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The diagnostic value of eosinophil-to-lymphocyte ratio in predicting contrast-induced nephropathy in patients with ST-segment elevation myocardial infarction

Orhan Karayiğit[1], Murat Gök[2], Ahmet Balun[3], Alparslan Kurtul[4]

[1]*Department of Cardiology, Yozgat City Hospital, Yozgat, Turkiye*
[2]*Department of Cardiology, Faculty of Medicine, Trakya University, Edirne, Turkiye*
[3]*Department of Cardiology, Faculty of Medicine, Bandırma Onyedi Eylül University, Balıkesir, Turkiye*
[4]*Department of Cardiology, Faculty of Medicine, Mustafa Kemal University, Hatay, Turkiye*

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ABSTRACT

Aims: Inflammation is considered a major contributor to the development of contrast-induced nephropathy (CIN). The purpose of this study was to assess the effectiveness of eosinophil-to-lymphocyte ratio (ELR) as a predictor of CIN among patients who experienced percutaneous coronary intervention (PCI) for ST-segment elevation myocardial infarction (STEMI).

Methods: The study involved 440 patients diagnosed with STEMI who underwent primary PCI. The participants were categorized into two classes based on whether they had CIN or not. ELR was calculated by dividing the eosinophil count by the lymphocyte count.

Results: The group with CIN (+) showed significantly higher ELR levels (0.134±0.063 vs. 0.069±0.037, p<0.001). According to the ROC curve assessment, the best threshold level of ELR to predict CIN development was identified as 0.093, with 75.9% sensitivity and 79.1% specificity (AUC: 0.836; 95% CI: 0.783–0.889; p<0.001). Logistic regression analysis revealed that estimated glomerular filtration rate (eGFR), C-reactive protein, left ventricular ejection fraction, and ELR were independent predictors of CIN.

Conclusion: ELR could be an effective and reliable marker to predict CIN development in individuals with STEMI who undergo primary PCI. Early prediction of CIN risk is critical to provide intensive preventive measures for high-risk patients.

Keywords: Eosinophil-to-lymphocyte ratio, contrast-induced nephropathy, percutaneous coronary intervention, ST-segment elevation myocardial infarction

INTRODUCTION

Contrast-induced nephropathy (CIN) represents a significant complication that occurs after percutaneous coronary intervention (PCI).[1] High-risk conditions such as diabetes and hypertension, combined with the increasing number of invasive cardiac procedures, CIN remains a clinically significant concern. CIN is characterized by an elevation in serum creatinine concentrations of a minimum of 0.5 mg/dl or a 25% rise from baseline, taking place within 72 hours following exposure to contrast agents.[2] Based on current studies, the risk of developing CIN after PCI varies between 6% and 24%.[3] The primary cause of CIN is pre-existing chronic kidney disease (CKD). Additionally, patients with triggering factors such as acute coronary syndrome, hypotension, nephrotoxic drug use, and anemia have an increased likelihood of experiencing CIN. CIN can lead to extended hospital stays, escalating treatment costs, and an increase in mortality rates.[4]

While the exact mechanism behind CIN remains unclear, there is a strong connection between inflammation and the onset of CIN.[5] As a result, biomarkers associated with inflammation are currently a focal point of intense study in this field. Eosinophils are actively involved in the processes of inflammation, endothelial injury, and vascular thrombosis.[6] The cytotoxic granules they release (such as major basic protein-1) and the various enzymes, cytokines, and chemokines they produce contribute to the progression or resolution of inflammation. Reduced lymphocyte counts are closely tied to inflammation and significantly influence both the onset and progression of atherosclerosis.[7] An increase in eosinophils and low lymphocyte levels reflect systemic inflammation. The eosinophil-to-lymphocyte ratio (ELR) is an inflammatory marker that takes into account both eosinophil and lymphocyte counts. Recent studies have

Corresponding Author: Orhan Karayiğit, orphan_8_9@hotmail.com

indicated that ELR is connected to clinical outcomes in a range of cardiovascular conditions.[8,9]

CIN is linked to adverse clinical outcomes.[10] By implementing existing preventive strategies before the PCI procedure, we can significantly lower the risk of developing CIN and effectively prevent its progression. Therefore, identifying patients at higher risk early is essential for significantly improving clinical outcomes. This research sought to assess the effectiveness of ELR as a predictor of CIN among patients who experienced PCI for ST-elevation myocardial infarction (STEMI).

