ETMHRJ (2026) 1: 1-8
https://doi.org/.....

Check for updates

Original Articles

OPEN ACCESS

The relationship between blood platelet levels and the ratio of platelets to lymphocytes and monocytes with prevalence and severity of pulmonary thromboembolism

Mahsa Alimoradi[1], Atabak Sedigh-Namin[2], Ghasem Aboutalebi dariasari[3], Shafagh Aliasgarzadeh[4*], Nazli Javaheri[4], Sadaf Motevali[5]

[1] *Islamic Azad University, Ardabil Branch, Ardabil, Iran.*
[2] *Students Research Committee, School of Medicine, Ardabil University of Medical Sciences, Ardabil, Iran.*
[3] *Islamic Azad University, Ardabil, Iran*
[4] *Department of Internal Medicine, School of Medicine, Ardabil University of Medical Sciences, Ardabil, Iran*
[5] *Faculty of Paramedical Sciences, Tabriz University of Medical Sciences, Tabriz, Iran*

Abstract

Introduction: Pulmonary thromboembolism (PTE) is a life-threatening condition caused by an occlusive clot in the pulmonary vasculature. While pulmonary angiography remains the gold standard for diagnosis, its high cost, radiation exposure, and limited availability necessitate simpler, accessible biomarkers for initial assessment. This study aimed to evaluate the association of platelet count, platelet-to-lymphocyte ratio (PLR), and platelet-to-monocyte ratio (PMR) with the prevalence and severity of PTE.

Methods: This case-control study enrolled 80 patients with confirmed PTE and 80 healthy controls at Imam Khomeini Hospital, Ardabil. Demographic data and complete blood counts were collected. Platelet count, PLR, and PMR were compared between groups and further stratified by thromboembolism severity (massive vs. non-massive). Statistical analysis was performed using SPSS version 27.

Results: The mean age of PTE patients was 50.3 years, and 56.3% were female, with no significant demographic differences from controls. Platelet count, PLR, and PMR were significantly lower in the PTE group compared to controls ($P=0.032$, $P<0.001$, and $P<0.001$, respectively). Among PTE patients, 20% had massive embolism and 80% had non-massive embolism. All three parameters were significantly lower in patients with massive thromboembolism compared to those with non-massive disease ($P=0.042$, $P<0.001$, and $P<0.001$, respectively).

Conclusion: Reduced platelet count, PLR, and PMR—reflecting both inflammatory and coagulation pathways—are significantly associated with the occurrence and severity of PTE. These inexpensive, readily available laboratory markers may serve as valuable adjuncts for early diagnosis and prognostic stratification in suspected PTE patients, particularly in settings where advanced imaging is not immediately accessible.

Keywords: Pulmonary Thromboembolism, Thrombosis, Platelet, Platelet-to-Lymphocyte Ratio

Introduction

Pulmonary thromboembolism (PTE) is a circulatory disorder caused by a blood clot (embolus) that travels to the lungs and blocks a pulmonary artery. Pulmonary thromboembolism (PTE) ranks as the third leading cause of death attributed to vascular diseases, following myocardial infarction and cerebrovascular diseases. Initial mortality rates post-diagnosis has been reported to be as high as 15%, with some studies citing PTE mortality rates of up to 65% [1]. A blood clot in a deep vein that travels to the lungs is a common cause

* Corresponding author. E-mail: sh.aliasghrzadeh@arums.ac.ir (S. Aliasgarzadeh).
Received 10 June 2026; Revised 08 August 2026; Accepted 15 August 2026.

of pulmonary thromboembolism (PTE). PTE can also be caused by factors that raise the risk of blood clots, such as prolonged immobility, certain drugs, and active cancer. A hypercoagulable state, where blood is more likely to clot, can result from these conditions [2,3]. Several prognostic factors have been proposed to predict the course of pulmonary thromboembolism. For example, increased serum levels of C-reactive protein (CRP) and cardiac troponin I (cTnI) have been related result of increased poorer prognosis disease severity [2]. Pulmonary embolism can significantly impact patients' lives, not only by causing death but also by reducing their quality of life and leading to complications. By identifying risk factors and predicting outcomes, doctors can diagnose the condition earlier and develop strategies to prevent it in people who are most at risk [4].

