcomes in this patient population.

Table 4 demonstrates the association of platelet and eosinophil percentages with mortality and arrhythmia. A significant relationship between platelet and eosinophil percentages with the mortality rate of the patients was evident (p=0.008, p=0.032, respectively). The logistic regression analysis effectively adjusted for potential confounding factors and revealed the

Table 1. Demographic and Clinical Characteristics of STEMI Patients

Demographic characteristics	mean ± SD or n (%)
Age (years)	63 ± 13.26
Male, n (%)	75 (75)
Past medical history	
Systolic blood pressure (mmHg)	132 ± 26
Diastolic blood pressure (mmHg)	86 ± 17
Hypertension, n (%)	50 (50)
Diabetes Mellitus, n (%)	33 (33)
Drug history	
Aspirin, n (%)	14 (14)
ARBs, n (%)	20 (20)
ACE inhibitor, n (%)	2 (2)
Statin, n (%)	8 (8)
Biochemical profile	
Triglycerides (mg/dl)	127 ± 50
Total cholesterol (mg/dl)	155 ± 37

ARBs: Angiotensin receptor blockers, ACE inhibitor: Angiotensin-converting enzyme inhibitor

Table 2. Endpoints of study

Characteristic	mean ± SD or n (%)
Ejection Fraction (%)	38.11 ± 11.27
Arrhythmia (%)	12 (12)
Mortality (%)	13 (13)
Troponin level (ng/mL)	10.72 ±17.88
Hospitalization days (day)	10.32 ± 6.28
Platelet count ($10^3/\mu L$)	223 ± 67
Eosinophil percentage	2 [1 -2]

independent associations of these hematological parameters (platelet) with the outcome of interest. The adjusted odds ratio for platelet count is also statistically significant, suggesting that a higher platelet count is associated with an increased risk of mortality, even after accounting for the confounding effects of age (Odds ratio 1.011, CI 95%: 1.001 - 1.020).

However, no significant relation was observed between platelet and eosinophil percentage with arrhythmia (p=0.571 and p=0.679, respectively).

Table 5 shows the relationship between platelet and eosinophil percentage and EF, troponin level, and hospitalization days. We found that platelet count was related to troponin level (ρ=0.20, p=0.048).

Table 3. The binary logistic regression analysis for predicting mortality in patients with STEMI

Variables	Odds Ratio	95% Confidence Interval
Age (years)	1.086	1.016- 1.160
Sex (male)	1.004	0.215- 4.679
Diabetes mellitus (yes)	0.276	0.061- 1.240
Hypertension (yes)	3.014	0.744- 12.217
Statin	3.741	0.182- 76.697
Acetylsalicylic acid (aspirin)	0.584	0.060- 5.715
ACE (angiotensin converting enzyme) inhibitors/ ARBs (angiotensin receptor blockers)	0.345	0.080- 1.497

Variable(s) entered on step 1: Age, sex (male), Diabetes mellitus, Hypertension, Statin, ASA, angiotensin converting enzyme inhibitors/ angiotensin receptor blockers

Table 4. The Association of Platelet Counts and Eosinophil percent with Mortality and Arrhythmia in study Patients

Variables		Platelet count (10^3/μL)		Eosinophil %	
		mean ± SD	P value	Median [IQR]	P value
Mortality	Dead patients	269.54 ± 90.36	0.008*	1.08 [0 – 2]	0.032**
	Living patients	216.9 ± 61.070		1.95 [1 - 2.5]	
Arrhythmia	With	213.33 ± 85.10	0.571*	2.33 [0.25 - 3.75]	0.679**
	Without	223.70 ± 64.19		1.79 [1 – 2]	

** t-test, **Mann-Whitney Test, IQR: Interquartile Range*

Table 5. Association of platelet and eosinophil percentage with ejection fraction, troponin level, and hospitalization days

Variables	Platelet count (10^3/μL)		Eosinophil percentage (%)	
	ϱ	p value	ϱ	p value
Ejection Fraction	0.19	0.062	0.37*	<0.001
Troponin level	0.201*	0.048	-0.10	0.309
Hospitalization days	-0.088	0.385	0.04	0.699