METHODS

Ethics

The study protocol received approval from Yozgat Bozok University Non-interventional Clinical Researches Ethics Committee (Date: 18.12.2024, Decision No: 2024-GOKAEK-2415_2024.12.18_251). All procedures were carried out in accordance with the ethical rules and the principles of the Declaration of Helsinki. Participants' formal informed permission was not acquired because the study was a retrospective design.

Study Design

An overall of 440 consecutive STEMI patients, who were admitted to the cardiology unit and underwent PCI from January 2023 to October 2024, were part of this cross-sectional, single-center study. According to current clinical practice guidelines, STEMI was diagnosed when there was ST-segment elevation in two or more adjacent leads on the electrocardiography or the presence of newly developed left bundle branch block, along with signs of ischemia and/or increased levels of cardiac biomarkers indicating myocardial injury.[11] The exclusion criteria were: patients who received thrombolytic therapy before PCI, those with end-stage renal failure [estimated glomerular filtration rate (eGFR) <15 ml/min/1.73 m^2] or undergoing dialysis, evidence of active infection or active malignancy, chronic liver disease, a history of hematologic disorders, autoimmune, allergic, or chronic inflammatory diseases, those on chronic steroid or immunosuppressant/immunomodulatory therapy, those using nephrotoxic drugs, those who had contrast-enhanced imaging other than coronary angiography in the previous days, and those who did not undergo PCI.

All demographic details, such as gender, age, smoking status, and coronary artery disease (CAD) history, were collected from the hospital's record system. Additionally, data related to vital signs at admission, including both systolic and diastolic blood pressure readings, was documented for the research.

Laboratory Measurements

Venous blood samples for routine analysis were drawn from all subjects before undergoing coronary angiography. Complete blood count parameters were evaluated using an automated cell counter (Beckman Coulter LH 750; Beckman Coulter Inc., USA). Biochemical tests, which included measurements of serum glucose, lipid levels, and creatinine, were conducted using established laboratory methods (Beckman Coulter Inc., USA). In all patients analyzed, serum creatinine was assessed once daily during their hospital stay, both before and after primary PCI. The equation from the CKD epidemiology collaboration (CKD-EPI) was employed to compute the eGFR.[12] The ELR was calculated by taking the eosinophil count and dividing it by the lymphocyte count. Left ventricular ejection fraction (LVEF) was identified using Simpson's method through standard transthoracic echocardiography (Vivid 7 GE Medical System) within 24 hours following the PCI.

CIN was defined by an elevation in serum creatinine levels, specifically a rise of at least 0.5 mg/dl or 25% over the baseline, occurring within 72 hours following exposure to contrast agents.[2] Based on this standard, the subjects were divided into two classes: CIN (+) and CIN (-).

Angiographic Analysis

Based on the operator's choice, coronary angiography was conducted using the Standard Judkins technique, via either femoral or radial access. The PCI procedures were carried out following international guidelines, with Iohexol used as the contrast agent. Each patient was administered a loading dose of acetylsalicylic acid (300 mg) along with a P2Y12 receptor inhibitor, which included a loading dose of either 600 mg of clopidogrel, 60 mg of prasugrel, or 180 mg of ticagrelor before the procedure. Following the administration of a 70 IU/kg bolus of unfractionated heparin during the PCI procedure, the operator had the option to use tirofiban to block the platelet glycoprotein IIb/IIIa receptor.

