Neutrophil-Lymphocyte Ratio and Platelet-Lymphocyte Ratio as Biomarkers for Risk Stratification and Predictors of 90-day Mortality in Acute Pulmonary Embolism

Dr Kowsika R[1], Dr Muthukumaran L[2], Dr Sridhar R[3], Dr Meenakshi N[4], Dr Vigneshwaran J[5], Dr Ramya Priya Anbalagan[6], Dr Anudeep Aveleli[7]

Affiiliations:
1- Postgraduate, Department of Respiratory Medicine, Chettinad Hospital and Research Institute; Orcid id: http://orcid.org/0009-0004-8084-2415
2- Associate Professor, Department of Respiratory Medicine, Chettinad Hospital and Research Institute; Orcid id: https://orcid.org/0000-0001-6400-6733*
3- Professor, Department of Respiratory Medicine, Chettinad Hospital and Research Institute; Orcid id: https://orcid.org/0000-0001-7661-2351
4- Professor & HOD, Department of Respiratory Medicine, Chettinad Hospital and Research Institute; Orcid id: https://orcid.org/0000-0002-8743-149X
5- Professor, Department of Internal Medicine, Chettinad Hospital and Research Institute Orcid id: https://orcid.org/0000-0002-1509-3137
6- Senior Resident, Department of Respiratory Medicine, Chettinad Hospital and Research Institute, Orcid id: https://orcid.org/0000-0003-4977-6080
7- Postgraduate, Department of Cardiology, Chettinad Hospital and Research Institute; Orcid id: https://orcid.org/0009-0003-5677-7249

*Corresponding Author - Dr Muthukumaran, Associate Professor, Department of Respiratory Medicine, Chettinad Hospital and Research Institute; Orcid id: https://orcid.org/0000-0001-6400-6733

KEYWORDS
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ABSTRACT

Background: Acute Pulmonary Embolism (APE) is a critical cardiovascular emergency requiring timely risk stratification. Neutrophil-to-Lymphocyte Ratio (NLR) and Platelet-to-Lymphocyte Ratio (PLR) are emerging inflammatory biomarkers that may enhance existing risk models, such as the European Society of Cardiology (ESC) guidelines.

Objectives: To assess the prognostic value of NLR and PLR in predicting 90-day mortality in APE patients and their potential role in improving ESC risk stratification.

Methods: A retrospective cohort study of 42 APE patients at Chettinad Hospital (2021-2024) was conducted. Clinical, laboratory, and radiological data were analyzed. Statistical tests, including logistic regression, were used to evaluate associations between NLR, PLR, and 90-day mortality.

Results: The study included 42 patients (57.1% male, mean age 54.19 ± 15.98 years). Dyspnea (85.7%) and palpitations (54.8%) were the most common symptoms. Elevated NLR and PLR were significantly associated with higher mortality risk. Malignancy ($p < 0.001$) and right ventricular dysfunction ($p < 0.001$) were independent mortality predictors. Integrating NLR and PLR into the ESC model improved predictive accuracy with AUC of 0.848 as compared to only ESC stratification with AUC of 0.771.

Conclusion: NLR and PLR are valuable biomarkers for APE risk stratification and mortality prediction. Their inclusion in ESC guidelines may refine prognostication, facilitating timely interventions. Prospective multicenter studies are needed for validation.

INTRODUCTION:

Acute pulmonary embolism (APE) is a critical cardiovascular emergency characterized by occlusion of pulmonary arteries, often resulting from thromboembolic events developing in the deep veins of the lower extremities [1]. It is associated with high morbidity and mortality rates, in particular when diagnosis and treatment are delayed [2]. Early risk stratification is crucial for optimizing patient outcomes, as it helps identify individuals at high risk of adverse events who may benefit from aggressive therapeutic interventions [3]. Recently, hematological biomarkers like neutrophil-to-lymphocyte ratio (NLR) and platelet-to-lymphocyte ratio (PLR) have gained growing interest as prognostic indicators in APE [4]. These biomarkers are derived from routine complete blood count (CBC) tests, making them cost-effective and readily accessible for clinical use [5].

