

EARLY RISK STRATIFICATION IN ACUTE PULMONARY EMBOLISM USING INFLAMMATORY AND HEMATOLOGIC BIOMARKERS

Bekim Pocesta, Lidija Poposka, Elif Vrajnko,
Tomislav Konjanovski, Marijan Bosevski, Ljubica Georgievska-Ismail

University Clinic of Cardiology, Faculty of Medicine, University “Ss. Cyril and Methodius” Skopje, RN Macedonia

Corresponding author: Bekim Pocesta, University Clinic of Cardiology, Faculty of Medicine, University “Ss. Cyril and Methodius”, Skopje, Republic of North Macedonia, e-mail: bekimpoc@yahoo.com

ABSTRACT

Background: Accurate early risk stratification in acute pulmonary embolism (PE) remains challenging, particularly in intermediate-risk patients. Readily available hematologic and inflammatory markers may provide additional prognostic value.

Objective: To evaluate the predictive role of hematologic and inflammatory markers for in-hospital adverse events in patients with acute pulmonary embolism.

Methods: This retrospective study included 88 pts diagnosed with acute PE between 2023 and 2024. Clinical, echocardiographic, and laboratory data, including red blood cell distribution width (RDW), white blood cell (WBC) count, neutrophil-to-lymphocyte ratio (NLR), platelet-to-lymphocyte ratio (PLR), and C-reactive protein (CRP), were analyzed. Adverse events were defined as hemodynamic instability, cardiac arrest, or death during hospitalization. Logistic regression identified independent predictors; ROC curves assessed discriminative performance.

Results: Adverse events occurred in 13 pts (14.8%), including four deaths. Pts with adverse events had significantly higher RDW ($p = 0.008$), WBC ($p = 0.002$), NLR ($p = 0.001$), PLR ($p = 0.001$), and CRP ($p = 0.028$). Multivariable analysis identified RDW (OR 1.48; 95% CI 1.12–1.96; $p = 0.006$), WBC (OR 1.20; 95% CI 1.02–1.41; $p = 0.032$), and NLR (OR 1.14; 95% CI 1.02–1.27; $p = 0.018$) as independent predictors. ROC analysis showed improved discrimination when these markers were combined (AUC 0.880).

Conclusion: Red blood cell distribution width, white blood cell count, and neutrophil-to-lymphocyte ratio independently predict in-hospital adverse events in acute pulmonary embolism. Their combined use may enhance early risk stratification.

Keywords: pulmonary embolism, C-reactive protein, prognostic markers, in-hospital mortality

INTRODUCTION

Pulmonary embolism (PE) is a common and potentially life-threatening medical emergency, ranking third in incidence after myocardial infarction and stroke. It significantly impacts patient outcomes, with in-hospital mortality rates estimated at approximately 14%, and 3-month mortality at 20% [1, 2]. Advances in early diagnosis, therapeutic interventions, and adherence to clinical guidelines

have contributed to improved survival in recent years [2]. Accurate early risk stratification is critical for guiding treatment and improving outcomes. Existing models such as the simplified Pulmonary Embolism Severity Index (sPESI) and the 2019 European Society of Cardiology (ESC) classification are widely used but have limitations, particularly in intermediate-risk patients, who represent ap-

proximately one-third of all PE cases and in whom risk stratification remains challenging [2]. Thus, there is a need for additional biomarkers that can enhance the early risk prediction — ideally those that are readily available and routinely assessed upon admission.

Inflammation plays a central role in the pathophysiology of acute PE. Evidence suggests that elevated leukocytes counts, as well as C-reactive protein (CRP), as a marker of systemic inflammation, are associated with increased disease severity, right ventricular (RV) dysfunction, and/or early mortality in patients with acute PE [3-8].

In addition, hematologic indices such as red blood cell distribution width (RDW) [3,4,9-11], neutrophil-to-lymphocyte ratio (NLR), and platelet-to-lymphocyte ratio (PLR) [3,4,12-14] have also been identified as potential prognostic markers in cardiovascular diseases, including PE. These markers are easily accessible and inexpensive, making them attractive for early risk assessment.