Statistical Analysis

Statistical analyses were conducted using IBM SPSS for Windows version 23.0 (SPSS Inc., Chicago, IL, USA). The distribution patterns of variables were assessed with the Kolmogorov-Smirnov test. Categorical variables were presented as frequencies and percentages, while continuous variables were expressed as mean±standard deviation or median with interquartile range (IQR), depending on the data distribution. The Mann-Whitney U test was used to compare nonparametric continuous variables, and categorical variables were evaluated using Pearson's Chi-square test. Logistic regression analysis was carried out to determine factors independently correlated with CIN. The receiver operating characteristic (ROC) curve was also employed to determine the most accurate ELR cutoff for predicting CIN. A p-value under 0.05 was considered statistically significant.

RESULTS

This research encompassed 440 STEMI patients (average age: 59.2±13.0, 73% male) who underwent primary PCI, with 58 (13.1%) of them developing CIN. Based on the occurrence of CIN, the population was separated into two categories, with their demographic and clinical characteristics are illustrated and contrasted in **Table 1**. The CIN (+) group was older than the other group (70.9±12.5 vs. 57.4±12.2, p<0.001). Notable distinctions were not identified among the groups concerning gender, frequency of hyperlipidemia, history of CAD, medicines taken before to and during hospitalization, blood pressure at the time of admission, or body-mass index. Patients in the CIN (+) group exhibited a greater incidence

Table 1. Comparison of demographic, clinical and laboratory characteristics of patients

Variables	CIN (-) (n:382)	CIN (+) (n:58)	p
Age (years)	57.4±12.2	70.9±12.5	<0.001
Gender (male), n (%)	284 (74.3)	37 (63.8)	0.092
Diabetes mellitus, n (%)	108 (28.3)	24 (41.4)	0.042
Hypertension, n (%)	133 (34.8)	35 (60.3)	<0.001
Hyperlipidemia, n (%)	115 (30.1)	15 (25.9)	0.509
Current smoker, n (%)	211 (55.2)	10 (17.2)	<0.001
Body-mass index (kg/m²)	28.0±4.2	27.7±4.9	0.243
History of coronary artery disease, n (%)	56 (14.7)	10 (17.2)	0.608
Systolic blood pressure (mmHg) (at admission)	128.6±23.2	128.0±30.1	0.781
Diastolic blood pressure (mmHg) (at admission)	78.1±13.7	77.4±17.1	0.929
LVEF (%)	47.9±9.5	40.0±10.2	<0.001
Pre-hospital medications			
Statin, n (%)	36 (9.4)	7 (12.1)	0.527
ACE-İ/ARB, n (%)	86 (22.5)	18 (31.0)	0.155
Beta blocker, n (%)	58 (15.2)	12 (20.7)	0.285
Oral antidiabetic, n (%)	82 (21.5)	17 (29.3)	0.183
In-hospital medications			
Statin, n (%)	356 (93.2)	53 (91.4)	0.615
ACE-I/ARB, n (%)	298 (78.0)	40 (69.0)	0.128
Beta blocker, n (%)	339 (88.7)	50 (86.2)	0.574
MRA, n (%)	32 (8.4)	4 (6.9)	0.702
Glucose (mg/dl)	126 (107-170)	135 (108-234)	0.144
Creatinine (mg/dl) (at admission)	1.05±0.25	1.38±0.43	<0.001
eGFR (mL/min/1.73m²) (at admission)	76±19	51±20	<0.001
Triglyceride (mg/dl)	139 (95-199)	141 (80-201)	0.660
Total cholesterol (mg/dl)	195±45	187±51	0.149
HDL-cholesterol (mg/dl)	40±8	40±11	0.741
LDL-cholesterol (mg/dl)	125±40	121±40	0.517
C-reactive protein (mg/L)	0.56 (0.29-0.97)	0.83 (0.43-1.44)	0.005
WBC (x10³/μL)	10.4±2.9	10.5±3.4	0.810
Neutrophil (x10³/μL)	6.4±2.4	6.9±2.5	0.135
Lymphocyte (x10³/μL)	3.0±1.2	2.4±1.3	<0.001
Eosinophil (x10³/μL)	0.19±0.12	0.31±0.18	<0.001
Hemoglobin (g/dl)	14.4±1.6	13.0±2.4	<0.001
Platelet (x10³/μL)	230 (194-268)	245 (196-326)	0.068
ELR	0.069±0.037	0.134±0.063	<0.001