Platelets are necessary for blood clotting. Blood clot risk is related to increased platelet activation as defined by mean platelet volume (MPV) [1] and platelet distribution width (PDW) [2]. Larger activity and the potential for blood clots to form, particularly in deep veins, are linked to larger platelets, which may result in pulmonary embolism [5,6]. For example, elevated platelet counts have been reported as a risk factor for pulmonary thromboembolism in patients undergoing chemotherapy [7].

The coagulation system relies on different blood cells and particles requested in routine tests for admission besides platelets. For example, monocytes are the primary intravascular source of tissue factor (TF), which is an essential initiator of the coagulation cascade. Also, neutrophils and other leukocytes release cytoplasmic granules which can have an impact on the coagulation process. Additionally, the platelet-to-lymphocyte ratio (PLR) is a potential indicator of how serious or lasting the effects are in inflammation induced pulmonary thromboembolism [8].

Pulmonary angiography is now considered the gold standard for the diagnosis of pulmonary thromboembolism but its high price and high radiation exposure, nevertheless are not feasible even in many cases of pregnant mother; Studies, as per reports, from the United States indicate a 5–10% positive rate [9]. By looking at these aspects, seems that the establishment of accessible and non-invasive methods that can in future replace or confirm expensive and laborious procedures have meant a prompt thromboembolism diagnosis will substantially reduce the financial and physical burden imposed to the patient and healthcare system. Thus, the purpose of this study was to establish the connection between the platelet count and the ratios of platelet to lymphocyte as well as platelet to monocyte with the prevalence and severity of pulmonary embolism.

Materials & Methods

This study was conducted as a case-control study to investigate the association between blood platelet count and PLR and PML ratios and the prevalence and severity of pulmonary thromboembolism. The target population consisted of patients with pulmonary thromboembolism. A control group was selected from patients without blood or inflammatory disorders and no history of thromboembolism, matched for age, sex, and body mass index.

The case group was selected from patients hospitalized at Imam Khomeini Hospital in Ardabil, Iran, during 2023. The control group was selected from individuals referred to the hospital's laboratory. The required sample size was calculated based on the results of a previous study by Ali Dai and colleagues [10], considering a 5% error rate and a 95.1% confidence level. A total of 63 individuals were initially calculated. To reduce the error rate, 80 individuals were ultimately included in the case group and 80 in the control group.

Inclusion criteria for the case group included age over 18 years and a confirmed diagnosis of pulmonary embolism by accurate diagnostic methods (perfusion scan/pulmonary angiography). Exclusion criteria for the case group included concomitant presence of diseases affecting hematological parameters such as chronic anemia, cancers, coagulation disorders, or primary thrombocytosis.

Inclusion criteria for the control group included age over 18 years and no previous history of pulmonary embolism. Exclusion criteria for the control group included concomitant presence of diseases affecting hematological parameters such as chronic anemia, cancers, coagulation disorders, or primary thrombocytosis.

A researcher-prepared data checklist was used to collect information. Necessary information included

demographic characteristics, underlying diseases, pulmonary angiography findings (massive or non-massive pulmonary embolism), oxygen saturation, and paraclinical characteristics (number of leukocytes, lymphocytes, monocytes, and platelets). This information was extracted from patient records and recorded in the checklist.

Results

The demographic characteristics of the study population are presented in Table 1. The overall gender distribution was 55% female, with 56.3% in the PTE group and 53.8% in the control group. According to Fisher's exact test, there was no significant difference between the case and control groups in terms of gender (p=0.874).