The present study aimed to evaluate the prognostic significance of hematologic as well inflammatory markers for predicting in-hospital adverse events in patients with acute PE and to assess whether combining these biomarkers improves early risk stratification.

MATERIALS AND METHODS

This retrospective study included 88 consecutive adult patients (≥ 18 years) diagnosed with acute PE between 2023 and 2024 and admitted to the Intensive Coronary Care Unit at the University Clinic of Cardiology, Skopje, RN Macedonia.

The diagnosis of PE was confirmed by computed tomography pulmonary angiography (CTPA) demonstrating a filling defect or abrupt cutoff in at least two projections [2]. Exclusion criteria included: 1) death within 48 hours of admission, 2) prior thrombolytic or anticoagulant therapy before blood sampling, 3) recent infection, 4) use of corticosteroid or immunosuppressive agents, 5) blood transfusion within the preceding 2 weeks, 6) advanced hepatic or renal disease (including dialysis) and/or 7) known hematologic disorders or acute/chronic inflammatory diseases.

The study protocol was approved by the Ethics Committee of the Medical Faculty at University “Ss. Cyril and Methodius”, Skopje, RNM. Informed consent was waived due to the retrospective design.

Demographic characteristics, comorbidities, PE risk factors, vital signs, imaging findings (CTPA, transthoracic echocardiography [TTE], Doppler ultrasound [DUS] of lower limbs), laboratory values at admission, sPESI score, and ESC 2019 risk classification were recorded [2].

Blood sampling was performed immediately upon admission before initiating anticoagulation and processed in the central clinical laboratory.

Adverse events (AEs) were defined as any of the following: hemodynamic instability (cardiogenic shock, need for vasopressors/inotropes, or rescue thrombolysis), in-hospital cardiac arrest requiring resuscitation, or death within 30-days of hospitalization.

Statistical Analysis

The continuous variables were expressed as mean $\pm$ standard deviation (SD) and compared using the Student's t-test. The categorical variables were analyzed with the Pearson's Chi-square test. Correlations were assessed using Pearson or Spearman coefficients. Independent predictors of AEs were identified using stepwise logistic regression. The receiver operating characteristic (ROC) curve analysis evaluated the predictive accuracy of individual and combined biomarkers.

A p-value < 0.05 was considered statistically significant. Analyses were performed using SPSS v25 (IBM Corp., Armonk, NY, USA).

RESULTS

A total of 88 patients with mean age 63.1 ± 12.7 years (range: 34-89), including 54.5% males were included. Based on the sPESI [2], 20 patients (22.7%) were classified as low risk (30-day mortality risk of 1%) and 68 (77.3%) as high risk (30-day mortality risk of 10.9%). According to the ESC classification for early mortality risk (in-hospital or within 30 days), patients were categorized as follows (2): low risk

22 patients (25.0%), intermediate-low risk 34 patients (38.6%), intermediate-high risk 27 patients (30.7%), and high risk 5 patients (5.7%).

In-hospital AEs occurred in 13 patients (14.8%), including 4 deaths (4.5%). Two of these deaths were sudden, while the others followed hemodynamic deterioration. Seven patients (8.0%) with cardiopulmonary arrest were successfully resuscitated, and discharged.

Clinical Characteristics and Biomarkers

Baseline, paraclinical, and clinical characteristics of the study population are presented

in Table 1. Patients with AEs were older and with female predominance (not statistically significant), more often obese ($p=0,054$), with higher prevalence of prior deep vein thrombosis and pulmonary embolism ($p=0,058$ and $p=0,052$, respectively). However, no significant differences were observed between the groups in terms of recent surgery, active malignancy, previous trauma, or risk factors such as hypertension, diabetes, COPD, and smoking.