Data are shown as mean±standard deviation, median (25th and 75th interquartile range), and number (%), CIN: Contrast-induced nephropathy, ACE-I: Angiotensin converting enzyme inhibitor, ARB: Angiotensin receptor blocker, ASA: Acetylsalicylic acid, eGFR: Estimated glomerular filtration rate, ELR: Eosinophil/lymphocyte ratio, HDL: High-density lipoprotein, LDL: Low-density lipoprotein, LVEF: Left ventricular ejection fraction, MRA: Mineral corticosteroid receptor antagonists, OAD: Oral antidiabetic, WBC: White blood cell

of diabetes and hypertension, along with reduced LVEF and smoking rates compared to those in the CIN (-) group. The groups exhibited no notable differences regarding admission serum glucose, white blood cell (WBC) count, neutrophil and platelet levels, or serum lipid profile. In the CIN (+) group, the admission serum creatinine level, C-reactive protein (CRP), and eosinophil count were significantly higher, while lymphocyte count, eGFR, and hemoglobin levels were lower. Moreover, the ELR was notably elevated in the CIN (+) group when compared to the CIN (-) group (0.134±0.063 vs. 0.069±0.037; p<0.001) (**Figure 1**).

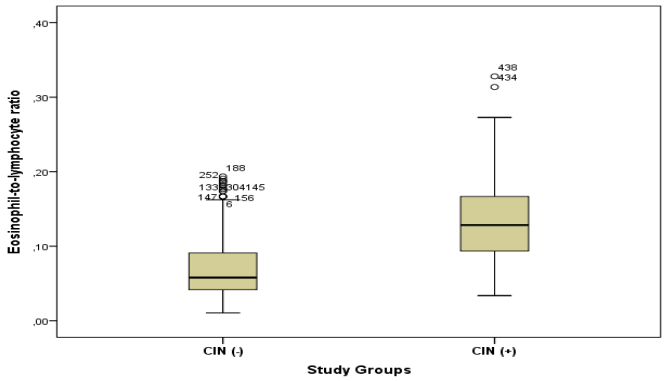

Figure 1. Comparison of ELR of the study groups
ELR: Eosinophil-to-lymphocyte ratio

The angiographic characteristics of the subjects are presented in **Table 2**. We detected statistically no remarkable differences in terms of the culprit coronary artery, total contrast volume, stent length, and stent diameter between the two groups. While the incidence of multivessel disease was slightly greater in the CIN (+) cohort compared to the CIN (-) cohort, this disparity did not achieve statistical significance (62.1% vs. 50.5%; p=0.101).

The multivariate logistic regression analysis revealed that elevated ELR levels (OR:1.251, 95% Confidence Interval (CI):1.149-1.362; p<0.001) were an independent predictor of CIN. Furthermore, lower eGFR (OR:0.961, 95%CI: 0.937-

Table 2. Angiographic features of patients according to the presence or absence of contrast nephropathy

Variables	CIN (-) (n:382)	CIN (+) (n:58)	p
Culprit coronary artery, n (%)			
LAD	166 (43.5)	28 (48.3)	0.491
LCX	70 (18.3)	14 (24.1)	0.294
RCA	146 (38.2)	16 (27.6)	0.118
Total amount of contrast media (ml)	167±71	170±72	0.822
Multivessel disease, n (%)	193 (50.5)	36 (62.1)	0.101
Stent length (mm)	25.0±11.9	26.5±10.8	0.127
Stent diameter (mm)	3.2±0.4	3.0±0.3	0.109

Data are shown as mean±standard deviation, CIN: Contrast-induced nephropathy, LAD: Left anterior descending artery, LCX: Left circumflex artery, RCA: Right coronary artery

0.985; p=0.002), higher CRP levels (OR:1.555, 95%CI: 1.164-2.077; p=0.003), and reduced LVEF (OR:0.943, 95%CI: 0.904-0.984; p=0.006) were also found to be independent predictors of CIN (**Table 3**).