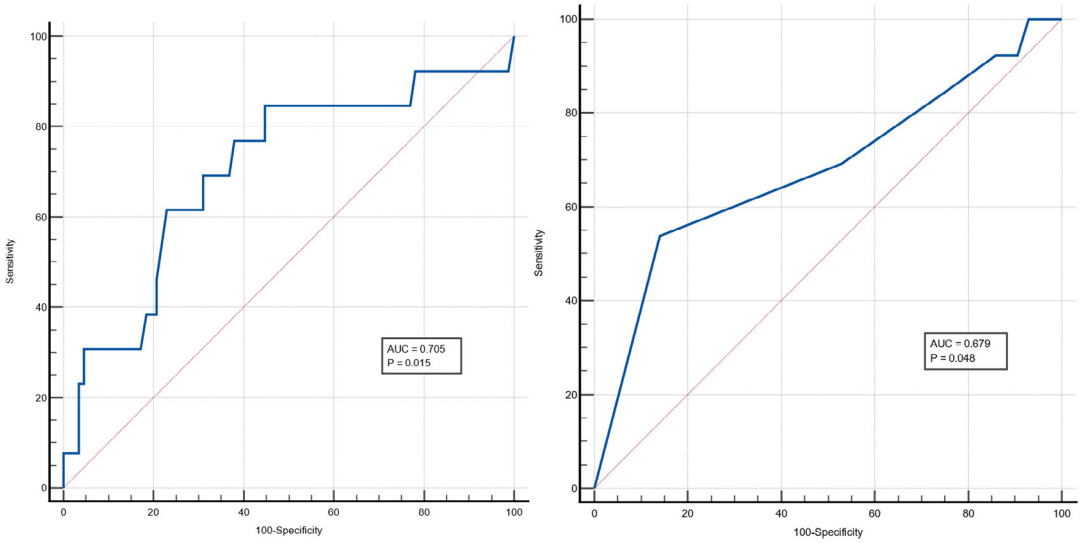
ϱ Spearman correlation coefficient, * significant

However, eosinophil percentage was significantly related to EF (ϱ=0.37, p<0.001), indicating that higher eosinophil percentage was associated with lower EF.

The area under the receiver operating characteristic (ROC) curve was 0.705 (95% Confidence Interval 0.605 - 0.792, p=0.015) when using platelet counts, and 0.679 (95% Confidence Interval 0.577 - 0.770, p=0.048) when using eosinophil percentage, to differentiate between live and expired STEMI patients during their in-hospital stay (Figure 2). A platelet count above 211 x 10^9/L has been associated with increased mortality in AMI patients. An eosinophil percentage of 0.5% or lower has been linked to a higher risk of mortality and poorer outcomes in AMI patients.

Discussion

The aim of this study was to examine the relationship between peripheral blood eosinophil percentage, platelet counts, and cardiac events in post-MI patients. We found that patients with STEMI who died during hospitalization had a significantly lower eosinophil percentage than those who survived. We also found a significant positive association between



platelet count

ROC for platelet	
Associated criterion (cut-off points)	>211
Sensitivity	84.62
Specificity	55.17
Positive Likelihood Ratio	1.89
Negative Likelihood Ratio	0.28
Positive Predictive Value (%)	22.0
Negative Predictive Value (%)	96.0

Eosinophile percentage

ROC for eosinophil	
Associated criterion (cut-off points %)	0.5
Sensitivity	53.85
Specificity	85.88
Positive Likelihood Ratio	3.81
Negative Likelihood Ratio	0.54
Positive Predictive Value (%)	36.3
Negative Predictive Value (%)	92.6

Figure 2. ROC curve analysis of platelet counts, and eosinophil percentage for differentiate between live and expired acute myocardial infarction patients during their in-hospital stay.

eosinophil percentage and EF, suggesting that a higher eosinophil percentage was associated with better cardiac function.

Recently, studies have shown that lower eosinophil levels are related to larger infarct size and long-term poor prognosis. Eosinophils usually peak two to three days after MI[15, 16]. Many factors play a role in cardiac remodeling after myocardial infarction. Immune system cells have both damaging and protective effects on cardiac remodeling. One key player in cardiac remodeling is the eosinophil[4].

Eosinophils can secrete various growth factors, such as epidermal growth factor, transforming growth factor alpha and beta, fibroblast growth factor, platelet-derived growth factor, and vascular endothelial growth factor, which may promote cardiac repair after MI. Eosinophils are also the source of protectin D1 and different resolvins, which can reduce inflammation and neutrophil infiltration[17, 18]. Post-mortem studies have shown that eosinophils accumulate in the myocardium of MI patients, but their distribution is scattered[19]. Similarly, experimental studies in mouse models have shown that eosinophils decrease in peripheral blood, but increase in the myocardium within 24 hours and peak at four days after MI[20].

