INTERVENTIONAL CARDIOLOGY PERSPECTIVES

Inter Cardio Pers 2025;1(3):98-103

DOI: 10.4274/intercardiopers.2025.2025-9-3

ORIGINAL ARTICLE

Impact of Eosinophil Count on Short-Term Prognosis in Patients with Acute Pulmonary Embolism

Murat Gök[1], Furkan Karahan[1], Umutcan Vurucu[1], Alparslan Kurtul[2], Kenan Yalta[1]

[1]Department of Cardiology, Trakya University Faculty of Medicine, Edirne, Türkiye

[2]Department of Cardiology, Hatay Mustafa Kemal University Faculty of Medicine, Hatay, Türkiye

ABSTRACT

Background: Eosinophils, a type of inflammatory cell, have been implicated in thrombosis and proposed as a prognostic marker for various atherosclerotic cardiovascular diseases.

Aim: This study determine the value of an eosinophil count (EOSC) in predicting short-term prognosis in patients with acute pulmonary embolism (APE).

Study Design: Retrospective cohort study.

Methods: In this study, a total of 453 patients who were admitted with APE from September 2015 to July 2020 were retrospectively examined, and their admission complete blood cell counts were measured. The optimal cut-off point for EOSC was determined using receiver operating characteristic analysis. Patients were classified into the low EOSC (≤0.45) and high EOSC (>0.45) groups. Multivariate logistic regression analysis was performed to investigate the independent association between EOSC and early mortality (in-hospital and 30-day) outcomes in patients with APE.

Results: Throughout the hospitalization and 30-day follow-up, a total of 33 deaths (7.3%) occurred. Of the patients who survived, those who died were older and had significantly lower EOSC (p<0.001) but significantly higher white blood cell (WBC) (p<0.001) and platelet (p=0.025) counts. Multivariate regression analyses identified that decreased EOSC (≤0.45) [odds ratio (OR): 4.518; p=0.003], increased WBC count (OR: 1.135; p=0.012), and older age (OR: 1.048; p=0.029) were independently associated with short-term mortality.

Conclusion: A significant correlation was observed between low EOSC and elevated short-term mortality in patients with APE. Thus, blood EOSC can be used in daily clinical practice as a novel marker that correlates with the risk stratification of patients with APE.

Keywords: Eosinophil, mortality, inflammation, thrombus, acute pulmonary embolism

INTRODUCTION

Acute pulmonary embolism (APE) is regarded as the third most prevalent cause of mortality from cardiopulmonary disease, after acute myocardial infarction and stroke.[1,2] The incidence of APE is expected to increase further owing to the enhanced sensitivity of diagnostic imaging, population aging, and the rising prevalence of risk factors such as obesity and cancer linked to venous thromboembolism. Nevertheless, APE mortality rates remain a concern despite the advent of novel diagnostic and therapeutic procedures.[3] Consequently, the early identification of patients at elevated risk of mortality is critical. For this purpose, several risk classification systems have been formulated, including the simplified pulmonary embolism severity index (sPESI), and these indexes comprise several parameters such as blood pressure, echocardiographic evidence of right ventricular dysfunction (RVD), and computed tomography pulmonary angiography.[4] Nevertheless, the need for a reliable indicator that can be accurately and efficiently measured to predict adverse outcomes remains.

Cytokines are produced during inflammation and can trigger blood coagulation.[5] As our knowledge of inflammation and thrombogenesis associated with APE advances, multiple systemic inflammatory index models have been proposed to predict patient prognosis.[6] In this context, C-reactive protein (CRP),[7] D-dimer,[8] platelet count,[9] and white blood cell (WBC)[10] count have been identified as factors associated with APE mortality. These markers exhibit a close relationship between local inflammation and thrombosis within the pulmonary artery.[11] Eosinophils have also been implicated in various inflammatory responses[12] and in the pathogenesis of thrombosis,[13,14] suggesting a close link with APE occurrence. Previous studies have demonstrated that a low eosinophil count (EOSC) is significantly correlated with poor prognosis in various cardiovascular diseases.[15-21]

Address for Correspondence: Murat Gök MD, Department of Cardiology, Trakya University Faculty of Medicine, Edirne, Türkiye

E-mail: drmuratg@hotmail.com **ORCID ID:** orcid.org/0000-0003-3118-9064

Cite as: Gök M, Karahan F, Vurucu U, Kurtul A, Yalta K. Impact of eosinophil count on short-term prognosis in patients with acute pulmonary embolism. *Inter Cardio Pers.* 2025;1(3):98-103

Received: 26.09.2025

Accepted: 27.11.2025

Publication Date: 10.12.2025

However, the association between eosinophils and APE remains to be established. Therefore, this study aimed to examine EOSC's ability to predict the early prognosis (in-hospital or 30-day) of patients with APE.