Based on the ROC curve analysis, the best ELR threshold level for predicting CIN development was 0.093, demonstrating a sensitivity of 75.9% and specificity of 79.1% [area under curve (AUC): 0.836; 95% CI: 0.783–0.889; p<0.001] (**Figure 2**).

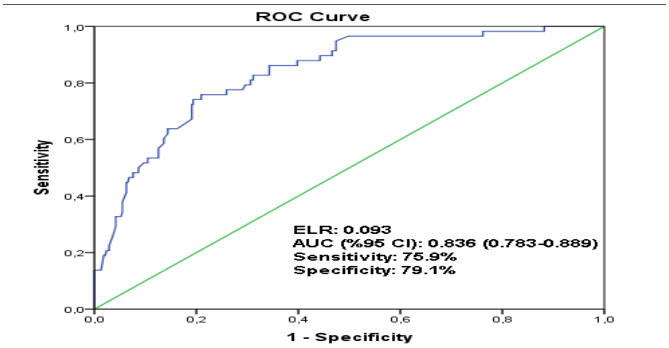

Figure 2. ROC curves analysis of CIN prediction using ELR
ROC: Receiver Operating Charasteristics, CIN: Contrast-induced nephropathy, ELR: Eosinophil-to-lymphocyte ratio

DISCUSSION

The findings of this study indicated that ELR independently predicts the occurrence of CIN in STEMI patients undergoing primary PCI. Furthermore, eGFR, CRP, and LVEF were identified as factors linked to the onset of CIN.

Earlier studies have pinpointed various risk elements contributing to the onset of CIN. However, due to the nephrotoxic effects of contrast agents can vary significantly from one individual to another, predicting CIN can sometimes be challenging in clinical settings. In STEMI patients, CIN occurs at higher rates compared to elective PCI patients due to the more complex nature of primary PCI, increased contrast usage, hemodynamic instability, and the limited applicability of preventive measures.[13] In our research, the rate of CIN among STEMI patients was found to be 13.1%.

The pathophysiology of CIN involves complex mechanisms that are likely influenced by multiple factors. Previous research has indicated that potential causes include direct damage to tubular epithelial cells, constriction of blood vessels within the kidneys, hypoxia in the medulla, endothelial dysfunction, and reactive oxygen species.[5,14] Contrast agents lead to prolonged vasoconstriction of renal blood vessels by directly affecting vascular smooth muscle cells and triggering the release of inflammatory mediators at the cellular level. Additionally, they reduce water reabsorption, leading to an increase in interstitial pressure. This leads to a reduction in eGFR and exacerbation of medullary hypoxia. Moreover, contrast agents increase blood viscosity, which raises resistance to blood flow by reducing the deformability of red blood cells. The formation of intravascular sludge can result in localized ischemia and trigger the production of reactive oxygen products. This process contributes to cellular damage within the tubules, which is associated with the progression of acute kidney injury. On the other hand, contrast agents activate inflammatory pathways, which can result in acute kidney injury.[15] Studies in experimental animals supporting this mechanism have illustrated that inflammatory cytokines, such as TNF-α, IL-1, and IL-6, rise markedly right following exposure to contrast agents, resulting in acute tubular damage.[16] A prospective study conducted by Kwasa et al.[5] showed that individuals with CIN exhibited elevated CRP levels in contrast to those without. These findings indicate that inflammation is considered a major contributor to the emergence of CIN.[5,14] High eosinophil levels and low lymphocyte levels reflect systemic inflammation. ELR is an inflammatory marker that considers both eosinophil and lymphocyte counts. Our study found ELR and CRP, inflammatory markers, as independent predictors of CIN.