Patients with AEs more frequently had hypoxemia (lower SpO_2 , $p = 0.045$), right bundle branch block (RBBB, $p = 0.031$), higher pulmo-

Table 1. Evaluation of baseline characteristics and paraclinical and clinical parameters in patients exhibiting and not exhibiting unfavorable in-hospital outcomes

Variables	Total cohort n=88	With of AE n=13	Without AE n=75	p Value
Age (years)	63.14± 12.76	64.85± 12.39	62.84± 12.88	0.604
Sex-m/f (n/%)	m 48/54.5; 40/45.5	m 5/38.5; f 8/61.5	m 43/57.3; f 32/42.7	0.169
BMI (kg/m ²)	28.91± 5.82	33.61 ± 10.05	28.29 ± 4.92	0.054
History of PE (n/%)	8/9.1	3/23.1	5/6.7	0.059
Recent surgery (n/%)	6/6.8	1/7.7	5/6.7	0.628
Malignancy (n/%)	15/17.0	4/30.8	11/14.7	0.152
Major trauma (n/%)	6/6.8	1/7.7	5/6.7	0.628
Hist. of DVT (n/%)	12/13.6	4/30.8	8/10.7	0.053
Hypertension (n/%)	41/46.6	5/38.5	36/48.0	0.371
DM (n/%)	14/15.9	4/30.8	10/13.3	0.122
COPD (n/%)	10/11.4	3/23.1	7/9.3	0.163
Smoking (n/%)	10/11.4	3/23.1	7/9.3	0.163
Systolic BP (mmHg)	138.53± 24.12	141.46± 27.14	138.03± 23.72	0.638
HR. (bpm)	103.84± 21.95	110.92± 17.40	102.61± 22.52	0.210
SpO ₂ (%)	89.91± 8.40	85.00± 12.82	90.73± 7.26	0.045
AF-ECG (n/%)	14/15.9	4/30.8	10/13.3	0.113
RBBB-ECG (n/%)	18/20.5	5/38.5	13/17.3	0.031
S1Q3T3-ECG (n/%)	21/34.4	5/38.5	24/32.0	0.647
DVT-DUS (n/%)	25/39.7	5/55.6	20/37.0	0.293
PAPs-TTE (mmHg)	42.62 ± 18.62	54.63 ± 20.72	40.26 ± 17.42	0.018
RVD-TTE (n/%)	35/40.68	8/61.5	27/37.0	0.089
sPESI				
0	20/22.7	0	20/26.7	0.026
≥1	68/77.3	13/100	55/73.3	
Early risk of mortality (n/%)				0.0001
low	22/25.0	0	22/29.3	
intermediate-low	34/38.6	3/23.1	31/41.3	
intermediate-high	27/30.7	5/38.5	22/29.3	
high	5/5.7	5/38.5	0	

AE=adverse events, m=male, f=female, BMI=body mass index, PE=pulmonary embolism, DVT=deep vein thrombosis, DM=diabetes, COPD=chronic obstructive pulmonary disease, BP=blood pressure, HR= heart rate, SpO₂=oxygen saturation, AF=atrial fibrillation, RBBB=right brunch bundle block, ECG=electrocardiography, RVD=right ventricular dysfunction, DUS= Doppler ultrasound of the lower limb veins, PAPs=pulmonary artery systolic pressure, TTE=transthoracic echocardiography, sPESI=simplified Pulmonary Embolism Severity Index.

nary artery pressure (PAPs, $p = 0.018$), and more often belonged to the intermediate-high/high ESC risk groups ($p = 0.0001$).

The results of the hematological analyses are presented in Table 2. All measured parameters were worse in the adverse event group. Notably, leukocyte count, neutrophil count, neutrophil-to-lymphocyte ratio, platelet-to-lymphocyte ratio, red cell distribution width, and C-reactive protein were significantly higher in the adverse event group.

Significant correlation with AEs were found for higher heart rate ($r = 0.246$, $p = 0.021$), lower SpO₂ ($r = -0.240$, $p = 0.045$), presence of RBBB on ECG ($r = 0.230$, $p = 0.031$), higher systolic pulmonary artery pressure (PAPs) ($r = 0.288$, $p = 0.018$), presence of thrombus in the right atria ($r = 0.257$, $p = 0.017$) and lower TAPSE/PAPs ratio ($r = -0.267$, $p = 0.030$) assessed by TTE.