METHODS

Study Design

This retrospective cohort study included patients with APE admitted between September 2015 and July 2020. Eligible participants were adults aged ≥18 years who presented with clinical signs suggestive of APE, exhibited computed tomography evidence of proximal filling defects involving at least one primary or lobar pulmonary artery, and demonstrated RVD on transthoracic echocardiography or computed tomography. All patients underwent computed tomography pulmonary angiography to confirm the diagnosis of APE. Exclusion criteria: (1) age <18 years; (2) missing EOSC data; (3) time of onset >14 days; (4) sepsis, malignancy, chronic inflammatory conditions, active infection, and use of immunosuppressive therapy at the time of diagnosis of APE.

This study was approved by the Non-Interventional Scientific Research Ethics Committee of Trakya University (approval number: 06/14, date: 01.04.2024).

Data Collection and Definitions

A comprehensive set of baseline demographic and health-related data was retrieved from the hospital's electronic database, including patient age, sex, heart rate, smoking history, presence of diabetes mellitus (DM), and systolic and diastolic blood pressure. The laboratory data collected at admission comprised hemoglobin, WBC count, platelet count, EOSC, serum creatinine, high-sensitivity CRP (hs-CRP), troponins, and D-dimer. All patients were followed up during their hospitalization. The primary endpoint was short-term mortality (in-hospital and 30-day).

Transthoracic echocardiography was performed at the time of hospitalization, and RVD and pulmonary artery systolic pressure were determined by experienced cardiologists.

Statistical Analysis

The distribution of continuous variables was initially evaluated for normality using the Shapiro–Wilk test, supported by visual inspection of Q–Q plots. Variables meeting normality assumptions were summarized as means with standard deviations and compared using the independent-samples t-test. Non-normally distributed variables were described as medians with interquartile ranges and assessed using the Mann–Whitney U test. Categorical variables were presented as counts with percentages and compared using either Pearson's chi-square test or Fisher's exact test, as appropriate. The area under the receiver operating characteristic (ROC) curve (AUC) was calculated to determine the optimal cut-off value for EOSC, after which patients were stratified into two groups based on this threshold. Independent prognostic factors for in-hospital mortality were examined using logistic regression. Variables with $p<0.05$ in univariate analyses were entered into multivariate models. The results were expressed as odds ratios (ORs) with 95% confidence intervals. Analyses were conducted using SPSS Statistics version 21.0 (IBM Corp., Armonk, NY, USA). Statistical significance was defined as $p<0.05$.

RESULTS

Patient Characteristics

The baseline characteristics of the study participants are summarized in Table 1. This study involved 453 patients with APE. Relative to survivors, patients who died during hospitalization and 30-day follow-up had significantly lower EOSC ($p<0.001$) but significantly higher WBC ($p<0.001$) and platelet ($p=0.025$) counts (Figure 1). A comparative analysis of the two groups revealed that those who died early were older than those who survived. In terms of comorbidities and risk factors, the non-survivor patients had a higher incidence of DM ($p=0.014$) and RVD ($p=0.001$) and a lower incidence of active smoking ($p=0.014$). Patients who met the primary endpoint also had higher systolic pulmonary artery pressure (SPAP) and hs-CRP levels but lower arterial oxygen saturation (SaO_2). However, the other parameters did not differ significantly.

ROC curve analysis showed that admission EOSC had the best predictive value for short-term mortality in the overall APE population, with a sensitivity of 79.5% and a specificity of 72.7% (AUC=0.809, 95% CI: 0.743–0.875; $p<0.001$). The optimal cut-off value for the admission EOSC to identify short-term mortality risk in patients with APE was 0.45 (Figure 2). Based on the optimal cut-off point for EOSC determined using the ROC curve, patients were classified into the low (EOSC ≤0.45) and high (EOSC >0.45) EOSC groups. Compared with patients exhibiting high EOSC, those with low EOSC were older and had significantly higher WBC and D-dimer levels but lower SaO_2 and systolic blood pressure and less active smoking. In addition, hs-CRP, troponin values, and RVD prevalence were higher in the high EOSC group, and the difference was borderline significant ($p=0.051$, 0.067, and 0.066, respectively) (Table 2).