Numerous indices based on complete blood counts have been created recently to examine the connection between inflammation and cardiovascular disorders. Eosinophils are multipurpose white blood cells that contribute to the emergence of several inflammatory diseases, including cancer, allergic disorders, and systemic and local infections.[17] Eosinophils perform a vital role, particularly in vascular inflammation and thrombosis.[6] A study conducted by Colon et al.[18] demonstrated that eosinophils accumulating in the renal interstitium could trigger renal fibrosis by modulating the inflammatory response and eosinophil peroxidase activity. A study conducted by Kielar et al.[19] demonstrated that individuals with CKD and high eosinophil levels encountered

Table 3. Logistic regression analysis of potential predictors for contrast nephropathy

Variables	Univariate analysis		Multivariate analysis	
	OR (95% CI)	p	OR (95% CI)	p
Age	1.086 (1.060-1.112)	<0.001		
Diabetes mellitus	1.791 (1.015-3.160)	0.044		
Hypertension	2.849 (1.617-5.021)	<0.001		
C-reactive protein	1.363 (1.161-1.600)	<0.001	1.555 (1.164-2.077)	0.003
ELR	1.295 (1.213-1.382)	<0.001	1.251 (1.149-1.362)	<0.001
Current smoker	0.169 (0.083-0.344)	<0.001		
Hemoglobin	0.690 (0.598-0.797)	<0.001		
LVEF	0.923 (0.896-0.952)	<0.001	0.943 (0.904-0.984)	0.006
eGFR	0.940 (0.924-0.955)	<0.001	0.961 (0.937-0.985)	0.002
Eosinophil	1.631 (1.363-1.951)	<0.001		
Lymphocyte	0.628 (0.474-0.833)	0.001		

OR: Odds ratio, CI: Confidence interval, eGFR: Estimated glomerular filtration rate, ELR: Eosinophil/lymphocyte ratio, LVEF: Left ventricular ejection fraction

a heightened likelihood of progressing to end-stage renal disease. In contrast, lymphocytes are essential in regulating inflammatory responses. Lower lymphocyte counts, associated with increased inflammation and lymphocyte apoptosis, make STEMI patients more susceptible to endothelial dysfunction, platelet activation, and thrombosis.[20] Low lymphocyte levels (lymphopenia) have been linked to heightened increased inflammatory activity and adverse cardiovascular events.[21]

ELR is a new inflammatory biomarker based on serum eosinophil and lymphocyte counts. Recent research has highlighted a notable connection between ELR and adverse outcomes in cancer patients.[22] Furthermore, the predictive value of this index has been investigated across various cardiovascular diseases, including CAD, heart failure, isolated coronary artery ectasia, microvascular angina, and slow coronary flow.[8,9,23-25] However, the relationship between ELR and CIN has not been previously explored. Given that an elevated ELR is strongly linked to inflammatory processes and atherosclerosis, our research sought to determine whether ELR correlates with CIN in STEMI patients. The findings from this study suggest that ELR serves as an independent predictor for CIN occurrence following the PCI procedure.

As the occurrence of CIN results in considerable morbidity and mortality, it is essential to identify useful biomarkers to decrease the rate of CIN through preventive strategies. ELR, a practical and reliable indicator that can be straightforwardly derived from complete blood analysis, could serve as a predictor of CIN. Preventive strategies, such as prophylactic hydration and/or using low-dose iso-osmolar contrast agents, can be easily applied to subjects with elevated ELR levels, regardless of kidney function.

Limitations

Our investigation has some constraints. To begin with, the analysis is retrospective, conducted at a single center, and possesses a restricted sample size. Consequently, the results need to be further validated and confirmed in larger populations. Second, only a baseline ELR value was determined in the study; temporal measurements of ELR values could provide additional data. Finally, because changes in serum creatinine may extend beyond the 72-hour time frame due to delayed effects of the contrast agent, kidney function deterioration may have occurred after hospital discharge in some subjects; therefore, the correct rate of CIN may have been overlooked.

CONCLUSION

Inflammation has been identified as a crucial factor in the progression of CIN, with various inflammation-related biomarkers recognized as being associated with its occurrence. This study emphasizes the correlation between the emergence of CIN in individuals with STEMI and ELR, a relatively new inflammatory marker. This index could assist clinicians in identifying high-risk patients who would benefit from preventive strategies prior to and following the primary PCI procedure.