Figure 1 illustrates the age distribution, demonstrating a normal distribution with a majority of patients aged between 40 and 60 years. The mean age of all patients in both groups was 52.7 ± 3.03 years, with a range of 18 to 89 years. The mean age in the case group was 50.3 ± 19.7 years, and in the control group, it was 55.2 ± 14.4 years. According to the independent t-test results, there was no significant difference in age between the case and control groups (P = 0.068).

Table 1. Gender distribution in case and control groups

Gender	Group		P Value
	Cases (PTE)	Controls (non-PTE)	
Male	35 (43.8 %)	37 (46.3 %)	0.874
Female	45 (56.3 %)	43 (53.8 %)	

According to the Kolmogorov-Smirnov test, platelet counts in the patients were normally distributed. Therefore, an independent samples t-test was used at a significance level of 0.05 to compare platelet counts between the case and control groups. As presented in Table 2, the mean and standard deviation of platelet counts in the case group (PTE) were 230,837.5/µL and 83,843.04/µL, respectively. In the control group (non-PTE), the mean and standard deviation were 260,412.5/µL and 83,503.3/µL, respectively. The independent samples t-test revealed a statistically significant difference in platelet counts between the PTE and Non-PTE groups (P = 0.032). In other words, patients with PTE had significantly lower platelet counts compared to the control group. This finding suggests a significant association between the occurrence of thromboembolism and lower platelet counts.

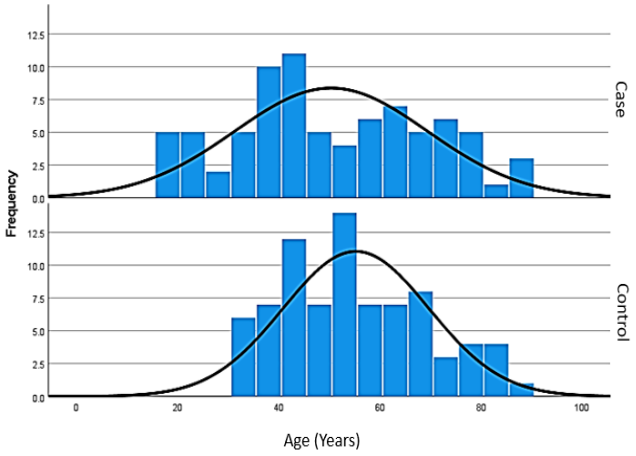


Figure 1. Age distribution of study groups

The platelet-to-lymphocyte ratio (PLR) was calculated by dividing the platelet count (cells/µL) by the lymphocyte count (cells/µL). The Kolmogorov-Smirnov test confirmed that the PLR data was normally distributed. Therefore, an independent t-test was used to compare the PLR between the case and control groups. As presented in Table 2, the mean and standard deviation of PLR in the PTE group was 1.27 ± 0.58, compared to the control group which had a mean PLR of 2.19 ± 1.23. According to the independent t-test, the mean PLR was significantly lower in the PTE group compared to the control group (p < 0.001). This finding indicates a significant inverse correlation between the occurrence of PTE and PLR. In other words, patients with PTE had a lower PLR compared to those without PTE.

The platelet-to-monocyte ratio (PMR) was calculated by dividing the platelet count (cells/µL) by the monocyte count (cells/µL). According to the Kolmogorov-Smirnov test, the PMR data were normally distributed. Therefore, an independent samples t-test was used to compare the PMR between the case and control groups. As presented in Table 2, the mean PMR in the group of patients with PTE was 8.78 ± 5.52, while the mean PMR in the control group was 12.25 ± 6.16. According to the independent samples t-test, the PMR was significantly lower in the group of patients with PTE compared to the control group (P < 0.001). In other words, the occurrence of thromboembolism was significantly associated with a lower PMR.