However, the role of eosinophils in thrombosis and STEMI is unclear; whether it affects thrombosis or healing after STEMI. The question is whether the low eosinophil count in peripheral blood is related to the consumption of eosinophils during thrombosis[21] or if it was initially low in peripheral blood. As a result, there are many questions about the role of eosinophils in the post-MI heart. Xu et al. permanently ligated the left anterior descending coronary artery in mice. It was revealed that the amount of interleukin 5 (which induces eosinophils) increased five days after MI. Additionally, by administering interleukin 5, infarction size decreased, and angiogenesis and the ejection fraction rose. Consequently, they showed

that interleukin 5, by inducing eosinophils, accelerates cardiac recovery after MI[22].

Jiang et al. assessed patients with angina pectoris and acute MI. The peripheral blood eosinophil percentage in patients suffering from acute MI was considerably higher than in those with angina pectoris. There was also an inverse association between eosinophil percentage and troponin I level. Consequently, lower eosinophil percentages were related to more myocardial damage[23]. In the present survey, we did not find any significant relationship between troponin I levels and eosinophil percentage.

Many studies have also evaluated eosinophils' role as a prognostic biomarker in patients with acute MI. Our findings align with a study carried out by GÜNER et al.[5], which found a connection between eosinophil percentage and in-hospital prognosis among patients with STEMI. The study defined major adverse cardiac events (MACE) as re-infarction, ventricular arrhythmia, target vessel revascularization, the need for cardiopulmonary resuscitation, congestive heart failure, and cardiovascular mortality during the patient's hospital stay. The research found a link between eosinophil percentage (EOS%) and MACE, suggesting that eosinophils could have a role in affecting thrombotic processes beyond their known proinflammatory characteristics. Patients who had a lower percentage of eosinophils (EOS%) upon admission exhibited a higher risk of experiencing MACE, suggesting that EOS% could serve as a valuable biomarker for risk assessment in STEMI cases.

Ye et al.[7] evaluated the role of eosinophil percentage as a prognostic biomarker for MACEs. Their findings revealed that a lower eosinophil percentage was linked to an increased incidence of cardiac arrest, rupture, malignant arrhythmia, and poorer cumulative survival. Similarly, Firani et al. demonstrated the prognostic significance of eosinophil percentage and the eosinophil to leukocyte ratio for in-hospital mortality in patients with acute MI[24]. Furthermore, Konishi et al. found an association between a lower eosinophil to leukocyte ratio (ELR) within the first 24 hours of hospital admission and a heightened occurrence of MACEs within one year[25]. An analysis of 660 patients with cardiac diseases over a 3.5-year follow-up period showed that eosinopenia was associated with a larger infarct size and poorer clinical outcomes[26]. In China, a study assessed patients with triple-vessel coronary artery disease to determine the prognostic value of white blood cell function for mortality. The study found that an increase in eosinophils predicted death[27]. The findings of this study were in contrast to ours and previously discussed reports, possibly due to differences in the cases assessed.

In our study, the ROC curve analysis revealed that eosinophil levels do not have significant predictive power for mortality. Consequently, eosinophil levels may not serve as a relevant biomarker for in-hospital mortality in this specific patient population. Notably, our results contrast with some previous studies, which may be attributed to differences in the populations examined and the focus on in-hospital mortality as a short-term outcome.

In our study, we found that platelet counts were significantly higher in patients who did not survive. This is consistent with the findings of Sharif et al., who reported that lower platelet counts at admission in STEMI patients who underwent PPCI were associated with improved myocardial perfusion and diastolic velocities in the left anterior descending coronary artery. On the other hand, higher platelet counts were associated with poorer left ventricular systolic function both at admission and before discharge[13]. Another study assessed the impact of platelet counts on STEMI patients and found that higher platelet counts were related to a significantly higher risk of in-hospital mortality and heart failure among STEMI patients, while the difference in reinfarction rates was not statistically significant[11]. The findings of our study align with these reports, indicating that higher platelet counts are associated with a higher in-hospital mortality rate.

Conclusion

In conclusion, our study suggests that both platelet count could be potential biomarkers for mortality in STEMI patients. These findings highlight the potential role of platelet in predicting mortality in STEMI patients, and the specific association of eosinophil percentage with EF. However, further research is needed to confirm these findings and to explore the potential mechanisms underlying these associations.

Limitation

This study had a relatively low number of included patients and was conducted in a single center. Multi-

center prospective studies with more cases and longer duration of follow-up would be valuable to find the exact relation between eosinophil percentage and in-hospital adverse events.