As for hematological parameters, significant correlation with AEs were found for higher value of RDW% ($r = 0.280$, $p = 0.028$), leukocytes ($r = 0.321$, $p = 0.008$), NLR ($r = 0.352$, $p = 0.001$), PLR ($r = 0.336$, $p = 0.001$) and CRP level ($r = 0.235$, $p = 0.028$).

Higher sPESI and higher risk of early mortality were significantly related to AEs ($r = 0.226$, $p = 0.034$, $r = 0.471$, $p = 0.0001$; respectively).

Predictive Analysis

When all variables with significant correlations were entered into a multivariable stepwise logistic regression analysis, with AEs as the dependent variable, the model identified the following independent predictors: Higher RDW%: OR 1.686 (95% CI 1.099–2.587, $p = 0.017$), higher NLR: OR 1.237 (95% CI 1.032–1.484, $p = 0.022$) and high early mortality risk according to ESC classification: OR 11.448 (95% CI 1.074–122.042, $p = 0.043$).

When regression was limited to hematological variables, the final results are shown in Table 3. RDW% (OR 1.48; 95% CI 1.12–1.96; $p = 0.006$), leukocyte count (OR 1.20; 95% CI 1.02–1.41; $p = 0.032$), and NLR (OR 1.14; 95% CI 1.02–1.27; $p = 0.018$) emerged as independent predictors of adverse in-hospital events. For each increase in RDW%, the odds of an AEs increased by 48%; for each $1 \times 10^9/L$ increase of leukocyte count, the odds increased by 19.5%; and for each unit increase in NLR ratio, the odds increased by 13.8%. CRP, neu-

Table 2. Baseline hematologic parameters in patients stratified by presence/absence of adverse events.

Parameters	Total cohort n=88	Referent values	With AE n=13	Without AE n=75	p
RDW (%)	14.53± 2.27	11.0-16.0	16.05± 2.81	14.27± 2.07	0,008
Leukocytes (10 ⁹ /L)	10.59± 3.89	4.0-9.0	13.56± 5.76	10.07± 3.25	0,002
Lymphocyte (10 ⁹ /L)	2.56± 5.02	0.5-5.0	3.66± 8.85	2.37± 4.09	0,071
Neutrophils (10 ⁹ /L)	8.75± 6.07	2,4-7,5	11.24± 5.36	8.31± 6.11	0,031
Platelets (10 ⁹ /L)	234.81± 100.34	150-450	277.46 ± 113.84	227.46± 96.75	0,097
CRP (mg/L)	48.80± 56.97	< 6	80.74± 90.97	43.26± 47.57	0,028
HsTroponin (ng/ml)	136.63± 266.55	<15.6	194.16± 382.61	126.65± 243.12	0,402
NLR	6.55± 5.94	1-2*	11.51± 7.07	5.68± 5.30	0,001
PLR	171.36± 114.35	< 150*	262.57± 134.48	155.33± 103.39	0,001
RCI	2.60± 2.61	-	3.48± 3.65	2.45± 2.38	0,194

RDW=red blood distribution width, CRP= C-reactive protein, NLR=neutrophil-lymphocyte ratio, PLR=platelet-lymphocyte ratio, RCI= red cell index.

*Referent values are from the literature [15]

Table 3. Results from stepwise logistic regression using AEs as the dependent variable.

Variables	B	S.E.	Wald	Sig.	Exp (B)	95% CI for Exp (B)	
						lower	higher
RDW%	0.392	0.143	7.503	0.006	1.480	1.118	1.959
Leukocytes	0.178	0.083	4.601	0.032	1.195	1.015	1.406
NLR	0.129	0.055	5.587	0.018	1.138	1.022	1.267

trophils and PLR ratio lost predictive power in the final step of analysis.