Relationship between EOSC and mortality

Logistic regression analysis was performed to identify independent predictors of in-hospital mortality. A univariate logistic regression found the following as significant risk factors for early mortality: age, DM, active smoking, SaO_2, EOSC ≤0.45, SPAP, RVD, WBC count, platelet count, and hs-CRP. When these confounders were controlled for in multivariate logistic regression, low EOSC (≤0.45) (OR: 4.518; 95% CI: 1.693–12.058; $p=0.003$), older age (OR: 1.048; 95% CI: 1.005-1.092; $p=0.029$), and increased WBC count (OR: 1.135; 95% CI: 1.028 to 1.253; $p=0.012$) were significantly associated with short-term mortality (Table 3).

DISCUSSION

To the best of our knowledge, this study is the first to assess the predictive value of EOSC in APE mortality. The following findings were observed: (1) relative to surviving patients, those who died had lower EOSC levels and higher WBC and platelet counts, and (2) despite adjustment for potential confounders, low EOSC (≤0.45) was an independent predictor of early mortality in patients with APE.

APE is a prevalent cardiovascular disorder that has been associated with elevated morbidity and mortality rates.[1] Consequently, prompt and precise risk assessment at initial diagnosis is necessary to inform treatment decisions and mitigate mortality. APE-induced thrombo-

Table 1. Baseline characteristics of patients with and without in-hospital and 30-day mortality

Variables	Survivor (n=420)	Non-survivor (n=33)	p value
Age, years	63±16	73±9	<0.001
Male gender, n (%)	185 (44%)	18 (54%)	0.243
Diabetes mellitus, n (%)	43 (10%)	8 (24%)	0.014
Active smoker, n (%)	153 (36%)	5 (15%)	0.014
White blood cell count (x109/L)	10.3±3.7	15.2±5.3	<0.001
Platelet count (x109/L)	252±72	282±98	0.025
Hemoglobin (g/dL)	12.4±2.1	12.1±2.1	0.294
Total cholesterol (mg/dL)	173±41	157±40	0.068
Low density lipoprotein cholesterol (mg/dL)	107±36	100±33	0.378
High density lipoprotein cholesterol (mg/dL)	36±12	33±12	0.223
Triglyceride (mg/dL)	118 (89-151)	111 (88-147)	0.784
Creatinine (mg/dL)	0.89 (0.75-1.2)	1.07 (0.92-1.36)	0.105
Eosinophil count (x109/L)	1.6 (0.6-2.8)	0.3 (0.03-0.5)	<0.001
D-dimer (mg/L)	4514(2624-7084)	5343 (2514-8725)	0.199
Peak troponin value (ng/mL)	0.8 (0.07-8.3)	2.4 (0.1-4.6)	0.519
High-sensitivity C-reactive protein (mg/L)	22.7 (7.0-79.8)	46.6 (22.3-100)	0.017
Systolic blood pressure (mmHg)	116±16	111±25	0.106
Diastolic blood pressure (mmHg)	72±12	68±13	0.074
Arterial oxygen saturation (%)	89.2±8.8	81.2±11.6	<0.001
Systolic pulmonary artery pressure (mmHg)	49±16	58±13	0.005
Thrombolytic therapy, n (%)	157 (37%)	14 (42%)	0.565
Right ventricular dysfunction, n (%)	258 (61%)	30 (90%)	0.001