Table 2. Platelet counts, Platelet to lymphocyte ratio and Platelet to Monocyte ratio in the groups

Parameter	Group		P Value
	Cases (PTE)	Controls (non-PTE)	
Platelet count (/µL)	230837.5 ± 83843.04	260412.5 ± 83503.3	0.032
Platelet to lymphocyte ratio (PLR)	1.27 ± 0.58	2.19 ± 1.23	<0.001
Platelet to Monocyte ratio (PMR)	8.78 ± 5.52	12.25 ± 6.16	<0.001

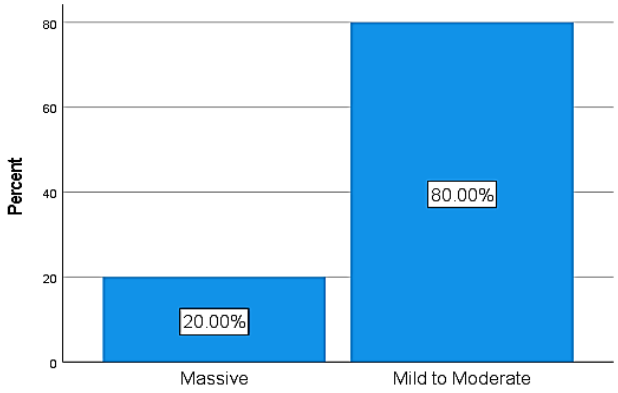


Figure 2. Frequency of severity of thromboembolism in studied patients

As presented in Table 3, in comparing patients with massive and non-massive pulmonary thromboembolism, the mean platelet count in the group of patients with massive pulmonary thromboembolism was 196,250.00 ± 68,816.18 platelets/µL. In contrast, the mean platelet count in the group of patients with non-massive pulmonary thromboembolism was 239,484.38 ± 85,484.79 platelets/µL. According to the independent t-test, there was a statistically significant difference in platelet counts between the two groups (P = 0.042). Specifically, patients with massive pulmonary thromboembolism had significantly lower platelet counts compared to those with non-massive pulmonary thromboembolism. These findings suggest a significant association between the occurrence of massive pulmonary thromboembolism and lower platelet counts.

Regarding the platelet-to-lymphocyte ratio (PLR) in relation to the severity of pulmonary embolism, the mean PLR in the group of patients with massive pulmonary thromboembolism was 1.27 ± 0.58, while the mean PLR in the group of patients with non-massive pulmonary thromboembolism was 2.19 ± 1.23. According to the independent t-test results, the PLR was significantly lower in patients with massive pulmonary thromboembolism compared to those with non-massive pulmonary thromboembolism (P < 0.001). In other words, the occurrence of massive pulmonary thromboembolism was significantly associated with a lower PLR.

As presented in Table 3, the mean platelet-to-monocyte ratio (PMR) was significantly lower in the group with massive pulmonary thromboembolism (mean = 5.89 ± 3.46) compared to the group with non-massive pulmonary thromboembolism (mean = 5.72 ± 9.52) (p < 0.001). This finding suggests that a lower PMR is associated with a higher likelihood of developing massive pulmonary thromboembolism.

Table 3. Platelet counts, Platelet to lymphocyte ratio and Platelet to Monocyte ratio in the groups between pulmonary thromboembolism patients

Parameter	pulmonary thromboembolism		P
	Massive (n=16) Mean ± SD	non-Massive (n=64) Mean ± SD	
Platelet count (/µL)	196250.00 ± 68816.18	239484.38 ± 85484.79	0.042
Platelet to lymphocyte ratio (PLR)	1.27 ± 0.58	2.19 ± 1.23	<0.001
Platelet to Monocyte ratio (PMR)	5.89 ± 3.46	5.72 ± 9.52	<0.001

Discussion

"According to the present study, patients with pulmonary embolism had a mean age of approximately 50 years, and there was a near equal gender distribution. Similarly, Ogung and colleagues (South Africa, 2011) reported a nearly equal male-to-female ratio in pulmonary embolism patients, although the mean age was lower at around 40 years [11]. Naglamalash and colleagues also reported a similar gender distribution and age range in their study [12]. Moghadam and colleagues' study in eastern Iran, aligned with the present study, demonstrated a balanced gender distribution

among pulmonary embolism patients, with a mean age similar to our findings [13].