ROC curve analysis (Figure 1) showed that NLR had the highest area under the curve (AUC) among individual markers (0.736; 95% CI 0.543–0.929; $p = 0.007$), followed by RDW% (0.722; 95% CI 0.573–0.871; $p = 0.011$) and leukocyte count (0.680; 95% CI 0.487–0.872; $p = 0.039$). The cutoff values were as follows: for RDW% was 15,1% (sensitivity 69,2%, specificity 68,4%), for leukocyte count was $13.4 \times 10^9/L$ (sensitivity 61.5%, specificity 70.7%) and for the NLR ratio was 9.3 (sensitivity 69.2%, specificity 76.0%).

Although each marker alone showed moderate predictive accuracy, their combi-

nation significantly improved discrimination (Figure 2), as reflected by an AUC of 0.880, indicating excellent predictive ability for AEs and suggesting potential utility for early risk stratification.

DISCUSSION

In this study, we evaluated the prognostic value of inflammatory and hematologic biomarkers in predicting in-hospital adverse events (AEs) among patients with acute PE. Our main findings demonstrate that RDW%, leukocyte count, and NLR ratio, when mea-

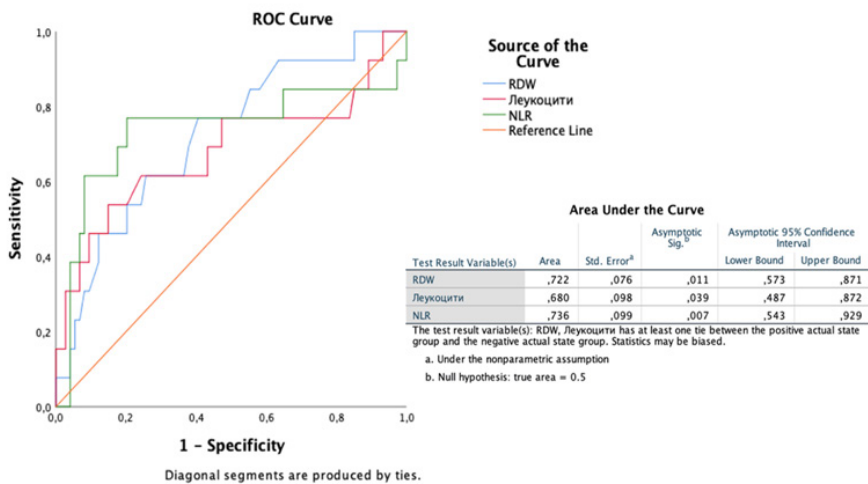


Figure 1. ROC curves for RDW, leukocytes, and NLR

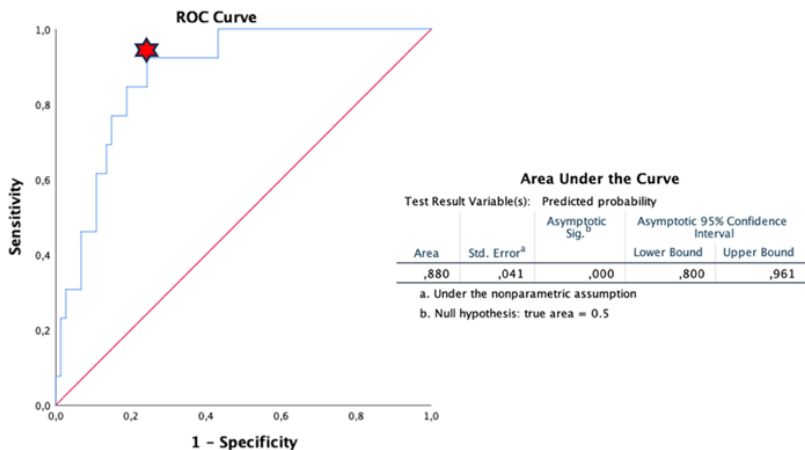


Figure 2. ROC analysis of RDW%, leukocytes, and NLR as a combined model for predicting AEs
The red star indicates the point of optimal sensitivity and specificity on the curve.

sured at admission, were independent predictors of adverse in-hospital outcomes, whereas PLR ratio and CRP were significantly higher in patients with AEs but lost predictive power in multivariable analysis.