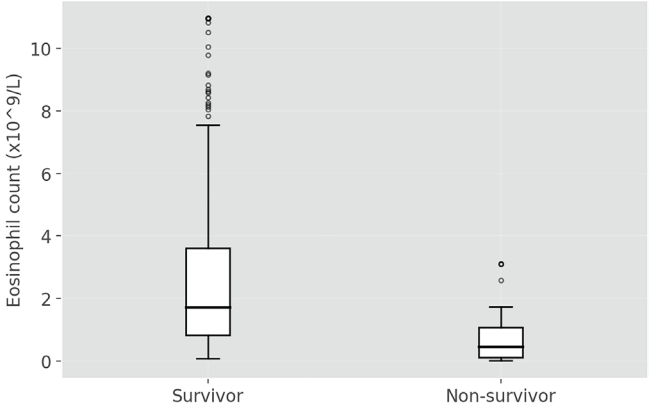

Figure 1. Comparison of eosinophil ratios between the two groups

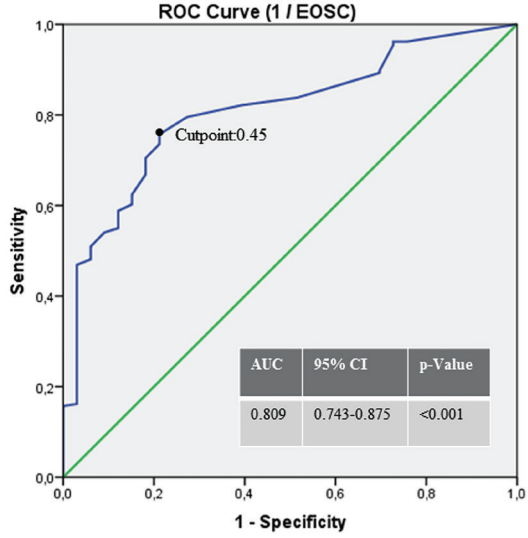

Figure 2. ROC analyses table; cut point values and ROC curves

ROC: Receiver operating characteristic, AUC: Area under the curve, CI: Confidence interval, EOSC: Eosinophil count

inflammation plays a vital role in the patient's prognosis. In this regard, several prognostic biomarkers are independent predictors for adverse outcomes in patients with APE.[6,7] Eosinophils are also involved in various inflammatory responses and thrombotic processes.[8-10] Toor et al.[11] examined the prognostic significance of EOSC in patients with coronary artery disease, reporting that elevated preprocedural EOSC levels were associated with enhanced outcomes within the initial 6 months following percutaneous coronary intervention. An earlier study has shown the relevance of eosinopenia and unfavorable outcomes in patients with acute ischemic stroke.[12] Similarly, another retrospective study documented that eosinopenia is a predictor of unfavorable long-term cardiac outcomes in patients with ST-segment elevation myocardial infarction.[13] In addition, a Turkish study of 1,909 patients

Table 2. Baseline characteristics of patients stratified by the optimal cutoff point of EOSC

Variables	High EOSC (>0.45) (n=343)	Low EOSC (≤0.45) (n=110)	p value
Age, years	61±17	69±12	<0.001
Male gender, n (%)	153 (44.6%)	50 (45.5%)	0.912
Diabetes mellitus, n (%)	34 (9.9%)	17 (15.5%)	0.120
Active smoker, n (%)	131 (38.2%)	27 (24.5%)	0.011
White blood cell count (x109/L)	9.8±3.2	13.3±5.2	<0.001
Platelet count (x109/L)	255±75	250±74	0.569
Hemoglobin (g/dL)	12.5±2.2	12.2±2.01	0.218
Total cholesterol (mg/dL)	174±40	166±44	0.102
Low density lipoprotein cholesterol (mg/dL)	108±35	102±38	0.162
High density lipoprotein cholesterol (mg/dL)	36±12	34±13	0.092
Triglyceride (mg/dL)	117 (88-156)	118 (89-136)	0.810
Creatinine (mg/dL)	0.96 (0.88-1.25)	1.13 (0.95-1.34)	0.122
Eosinophil count (x109/L)	2.0 (1.1-3.3)	0.18 (0.08-0.2)	<0.001
D-dimer (mg/L)	4211(2437-6382)	5374 (2807-9938)	0.001
Peak troponin value (ng/mL)	0.8 (0.5-6.0)	2.4 (0.2-18)	0.067
High-sensitivity C-reactive protein (mg/L)	22 (7-75)	41 (7-93)	0.051
Systolic blood pressure (mmHg)	117±16	112±16	0.010
Diastolic blood pressure (mmHg)	72±13	71±11	0.611
Arterial oxygen saturation (%)	90±8	84±11	<0.001
Systolic pulmonary artery pressure (mmHg)	50±17	51±14	0.674
Right ventricular dysfunction, n (%)	210 (61.2%)	78 (70.9%)	0.690