ETHICAL DECLARATIONS

Ethics Committee Approval

The study was carried out with the permission of Yozgat Bozok University Non-interventional Clinical Researches Ethics Committee (Date: 18.12.2024, Decision No: 2024-GOKAEK-2415_2024.12.18_251).

Informed Consent

Because the study was designed retrospectively, no written informed consent form was obtained from patients.

Referee Evaluation Process

Externally peer-reviewed.

Conflict of Interest Statement

The authors have no conflicts of interest to declare.

Financial Disclosure

The authors declared that this study has received no financial support.

Author Contributions

All of the authors declare that they have all participated in the design, execution, and analysis of the paper, and that they have approved the final version.

REFERENCES

1. Elserafy AS, Okasha N, Hegazy T. Prevention of contrast induced nephropathy by ischemic preconditioning in patients undergoing percutaneous coronary angiography. *Egypt Heart J.* 2018;70(2):107-111. doi:10.1016/j.ehj.2017.12.004
2. Moro AB, Strauch JGN, Groto AD, Toregeani JF. Creatinine level variation in patients subjected to contrast-enhanced tomography: a meta-analysis. *J Vasc Bras.* 2021;20:e20200161. doi:10.1590/1677-5449.200161
3. Moitinho MS, Santos ES, Caixeta AM, Belasco AGDS, Barbosa DA, Fonseca CDD. Contrast-induced nephropathy in patients submitted to percutaneous coronary intervention: an integrative review. *Rev Bras Enferm.* 2020;73(suppl 5):e20200190. doi:10.1590/0034-7167-2020-0190
4. Brar SS, Aharonian V, Mansukhani P, et al. Haemodynamic-guided fluid administration for the prevention of contrast-induced acute kidney injury: the POSEIDON randomised controlled trial. *Lancet.* 2014;383(9931):1814-1823. doi:10.1016/S0140-6736(14)60689-9
5. Kwasa EA, Vinayak S, Armstrong R. The role of inflammation in contrast-induced nephropathy. *Br J Radiol.* 2014;87(1041):20130738. doi:10.1259/bjr.20130738
6. Wang JG, Mahmud SA, Thompson JA, Geng JG, Key NS, Slungaard A. The principal eosinophil peroxidase product, HOSCN, is a uniquely potent phagocyte oxidant inducer of endothelial cell tissue factor activity: a potential mechanism for thrombosis in eosinophilic inflammatory states. *Blood.* 2006;107(2):558-565. doi:10.1182/blood-2005-05-2152
7. Horne BD, Anderson JL, John JM, et al. Which white blood cell subtypes predict increased cardiovascular risk? *J Am Coll Cardiol.* 2005;45(10):1638-1643. doi:10.1016/j.jacc.2005.02.054
8. Kounis NG, Soufras GD, Tsigkas G, Hahalis G. White blood cell counts, leukocyte ratios, and eosinophils as inflammatory markers in patients with coronary artery disease. *Clin Appl Thromb Hemost.* 2015;21(2):139-143. doi:10.1177/1076029614531449
9. Vural A, Aydın E. The predictive value of eosinophil indices for major cardiovascular events in patients with acute decompensated HFrEF. *Medicina (Kaunas).* 2022;58(10):1455. doi:10.3390/medicina58101455
10. Vavalle JP, van Diepen S, Clare RM, et al. Renal failure in patients with ST-segment elevation acute myocardial infarction treated with primary percutaneous coronary intervention: predictors, clinical and angiographic features, and outcomes. *Am Heart J.* 2016;173:57-66. doi:10.1016/j.ahj.2015.12.001