RDW as a prognostic marker

Among all tested biomarkers, RDW showed the strongest association with AEs and emerged as the most powerful independent predictor in multivariable logistic regression (OR 1.480, 95% CI 1.118–1.959, $p = 0.006$). ROC analysis confirmed good discriminative performance (AUC = 0.722), with an optimal cut-off of 15.1% providing 69.2% sensitivity and 68.4% specificity. These results support previous reports that elevated RDW is a marker of poor prognosis in patients with acute PE [7,12–14] as well as recent studies by Ozcan et al. [16] and particularly Slajus et al. [17], who demonstrated that the predictive ability of RDW% was superior to sPESI alone, improving the accuracy of the composite sPESI model. Although the exact pathophysiological mechanism behind anisocytosis in PE is not fully understood, it may involve impaired erythropoiesis and increased oxidative stress during acute inflammation and hypoxemia, leading to worse outcome.

Leukocyte count, NLR and PLR in risk stratification

Leukocytosis and neutrophil predominance have long been recognized as indicators of systemic inflammation and tissue injury [5,6]. Both markers reflect the systemic inflammatory response to thromboembolic events, which can exacerbate pulmonary vascular injury and right ventricular strain [3,11]. In our study, both leukocyte count and NLR ratio were significantly associated with adverse events, and the latter showed the highest area under the ROC curve among individual markers.

A large study performed by Venetz et al. [5] on 14,228 patients with acute PE demonstrated independent prognostic value of leukocyte count, with a U-curve risk for 30-day mortality. Similarly, in the multicenter, international REPER registry, total WBC count was markedly elevated in non-survivors with acute PE and improved risk stratification in intermediate–high-risk patients within the ESC risk classification [18].

In our research, NLR as a discriminator between patients with and without AEs had an acceptable AUC (0.736). The sensitivity of NLR as a predictor of AEs was 69.2%, with a specificity of 76.0% for a cutoff value of 9.3 (95%CI 0.543–0.929, $p=0.007$). This aligns with the substantial evidence indicating that the NLR ratio is a reliable predictor of mortality and right ventricular dysfunction in acute PE [12–14, 16]. A meta-analysis by Wang et al. [13] identified a significant correlation between NLR and short-term (in-hospital and 30 days) mortality, which was confirmed by Tang et al. [14] meta-analysis for both short- and long-term mortality. In the study by Babes et al. [11], NLR was the strongest hematologic predictor of in-hospital complications, followed by neutrophils, lymphocytes, and leukocytes. NLR was also significantly correlated with PESI and sPESI scores and improved their discriminative power and sensitivity for predicting adverse outcomes.

Similar findings were reported in a recent study by Yurtseven et al. [19], where NLR demonstrated the strongest prognostic value, correlating with multiple clinical outcomes. Although PLR has also been reported as an independent predictor for short- and long-term mortality in the literature [13], we could not confirm this in our study. In multivariable analysis, PLR lost its independent predictive value, which is consistent with findings by Ma et al. [20] and the meta-analysis by Tang et al [14].

Combined predictive model

Importantly, combining RDW, leukocytes, and NLR into a single predictive model substantially improved the prognostic accuracy, achieving an AUC of 0.880, which indicates excellent discrimination. This suggests that integrating multiple hematologic parameters into risk assessment models could enhance early risk stratification, particularly in intermediate-risk patients, where current scoring systems such as sPESI and the ESC classification may be insufficient [2,7, 9–11].

Role of CRP and prognosis

Although CRP was significantly higher in patients with AEs in our study, it did not retain independent predictive power in the final regression model, likely due to overlapping prognostic

information with NLR and leukocytes. This finding indicates that while inflammation contributes to disease severity, not all inflammatory markers provide incremental prognostic information beyond the hematologic indices. It also supports the previous reports suggesting that hematologic ratios such as NLR may outperform single inflammatory markers for risk prediction [6, 11].