Regarding the relationship between age, sex, and the occurrence of pulmonary embolism, most studies align with our findings. The risk of thrombotic events such as deep vein thrombosis and pulmonary embolism increases with age, leading to recommendations for preventive treatments in older individuals, especially those over 50 years [14]. This difference is even more pronounced in individuals over 75 years [15].

Regarding sex and the occurrence of pulmonary embolism, basic studies have shown sex-based differences in platelet function, and animal models have reported sex differences in thrombosis [16]. Some studies have investigated differences in thrombotic events such as deep vein thrombosis and pulmonary embolism between sexes. Although some have reported that the overall incidence of first PTE is similar between men and women [17,18], results from other studies align with the hypothesis that female sex may be a protective factor for PTE. In a large-scale study based on UK health data, women had a lower risk of PTE compared to men [19]. Similarly, a community-based study in western France found a significantly lower annual incidence rate of PTE in men compared to women [20]. Silvestri and colleagues [21] reported that the incidence of DVT remained constant for men across all age groups, but decreased in women under 55 years and increased for women over 60 years. However, a recent systematic review by Romero and colleagues concluded that sex is not an independent risk factor for PTE [22]; thus, the observed gender parity among patients with pulmonary embolism in the present study is expected.

Our findings demonstrated a significantly lower platelet count in patients with pulmonary thromboembolism compared to those without thrombotic events. Consistent with our results, Moneral et al. also reported a significant association between pulmonary thromboembolism and decreased platelet counts [23]. Furthermore, our study revealed that platelet levels were significantly lower in cases of severe and major thromboembolism compared to moderate and mild cases, supporting the notion of decreased platelet levels following venous thrombus formation and pulmonary thromboembolism. Similarly, another study found that platelet counts decreased by approximately 20% following pulmonary thromboembolism [23]. Varol et al. also reported significantly lower platelet levels in patients with thromboembolism compared to the control group [24]. Moneral et al. suggested that the decrease in platelet count following thrombus formation in pulmonary thromboembolism could predict the severity and occurrence of pulmonary thromboembolism with high sensitivity and specificity [25]. However, further studies are needed, as considering only platelet count for diagnosing and assessing the severity of pulmonary thromboembolism may be misleading. In contrast to our findings, Hang et al. found no significant association between platelet count and pulmonary thromboembolism, suggesting that it cannot be considered a diagnostic factor [26]. Ali Dai et al. also found no significant association between platelet levels and the occurrence of pulmonary thromboembolism, but some platelet-related factors, such as mean platelet volume (MPV), were associated with pulmonary thromboembolism [10]. Rabieipour et al. (2019, Urmia) did not report a significant association between platelet count and pulmonary thromboembolism but found significant associations with other platelet parameters such as MPV and platelet distribution width (PDW) [27]. In line with these findings, Varol et al. also emphasized the association of MPV with pulmonary thromboembolism [24].

Overall, while various studies have shown associations between platelet-related indices, including count, mean volume, and distribution, and the occurrence and severity of pulmonary thromboembolism, the results are sometimes inconsistent and inconclusive. Further extensive research is needed to gain a comprehensive understanding and to identify specific cut-off values for these parameters to predict patient prognosis upon initial presentation with suspected pulmonary thromboembolism.