Acknowledgements

We wish to thank everyone who contributed to this study. Parts of the figure were drawn using pictures from Servier Medical Art. Servier Medical Art by Servier is licensed under a Creative Commons Attribution 3.0 Unported License (https://creativecommons.org/licenses/by/3.0/).

Conflict of interests

The authors declare no conflict of interest.

Funding

There is no funding in this study.

Author's Contributions

MR, MM and AA conceptualized the study, MM acquisition of data, MM, AA, NB, SA drafting the manuscript, AA, NB, MM, KRA revising for critical intellectual concept and approved of the version to be submit.

References

1. Choudhury T, West NE, El-Omar M. ST elevation myocardial infarction. Clin Med (Lond). 2016 Jun;16(3):277-82. https://doi.org/10.7861/clinmedicine.16-3-277
2. Bates ER, Jacobs AK. Time to treatment in patients with STEMI. N Engl J Med. 2013 Sep 5;369(10):889-92. https://doi.org/10.1056/nejmp1308772
3. Ahuja KR, Saad AM, Nazir S, Ariss RW, Shekhar S, Isogai T, et al. Trends in Clinical Characteristics and Outcomes in ST-Elevation Myocardial Infarction Hospitalizations in the United States, 2002-2016. Curr Probl Cardiol. 2022 Dec;47(12):101005. https://doi.org/10.1016/j.cpcardiol.2021.101005
4. Ramirez GA, Yacoub MR, Ripa M, Mannina D, Cariddi A, Saporiti N, et al. Eosinophils from Physiology to Disease: A Comprehensive Review. Biomed Res Int. 2018 Jan 28;2018:9095275. https://doi.org/10.1155/2018/9095275
5. Güner A, Zehİr R, Kalçlk M, Uslu A, Ösken A, Kalkan AK, et al. Eosinophil percentage as a new prognostic marker in patients with ST-segment elevation myocardial infarction undergoing primary percutaneous coronary intervention. Interv Med Appl Sci. 2019 Oct 7;11(3):146-53. https://doi.org/10.1556/1646.11.2019.17
6. Sincer I, Gunes Y, Mansiroglu AK, Aktas G. Differential value of eosinophil count in acute coronary syndrome among elderly patients. Aging Male. 2020 Dec;23(5):958-61. https://doi.org/10.1080/13685538.2019.1643310
7. Ye L, Bai HM, Jiang D, He B, Wen XS, Ge P, et al. Combination of eosinophil percentage and high-sensitivity C-reactive protein predicts in-hospital major adverse cardiac events in ST-elevation myocardial infarction patients undergoing primary percutaneous coronary intervention. J Clin Lab Anal. 2020 Sep;34(9):e23367. https://doi.org/10.1002/jcla.23367
8. Toor IS, Rückerl D, Mair I, Ainsworth R, Meloni M, Spiroski AM, et al. Eosinophil Deficiency Promotes Aberrant Repair and Adverse Remodeling Following Acute Myocardial Infarction. JACC Basic Transl Sci. 2020 Jul 8;5(7):665-81. https://doi.org/10.1016/j.jacbts.2020.05.005
9. Packham MA. Role of platelets in thrombosis and hemostasis. Can J Physiol Pharmacol. 1994 Mar;72(3):278-84. https://doi.org/10.1139/y94-043
10. Turner SJ, Ketch TR, Gandhi SK, Sane DC. Routine hematologic clinical tests as prognostic markers in patients with acute coronary syndromes. Am Heart J. 2008 May;155(5):806-16. https://doi.org/10.1016/j.ahj.2007.11.037
11. Paul GK, Sen B, Bari MA, Rahman Z, Jamal F, Bari MS, et al. Correlation of platelet count and acute ST-elevation in myocardial infarction. Mymensingh Med J. 2010 Jul;19(3):469-73.
12. Kurtul A, Yarlioglues M, Murat SN, Ergun G, Duran M, Kasapkara HA, et al. Usefulness of the platelet-to-lymphocyte ratio in predicting angiographic reflow after primary percutaneous coronary intervention in patients with acute ST-segment elevation myocardial infarction. Am J Cardiol. 2014 Aug 1;114(3):342-7. https://doi.org/10.1016/j.amjcard.2014.04.045
13. Sharif D, Abu-Salem M, Sharif-Rasslan A, Rosenschein U. Platelet counts on admission affect coronary flow, myocardial perfusion and left ventricular systolic function after primary percutaneous coronary intervention. Eur Heart J Acute Cardiovasc Care. 2017 Oct;6(7):632-9. https://doi.org/10.1177/2048872616643690
14. Thygesen K, Alpert JS, Jaffe AS, Simoons ML, Chaitman BR, White HD. Third universal definition of myocardial infarction. circulation. 2012;126(16):2020-35. https://doi.org/10.1161/