EOSC: Eosinophil count

Table 3. Independent predictors for in-hospital and 30-day acute pulmonary embolism mortality

Variables	Multivariate analysis		
	OR	95% CI	p value
Age	1.048	1.005-1.092	0.029
Diabetes mellitus	1.989	0.663-5.965	0.220
Active smoker	0.553	0.184-1.665	0.292
White blood cell count	1.135	1.028-1.253	0.012
Platelet count	3.538	0.530-23.618	0.192
High sensitivity C-reactive protein	1.006	1.000-1.012	0.057
Systolic pulmonary artery pressure	1.010	0.980-1.041	0.531
Arterial oxygen saturation	0.992	0.949-1.036	0.706
Right ventricular dysfunction	3.350	0.810-13.849	0.095
EOSC ≤0.45	4.518	1.693-12.058	0.003

OR: Odds ratio, CI: Confidence interval, EOSC: Eosinophil count

with acute myocardial infarction found that the rates of in-hospital mortality and major adverse cardiac events were significantly higher among patients with low blood EOSCs than among those with high EOSCs.[14] Furthermore, a study involving 5,287 patients who underwent coronary angiography revealed that patients with myocardial infarction had significantly lower blood EOSCs than those without coronary artery disease.[15] Sincer et al.[16] detected an inverse relationship between blood EOSCs and the severity of acute coronary syndrome subgroups in elderly patients. A more recent study revealed a significant correlation between low eosinophil percentage and elevated mortality rates in patients with acute type A aortic dissection.[17] Furthermore, blood EOSC was identified to be associated with the calcification scores of various arterial segments, ranging from the coronary to the iliac arteries.[18] No published studies have evaluated the link between eosinophils and APE. To our knowledge, this is the first study to demonstrate a correlation between low EOSCs and short-term (in-hospital and 30-day) APE mortality. Reduced circulating eosinophils were associated with a 4.5-fold increase in early mortality. Consequently, EOSC appears to be a more reliable predictor of adverse outcomes in patients with APA as it indicates both inflammation and thrombosis. This ROC cut-off value can be used to extend more careful and intensive care follow-ups in patients presenting with APE and an EOSC of <0.45. Its relationship with the need for thrombolysis can also be examined in future studies.

The blood EOSC can decrease for various reasons. Eosinophilia is often associated with allergic diseases, helminthic infections, eosinophilic

granulomatosis with polyarteritis (Churg–Strauss syndrome), Hodgkin lymphoma, and hypereosinophilic syndrome.[19] Certain pharmacological agents (beta-lactam antibiotics, nitrofurantoin, antiepileptics, and non-steroidal anti-inflammatory drugs) can cause drug-induced eosinophilia via immune-mediated mechanisms.[20] In contrast, glucocorticoids induce eosinopenia by suppressing the release of eosinophils from the bone marrow and increasing the redistribution of circulating cells to tissues.[21] In addition, hypercortisolemic or inflammatory conditions such as acute infections, severe stress, Cushing's syndrome, and sepsis are characterized by a marked decrease in eosinophil levels.[22] Therefore, quantitative assessment of EOSCs holds clinical value as a crucial biological marker in the diagnosis of underlying immunological, infectious, or pharmacological processes.

APE, which is related to chest pain and dyspnea, can trigger acute stress responses that stimulate the release of glucocorticoids, such as cortisol,[23] which can lead to eosinopenia via apoptosis.[24] Furthermore, eosinophils are proinflammatory cells that release large quantities of cytokines, growth factors, and chemokines, thereby enhancing inflammatory reactions.[25] Our data showed a negative correlation between EOSC and WBC count and hs-CRP levels. This observation suggests that a severe inflammatory reaction occurs when EOSC decreases. Multivariate regression analysis indicated that reduced EOSC, together with older age and increased WBC, were independent predictors of APE mortality, even after adjusting for all possible confounding factors. The presence of cytokines and chemokines at the site of a thrombus may lead to an accumulation of eosinophils. This buildup could potentially reduce the number of circulating eosinophils.[26] Another study by Riegger et al.[27] showed that eosinophils are present in all patients with stent thrombosis. Moreover, the presence of aggregated eosinophils in the pulmonary artery may contribute to the development and progression of APE by affecting the inflammatory response and thrombosis. Eosinophils can produce tissue factors and a procoagulant phospholipid surface. These factors activate the prothrombinase complex, generating thrombin and promoting fibrin formation.[28] Furthermore, eosinophils interact with platelets at the thrombus site, resulting in mutual activation. Eosinophils migrate into the thrombi and activate platelets, leading to platelet activation by eosinophils. Therefore, activated platelets may contribute to thrombus formation.[29] In the light of the possible mechanisms mentioned above, we suggest that greater inflammatory response and thrombus burden may explain the more serious clinical condition and, ultimately, increased mortality in patients with APE.