Inflammation has been proposed as a key mechanism linking PE and alterations in hematological parameters. Inflammation plays a pivotal role in thrombus propagation and the pathophysiology of PE [28], and prognostic values of various hematological parameters have been correlated with the inflammatory status in PE patients. Given the known role of inflammation in PE, neutrophils, lymphocytes, and platelets have been suggested as useful prognostic markers in these

patients [29]. Since various types of white blood cells, including neutrophils, eosinophils, and monocytes, are associated with inflammation, parameters formed from the ratios of these parameters are more practical as they combine two independent inflammatory markers [30]. The platelet-to-lymphocyte ratio (PLR) is a novel inflammatory marker that is inexpensive and readily available at the bedside. PLR has been used as a predictor of inflammation and mortality in various diseases, such as cardiovascular diseases and autoimmune diseases [31]. Karatas and colleagues [32] investigated the prognostic value of various parameters in patients with PE and showed that PLR was an independent prognostic factor for short-term and long-term mortality in these patients. Telo and colleagues [33] also showed that PLR was elevated in high-risk and high-severity PE patients. They demonstrated that PLR may have prognostic value for predicting 3-month mortality. Several other studies have reported similar findings regarding the prognostic role of PLR in PE [34,35], suggesting that it can be routinely used in the prognostic assessment of PE. The results of the present study are also consistent with previous studies, clearly demonstrating that the inflammatory parameter PLR was significantly lower in individuals with pulmonary thromboembolism compared to the control group, and this association was also significant with the severity of thromboembolism.

Another inflammatory parameter assessed in this study was the platelet-to-monocyte ratio (PMR). Our results demonstrated a significant association between PMR and both the incidence and severity of pulmonary thromboembolism. Notably, patients with severe pulmonary thromboembolism exhibited a significantly decreased PMR. PMR, a relatively novel inflammatory marker, has been the subject of limited research to evaluate its diagnostic value. For instance, a recent study by Zhou and colleagues revealed a significant correlation between PMR and short-term mortality in patients with hepatitis B [36]. Another study demonstrated a significant association between PMR and mortality and prognosis in patients with acute myocardial infarction [37]. Białas and colleagues showed that the monocyte-to-platelet ratio (inverse of PMR), particularly in individuals with chronic obstructive pulmonary disease (COPD), could significantly predict the occurrence of pulmonary thromboembolism [38]. However, comprehensive studies investigating the association between PMR and thrombotic events are still lacking, and the available data are limited, necessitating further research in this area.

Conclusion

The results of this study demonstrated that inflammatory parameters, such as the platelet-to-lymphocyte ratio (PLR) and platelet count, a factor associated with coagulation status, were correlated with the occurrence and severity of pulmonary thromboembolism. These parameters can be considered as predictive factors alongside risk stratification criteria like the Wells score, and can also be used to assess the prognosis of patients with suspected pulmonary thromboembolism.

Informed Consent

Written informed consent was obtained from all participants or their legal guardians prior to enrollment.

Ethics Approval

This study was approved by the Ethics Committee of Ardabil University of Medical Sciences (Ethics Code: IR.IAU.ARDEBIL.REC.1401.152).

Financial Support / Funding

This research received no specific grant from any funding agency.

Conflicts of Interest (COI)

The authors declare that they have no conflicts of interest.

References

1. Ucar EY. Update on Thrombolytic Therapy in Acute Pulmonary Thromboembolism. Eurasian J Med. 2019;51(2):186-90.
2. Tarbox AK, Swaroop M. Pulmonary embolism. Int J Crit Illn Inj Sci. 2013;3(1):69-72.
3. Konstantinides S. Acute pulmonary embolism. New England Journal of Medicine. 2008;359(26):2804-13.
4. Ghaffari S, Parvizian N, Pourafkari L, Separham A, Hajizadeh R, Nader ND, et al. Prognostic value of platelet indices in patients with acute pulmonary