LIMITATIONS

This study has several limitations. First, its retrospective single-center design may limit generalizability. Second, the sample size was relatively small, and the number of adverse events was limited, which may have influenced statistical power and precluded detailed subgroup analyses. Third, inflammatory markers were measured only at admission, without serial assessment, which could have provided additional prognostic information. Finally, our findings should be validated in larger prospective cohorts.

CONCLUSION

RDW, leukocyte count, and NLR are significant independent predictors of in-hospital adverse events in patients with acute PE. RDW >15.1%, leukocytes >13.4 ×10⁹/L, and NLR >9.3 identify patients at higher risk. A combined model incorporated these markers and provides excellent predictive accuracy. Given their simplicity and availability, these biomarkers could complement established risk scores to improve early risk stratification. Future research should focus on integrating hematologic indices into existing risk models and validating combined approaches in multicenter studies. Exploring the dynamic changes of these markers during hospitalization may further improve early prognostic accuracy.

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Резиме

СТРАТИФИКАЦИЈА НА РАНИОТ РИЗИК КАЈ АКУТНА БЕЛОДРОБНА ЕМБОЛИЈА СО КОРИСТЕЊЕ ВОСПАЛИТЕЛНИ И ХЕМАТОЛОШКИ БИОМАРКЕРИ

Беким Поцеста, Лидија Попоска, Елиф Врајнко,
Томислав Коњановски, Бошевски Маријан, Љубица Георгиевска-Исмаил

Универзитетска клиника за кардиологија, Медицински факултет, Универзитет „Св. Кирил и Методиј“ во Скопје, РС Македонија

Вовед: Точната рана стратификација на ризикот кај акутната белодробна емболија (БЕ) останува значаен клинички предизвик, особено кај пациентите со интермедијарен ризик. Лесно достапните хематолошки и инфламаторни маркери би можеле да обезбедат дополнителна прогностичка вредност и да го подобрат клиничкото одлучување.

Цел: Да се процени предиктивната улога на хематолошките и на инфламаторните маркери за појава на интрахоспитални несакани збиднувања кај пациенти со акутна БЕ.

Методи: Спроведена е ретроспективна студија на 88 пациенти со дијагностицирана акутна БЕ во периодот 2023–2024 година. Анализирани беа клиничките, ехокардиографските и лабораториските податоци, вклучувајќи: ширина на дистрибуцијата на еритроцитите (RDW), број на леукоцитите (WBC), однос неутрофили-лимфоцитите (NLR), однос тромбоцити-лимфоцити (PLR) и Ц-реактивен протеин (CRP). Несаканите збиднувања (НЕЗ) беа дефинирани како хемодинамска нестабилност, срцев застој или смрт за време на хоспитализацијата. Со логистичка регресија беа идентификувани независни предиктори, а дискриминативната способност беше проценета со ROC-криви.

Резултати: Интрахоспитални НЕЗ беа забележани кај 13 пациенти (14,8 %), од кои четворица починаа. Пациентите со НЕЗ имаа значајно повисоки вредности на RDW ($p = 0,008$), WBC ($p = 0,002$), NLR ($p = 0,001$), PLR ($p = 0,001$) и CRP ($p = 0,028$). Мултиваријантната анализа ги идентификува RDW (OR 1,48; 95% CI 1,12–1,96; $p = 0,006$), WBC (OR 1,20; 95% CI 1,02–1,41; $p = 0,032$) и NLR (OR 1,14; 95% CI 1,02–1,27; $p = 0,018$) како независни предиктори на интрахоспитална смртност. ROC-анализата покажа подобрена дискриминација при комбинирана употреба на овие маркери (AUC 0,880).

Заклучок: RDW, бројот на леукоцити и NLR се независни предиктори за интрахоспитални несакани збиднувања кај акутна БЕ. Комбинираното користење на овие маркери може да ја подобри раната стратификација на ризик и да ја поттикне индивидуализираната терапевтска стратегија.

Клучни зборови: белодробна емболија, Ц-реактивен протеин, прогностички маркери, интрахоспитална смртност