Study Limitations

This study has several potential limitations that must be acknowledged. First, owing to the relatively small sample size and single-center, retrospective nature of the study, some biases are inevitable. Other factors that could cause low or high EOSC, such as asthma, allergic diseases, and medication use, were not considered in this study. Moreover, the relationship between the sPESI index and EOSC in patients with APE was not examined in this study and warrants further investigation. Second, although elevated EOSCs were confirmed at admission in patients with APE, the mechanisms by which eosinophils contribute to the pathogenesis of APE remain undetermined. Thus, further studies are warranted to elucidate the role of eosinophils in APE development.

CONCLUSION

Our data demonstrated that decreased EOSC is a rapid, simple, and inexpensive tool for predicting early prognosis and is significantly associated with increased short-term mortality in patients with APE. Therefore, in daily clinical practice, blood EOSC can be used as a novel marker for risk classification in patients with APE. This finding indicates that eosinophils play a previously unreported dynamic role in the clinical course, and further studies should investigate whether EOSCs can serve as a guide for APE diagnosis and treatment.

Ethics Committee Approval: This study was approved by the Non-Interventional Scientific Research Ethics Committee of Trakya University (approval number: 06/14, date: 01.04.2024).

Informed Consent: Retrospective study.

Authorship Contributions: Concept: M.G., F.K., A.K., Design: M.G., F.K., A.K., Data Collection or Processing: M.G., F.K., U.V., A.K., K.Y., Analysis or Interpretation: M.G., F.K., Literature Search: U.V., A.K., K.Y., Writing: M.G., U.V., A.K., K.Y.

Conflict of Interest: No conflict of interest was declared by the authors.

Financial Disclosure: The authors declared that this study received no financial support.

REFERENCES

1. Goldhaber SZ, Visani L, De Rosa M. Acute pulmonary embolism: clinical outcomes in the International Cooperative Pulmonary Embolism Registry (ICOPER). *Lancet.* 1999;353:1386-1389.
2. Heit JA. The epidemiology of venous thromboembolism in the community. *Arterioscler Thromb Vasc Biol.* 2008;28:370-372.
3. Konstantinides SV, Meyer G, Becattini C, et al; The task force for the diagnosis and management of acute pulmonary embolism of the European Society of Cardiology (ESC). 2019 ESC Guidelines for the diagnosis and management of acute pulmonary embolism developed in collaboration with the European Respiratory Society (ERS): The Task Force for the diagnosis and management of acute pulmonary embolism of the European Society of Cardiology (ESC). *Eur Respir J.* 2019;54:1901647.
4. Kucher N, Goldhaber SZ. Risk stratification of acute pulmonary embolism. *Semin Thromb Hemost.* 2006;32:838-847.
5. Hess R, Wujak L, Hesse C, et al. Coagulation factor XII regulates inflammatory responses in human lungs. *Thromb Haemost.* 2017;117:1896-1907.
6. Phan T, Brailovsky Y, Fareed J, Hoppensteadt D, Iqbal O, Darki A. Neutrophil-to-lymphocyte and platelet-to-lymphocyte ratios predict all-cause mortality in acute pulmonary embolism. *Clin Appl Thromb Hemost.* 2020;26:1076029619900549.
7. Büyükşirin M, Anar C, Polat G, Karadeniz G. Can the level of CRP in acute pulmonary embolism determine early mortality? *Turk Thorac J.* 2021;22:4-10.
8. Marongiu F, Mameli A, Grandone E, Barcellona D. Pulmonary thrombosis: a clinical pathological entity distinct from pulmonary embolism? *Semin Thromb Hemost.* 2019;45:778-783.
9. Rothenberg ME, Hogan SP. The eosinophil. *Annu Rev Immunol.* 2006;24:147-174.