thromboembolism. J Cardiovasc Thorac Res. 2020;12(1):56-62.
5. Kirresh A, Candilio L, Stone GW. Intralesional delivery of glycoprotein IIb/IIIa inhibitors in acute myocardial infarction: review and recommendations. Catheterization and Cardiovascular Interventions. 2022;99(3):641-9.
6. Sadeghi F, Kovács S, Zsóri KS, Csiki Z, Bereczky Z, Shemirani AH. Platelet count and mean volume in acute stroke: a systematic review and meta-analysis. Platelets. 2020;31(6):731-9.
7. Song JE, Lee DW, Han J, Kim HG. The increment of platelet count is risk factor of pulmonary thromboembolism in patients with pancreatic cancer treated with gemcitabine. Pancreatology. 2016;1(16): S54.
8. Swystun LL, Liaw PC. The role of leukocytes in thrombosis. Blood, The Journal of the American Society of Hematology. 2016;128(6):753-62.
9. Perrier A, Roy P-M, Aujesky D, Chagnon I, Howarth N, Gourdier A-L, et al. Diagnosing pulmonary embolism in outpatients with clinical assessment, D-dimer measurement, venous ultrasound, and helical computed tomography: a multicenter management study. The American journal of medicine. 2004;116(5):291-9.
10. Alidaei N, Zarrin S, Eishi A, Rahimi M-H. Evaluation of the relationship between platelet count, mean platelet volume and pulmonary thromboembolism. Razi J Med Sci. 2012;19(94):20-6.
11. Ogeng'o JA, Obimbo MM, Olabu BO, Gatonga PM, Ong'era D. Pulmonary thromboembolism in an East African tertiary referral hospital. J Thrombos Thrombol. 2011;32(3):386-91.
12. Nagamalesh UM, Prakash VS, Naidu KCK, Sarthak S, Hegde AV, Abhinay T. Acute pulmonary thromboembolism: Epidemiology, predictors, and long-term outcome – A single center experience. Ind Heart J. 2017;69(2):160-4.
13. Ghasemian Moghaddam MR, Azdaki N, Ghasemi MS. Acute Pulmonary Embolism and 1-Year Outcome: An Epidemiological Study in East of Iran, 2011-2016. Mod Care J. 2020;17(1):12-20.
14. Montagnana M, Favaloro EJ, Franchini M, Guidi GC, Lippi G. The role of ethnicity, age and gender in venous thromboembolism. J Thrombos Thrombol. 2010;29(4):489-96.
15. Bai Y, Yue QM, Sun H, Guo SD, Wang ZZ, Zhong P, et al. Prevalence and sex- and age-related risk of pulmonary embolism in in-hospital patients with atrial fibrillation: a multicenter retrospective study from China. Ann Transl Med. 2020;8(23):15-58.
16. Bailey AL, Scantlebury DC, Smyth SS. Thrombosis and antithrombotic therapy in women. Arterioscler Thrombos Vasc Biol. 2009;29(3):284-8.
17. Anderson FA, Wheeler HB, Goldberg RJ, Hosmer DW, Patwardhan NA, Jovanovic B, et al. A population-based perspective of the hospital incidence and case-fatality rates of deep vein thrombosis and pulmonary embolism: the Worcester DVT Study. Arch Intern Med. 1991;151(5):933-8.
18. Nordström M, Lindblad B, Bergqvist D, Kjellström T. A prospective study of the incidence of deep-vein thrombosis within a defined urban population. J Intern Med. 1992;232(2):155-60.
19. Proctor MC, Wainess RM, Henke PK, Upchurch GR, Wakefield TW. Venous thromboembolism: regional differences in the nationwide inpatient sample, 1993 to 2000. Vascular. 2004;12(6):374-80.
20. Oger E, group E-Gs. Incidence of venous thromboembolism: a community-based study in Western France. Thrombos Haemostas. 2000;83(05):657-60.
21. Silverstein MD, Heit JA, Mohr DN, Petterson TM, O'Fallon WM, Melton LJ. Trends in the incidence of deep vein thrombosis and pulmonary embolism: a 25-year population-based study. Arch Intern Med. 1998;158(6):585-93.
22. Romero A, Alonso C, Rincón M, Medrano J, Santos J-M, Calderón E, et al. Risk of venous thromboembolic disease in women: A qualitative systematic review. Eur J Obstetr Gynecol Reproduct Biol. 2005;121(1):8-17.
23. Monreal M, Lafoz E, Casals A, Ruíz J, Arias A. Platelet Count and Venous Thromboembolism: A Useful Test for Suspected Pulmonary Embolism. Chest. 1991;100(6):1493-6.
24. Varol E, Icli A, Uysal BA, Ozaydin M. Platelet indices in patients with acute pulmonary embolism. Scand J Clin Lab Invest. 2011;71(2):163-7.
25. Monreal M, Lafoz E, Ruiz J, Gimenez G. Platelet count in acute pulmonary embolism: its relationship to recurrences. Haemostasis. 1993;23(5):263-8.
26. Huang J, Chen Y, Cai Z, Chen P. Diagnostic value of platelet indexes for pulmonary embolism. Am J Emerg Med. 2015;33(6):760-3.
27. Rabiepour M, Valipour H, Islampour P, pashaei MR. Predictive value of platelet markers in the patients with acute pulmonary thromboembolism. Stud Med Sci. 2022;33(1):62-9.
28. Marongiu F, Mameli A, Grandone E, Barcellona D. Pulmonary thrombosis: a clinical pathological entity distinct from pulmonary embolism? Semin Thrombos Hemostas. 2019;45(08):778-83.
29. Acanfora D, Gheorghiade M, Trojano L, Furgi G, Pasini E, Picone C, et al. Relative lymphocyte count: a prognostic indicator of mortality in elderly patients with congestive heart failure. Am Heart J 2001;142(1):167-73.
30. Ly HQ, Kirtane AJ, Murphy SA, Buros J, Cannon CP, Braunwald E, et al. Association of platelet counts on presentation and clinical outcomes in ST-elevation myocardial infarction (from the TIMI Trials). Am J Cardiol. 2006;98(1):1-5.