CIR.0b013e31826e1058
15. Liu J, Yang C, Liu T, Deng Z, Fang W, Zhang X, et al. Eosinophils improve cardiac function after myocardial infarction. Nat Commun. 2020;11(1):6396. https://doi.org/10.1038/s41467-020-19297-5
16. Rios-Navarro C, Gavara J, Vidal V, Bonanad C, Racugno P, Bayes-Genis A, et al. Characterization and implications of the dynamics of eosinophils in blood and in the infarcted myocardium after coronary reperfusion. PLoS One. 2018 Oct 26;13(10):e0206344. https://doi.org/10.1371/journal.pone.0206344
17. Wen T, Rothenberg ME. The Regulatory Function of Eosinophils. Microbiol Spectr. 2016 Oct;4(5):10.1128/microbiolspec.MCHD-0020-2015. https://doi.org/10.1128/microbiolspec.mchd-0020-2015
18. Isobe Y, Kato T, Arita M. Emerging roles of eosinophils and eosinophil-derived lipid mediators in the resolution of inflammation. Front Immunol. 2012 Aug 28;3:270. https://doi.org/10.3389/fimmu.2012.00270
19. Atkinson JB, Robinowitz M, McAllister HA, Virmani R. Association of eosinophils with cardiac rupture. Hum Pathol. 1985 Jun;16(6):562-8. https://doi.org/10.1016/s0046-8177(85)80105-2
20. Bass DA. Behavior of eosinophil leukocytes in acute inflammation. II. Eosinophil dynamics during acute inflammation. J Clin Invest. 1975 Oct;56(4):870-9. https://doi.org/10.1172/jci108166
21. Frangogiannis NG. The inflammatory response in myocardial injury, repair, and remodelling. Nat Rev Cardiol. 2014 May;11(5):255-65. https://doi.org/10.1038/nrcardio.2014.28
22. Xu JY, Xiong YY, Tang RJ, Jiang WY, Ning Y, Gong ZT, et al. Interleukin-5-induced eosinophil population improves cardiac function after myocardial infarction. Cardiovasc Res. 2022 Jul 20;118(9):2165-78. https://doi.org/10.1093/cvr/cvab237
23. Jiang P, Wang DZ, Ren YL, Cai JP, Chen BX. Significance of eosinophil accumulation in the thrombus and decrease in peripheral blood in patients with acute coronary syndrome. Coron Artery Dis. 2015 Mar;26(2):101-6. https://doi.org/10.1097/mca.0000000000000186
24. Firani NK, Hartanti KD, Purnamasari P. Hematological Parameter as Predictor Mortality in Acute Myocardial Infarction Patients. Int J Gen Med. 2022 Aug 23;15:6757-63. https://doi.org/10.2147/ijgm.s380659
25. Konishi T, Funayama N, Yamamoto T, Morita T, Hotta D, Nishihara H, et al. Prognostic Value of Eosinophil to Leukocyte Ratio in Patients with ST-Elevation Myocardial Infarction Undergoing Primary Percutaneous Coronary Intervention. J Atheroscler Thromb. 2017 Aug 1;24(8):827-40. https://doi.org/10.5551/jat.37937
26. Alkhalil M, Kearney A, Hegarty M, Stewart C, Devlin P, Owens CG, et al. Eosinopenia as an Adverse Marker of Clinical Outcomes in Patients Presenting with Acute Myocardial Infarction. Am J Med. 2019 Dec;132(12):e827-34. https://doi.org/10.1016/j.amjmed.2019.05.021
27. Zhao X, Jiang L, Xu L, Tian J, Xu Y, Zhao Y, et al. Predictive value of in-hospital white blood cell count in Chinese patients with triple-vessel coronary disease. Eur J Prev Cardiol. 2019 May;26(8):872-82. https://doi.org/10.1177/2047487319826398

How to cite this article: Mashayekhi M, Rezaei M, Allami A, Bazgir N, Mirzadeh M, Aliakbari Sh, et al. **Eosinophil percentage and platelet counts: Association with in-hospital mortality in ST-segment elevated myocardial infarction.** ARYA Atheroscler. 2024; 20(6): 34-42.