10. Uderhardt S, Ackermann JA, Fillep T, et al. Enzymatic lipid oxidation by eosinophils propagates coagulation, hemostasis, and thrombotic disease. *J Exp Med.* 2017;214:2121-2138.
11. Toor IS, Jaumdally R, Lip GY, Millane T, Varma C. Eosinophil count predicts mortality following percutaneous coronary intervention. *Thromb Res.* 2012;130:607-611.
12. Zhao HM, Qin WQ, Wang PJ, Wen ZM. Eosinopenia is a predictive factor for the severity of acute ischemic stroke. *Neural Regen Res.* 2019;14:1772-1779.
13. Alkhalil M, Kearney A, Hegarty M, et al. Eosinopenia as an adverse marker of clinical outcomes in patients presenting with acute myocardial infarction. *Am J Med.* 2019;132:e827-e834.
14. Güner A, Zehir R, Kalçık M, 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;11:146-153.
15. Gao S, Deng Y, Wu J, et al. Eosinophils count in peripheral circulation is associated with coronary artery disease. *Atherosclerosis.* 2019;286:128-134.
16. Sincer I, Gunes Y, Mansiroglu AK, Aktas G. Differential value of eosinophil count in acute coronary syndrome among elderly patients. *Aging Male.* 2020;23:958-961.
17. Shao Y, Ye L, Shi HM, et al. Impacts of eosinophil percentage on prognosis acute type a aortic dissection patients. *BMC Cardiovasc Disord.* 2022;22:146.
18. Meng Z, Zhang S, Li W, et al. Cationic proteins from eosinophils bind bone morphogenetic protein receptors promoting vascular calcification and atherogenesis. *Eur Heart J.* 2023;44:2763-2783.
19. Rothenberg ME. Eosinophilia. *N Engl J Med.* 1998;338:1592-600.
20. Shiohara T, Inaoka M, Kano Y. Drug-induced hypersensitivity syndrome (DIHS): a reaction induced by a complex interplay among herpesviruses and antiviral and antidrug immune responses. *Allergol Int.* 2006;55:1-8.
21. Weller PF, Bubley GJ. The idiopathic hypereosinophilic syndrome. *Blood.* 1994;83:2759-2779.
22. Bass DA. Behavior of eosinophil leukocytes in acute inflammation. II. Eosinophil dynamics during acute inflammation. *J Clin Invest.* 1975;56:870-879.
23. Zhang L, Zhou J, Jing Z, et al. Glucocorticoids regulate the vascular remodeling of aortic dissection via the p38 MAPK-HSP27 pathway mediated by soluble TNF-RII. *EBioMedicine.* 2018;27:247-257.
24. Druilhe A, Létuvé S, Pretolani M. Glucocorticoid-induced apoptosis in human eosinophils: mechanisms of action. *Apoptosis.* 2003;8:481-495.
25. Davoine F, Lacy P. Eosinophil cytokines, chemokines, and growth factors: emerging roles in immunity. *Front Immunol.* 2014;5:570.
26. Marx C, Novotny J, Salbeck D, et al. Eosinophil-platelet interactions promote atherosclerosis and stabilize thrombosis with eosinophil extracellular traps. *Blood.* 2019;134:1859-1872.
27. Riegger J, Byrne RA, Joner M, et al; Prevention of Late Stent Thrombosis by an Interdisciplinary Global European Effort (PRESTIGE) investigators. Histopathological evaluation of thrombus in patients presenting with stent thrombosis. A multicenter European study: a report of the prevention of late stent thrombosis by an interdisciplinary global European effort consortium. *Eur Heart J.* 2016;37:1538-1549.
28. Moosbauer C, Morgenstern E, Cuvelier SL, et al. Eosinophils are a major intravascular location for tissue factor storage and exposure. *Blood.* 2007;109:995-1002.
29. Ueki S, Melo RC, Ghiran I, Spencer LA, Dvorak AM, Weller PF. Eosinophil extracellular DNA trap cell death mediates lytic release of free secretion-competent eosinophil granules in humans. *Blood.* 2013;121:2074-2083.