31. Bonow RO, Fonarow GC, O'Gara PT, Yancy CW. Association of coronavirus disease 2019 (COVID-19) with myocardial injury and mortality. JAMA Cardiol. 2020;5(7):751-3.
32. Karataş MB, İpek G, Onuk T, Güngör B, Durmuş G, Çanga Y, et al. Assessment of prognostic value of neutrophil to lymphocyte ratio and platelet to lymphocyte ratio in patients with pulmonary embolism. Acta Cardiol Sin. 2016;32(3):313-20.
33. Telo S, Kuluöztürk M, Deveci F, Kirkil G. The relationship between platelet-to-lymphocyte ratio and pulmonary embolism severity in acute pulmonary embolism. J Int Union Angiol. 2019;38(1):4-9.
34. Ates H, Ates I, Kundi H, Yilmaz FM. Diagnostic validity of hematologic parameters in evaluation of massive pulmonary embolism. J Clin Lab. 2017;31(5):12-20.
35. Galliazzo S, Nigro O, Bertù L, Guasti L, Grandi AM, Ageno W, et al. Prognostic role of neutrophils to lymphocytes ratio in patients with acute pulmonary embolism: a systematic review and meta-analysis of the literature. Intern Emerg Med. 2018;13(4):603-
36. Zhou J, Li X, Wang M, Gu C, Liu J. Platelet-to-Monocyte Ratio as a Novel Promising Agent for the Prognosis of Hepatitis B Virus-Associated Decompensated Cirrhosis. Can J Gastroenterol Hepatol. 2023; 2023:12-20.
37. Çaltekin İ, Demirtaş E. Is platelet to monocyte ratio a useful inflammatory determinant of ST-elevation myocardial infarction? Disaster Emerg Med J. 2020;5(4):205-11.
38. Białas AJ, Kornicki K, Ciebiada M, Antczak A, Sitarek P, Miłkowska-Dymanowska J, et al. Monocyte to large platelet ratio as a diagnostic tool for pulmonary embolism in patients with acute exacerbation of chronic obstructive pulmonary disease. Pol Arch Intern Med. 2018;128(1):15-23.