11. Ibanez B, James S, Agewall S, et al. 2017 ESC Guidelines for the management of acute myocardial infarction in patients presenting with ST-segment elevation: the task force for the management of acute myocardial infarction in patients presenting with ST-segment elevation of the European Society of Cardiology (ESC). *Eur Heart J*. 2018;39(2): 119-177. doi:10.1093/eurheartj/ehx393
12. Levey AS, Stevens LA, Schmid CH, et al. A new equation to estimate glomerular filtration rate [published correction appears in Ann Intern Med. 2011;155(6):408]. *Ann Intern Med*. 2009;150(9):604-612. doi:10.7326/0003-4819-150-9-200905050-00006
13. Senoo T, Motohiro M, Kamihata H, et al. Contrast-induced nephropathy in patients undergoing emergency percutaneous coronary intervention for acute coronary syndrome. *Am J Cardiol*. 2010;105(5):624-628. doi:10.1016/j.amjcard.2009.10.044
14. Butt K, D'Souza J, Yuan C, et al. Correlation of the neutrophil-to-lymphocyte ratio (NLR) and platelet-to-lymphocyte ratio (PLR) with contrast-induced nephropathy in patients with acute coronary syndrome undergoing percutaneous coronary interventions. *Cureus*. 2020;12(12):e11879. doi:10.7759/cureus.11879
15. Zhang F, Lu Z, Wang F. Advances in the pathogenesis and prevention of contrast-induced nephropathy. *Life Sci*. 2020;259:118379. doi:10.1016/j.lfs.2020.118379
16. Lu Z, Cheng D, Yin J, et al. Antithrombin III protects against contrast-induced nephropathy. *EBioMedicine*. 2017;17:101-107. doi:10.1016/j.ebiom.2017.02.009
17. Ramirez GA, Yacoub MR, Ripa M, et al. Eosinophils from physiology to disease: a comprehensive review. *Biomed Res Int*. 2018;2018:9095275. doi:10.1155/2018/9095275
18. Colon S, Luan H, Liu Y, Meyer C, Gewin L, Bhave G. Peroxidasin and eosinophil peroxidase, but not myeloperoxidase, contribute to renal fibrosis in the murine unilateral ureteral obstruction model. *Am J Physiol Renal Physiol*. 2019;316(2):F360-F371. doi:10.1152/ajprenal.00291.2018
19. Kielar D, Jones AM, Wang X, et al. Association between elevated blood eosinophils and chronic kidney disease progression: analyses of a large United States electronic health records database. *Int J Nephrol Renovasc Dis*. 2023;16:269-280. doi:10.2147/IJNRD.S431375
20. Widmer A, Linka AZ, Attenhofer Jost CH, et al. Mechanical complications after myocardial infarction reliably predicted using C-reactive protein levels and lymphocytopenia. *Cardiology*. 2003;99(1): 25-31. doi:10.1159/000068448
21. Núñez J, Miñana G, Bodí V, et al. Low lymphocyte count and cardiovascular diseases. *Curr Med Chem*. 2011;18(21):3226-3233. doi:10.2174/092986711796391633
22. Holub K, Biete A. New pre-treatment eosinophil-related ratios as prognostic biomarkers for survival outcomes in endometrial cancer. *BMC Cancer*. 2018;18(1):1280. doi:10.1186/s12885-018-5131-x
23. Yilmaz M, Kayançiçek H, Korkmaz H, et al. A new inflammatory marker: elevated eosinophil-tolymphocyte ratio associated with presence and severity of isolated coronary artery ectasia. *Cardiovasc J Afr*. 2020;31(5):227-235. doi:10.5830/CVJA-2019-049
24. Çetin ZG, Demirtaş B. Understanding the significance of the eosinophil to lymphocyte ratio in individuals with microvascular angina. *Bozok Tıp Derg*. 2023;13(3):73-80. doi:10.16919/bozoktip.1341814
25. Tosu AR, Kalyoncuoğlu M, Biter Hİ, et al. Association of eosinophil-to-lymphocyte ratio with coronary slow-flow phenomenon in patients undergoing coronary angiography. *Arch Med Sci Atheroscler Dis*. 2022; 7(1):e29-e35. doi:10.5114/amsad.2022.116662