NLR, calculated as the ratio of absolute neutrophil count to absolute lymphocyte count, is a well-established marker of systemic inflammation and stress [6]. In the context of APE, elevated NLR levels have been associated with increased disease severity, right ventricular dysfunction, and higher mortality rates [7]. Neutrophils play a key role in the inflammatory response triggered by pulmonary embolism, contributing to endothelial damage and microvascular obstruction activating SIRS leading to neutrophilia, while lymphocytes are involved in modulating immune responses at the time of tissue injury- altering the T4/T8 lymphocyte ratio causing lymphocytopenia [8]. An imbalance in this ratio reflects a heightened inflammatory state and impaired immune regulation, which are hallmarks of poor prognosis in APE [9]. Studies have demonstrated that NLR is an independent predictor of short- and long-term mortality in patients with APE, making it a valuable tool for risk assessment [10][11].

Similarly, PLR, which is the ratio of platelet count to lymphocyte count, has gained attention as a prognostic marker in various cardiovascular and thromboembolic disorders [11]. Platelets are not only involved in thrombus formation but also contribute to inflammation and vascular remodelling, processes that are central to the pathophysiology of APE [12]. Elevated PLR levels have been linked to increased pulmonary artery pressure, right ventricular strain, and adverse clinical outcomes in APE patients [13]. The combination of thrombocytosis and lymphopenia, as reflected by a high PLR, indicates a prothrombotic and proinflammatory state, which is associated with worse prognosis [14]. Some recent researches suggest that PLR may complement NLR in risk stratification, providing additional prognostic information that can guide therapeutic decision-making, however they have not been studied widely like other inflammatory biomarkers in venous thromboembolism [15].

The integration of NLR and PLR into existing ESC risk stratification model has the potential to enhance the mortality prediction [16]. These biomarkers offer a simple, non-invasive, and cost-effective approach to identifying high-risk patients who may require intensive monitoring and advanced therapies, such as thrombolysis or embolectomy [17].

Thus, our study aimed to assess the association and correlation between Acute Pulmonary Embolism and inflammatory biomarkers such as NLR, PLR, Troponin I and D-Dimer, and eventually as tools to enhance the predictive accuracy of ESC Risk Stratification in 90-day mortality.

MATERIALS AND METHODS:

Our study was a single-centered, medical records based, retrospective cohort study carried out in the Departments of Respiratory Medicine, Internal Medicine and Cardiology of Chettinad Hospital and Research Institute, Chennai. After obtaining approval from the hospital's review board, we included 42 patients admitted inhospital with the definitive diagnosis of Acute Pulmonary Thromboembolism between the time period 2021-2024 and collected their data. We excluded patients with unavailable lab investigations/ clinical data. Information about the

presenting vitals, clinical presentation, co-morbidities, risk factors, ECG, 2D ECHO, lab results (Complete Blood Count, D-dimer, Troponin I), radiological investigations (USG venous doppler, Computed Tomography- Pulmonary Angiography) were collected. The risk stratification standard and treatment protocol for Acute Pulmonary Embolism were in accordance with European Society of Cardiology (ESC) guidelines. Patients classified as high risk and intermediate high risk were thrombolysed while those under intermediate low risk and low risk were treated with anti-coagulation. Patients and their attendants were contacted via the hospital database to assess their status 90 days post-admission.

Statistical analysis was performed using SPSS Version 17 (Microsoft Windows). Since the data lacked normal distribution, both parametric and non-parametric tests were applied. Descriptive statistics were presented as numbers, percentages, mean, and standard deviation (SD). For continuous variables, Kruskal-Wallis test or one-way ANOVA with Tukey HSD post hoc test was used, while comparisons between two groups utilized independent t-test or Mann-Whitney test. Categorical variables were analyzed using the chi-square test, and correlations were assessed via Pearson's correlation coefficient and Kendall's tau-b analysis. Statistical significance was set at $p < 0.05$.

RESULTS:

Baseline Characteristics:

42 patients were included in the study. 18 patients were female (42.9%) and 24 were male (57.1%) with mean age being 54.19 ± 15.98 years.

Table 1: Vitals at admission:

Parameter	Mean ± SD
O_2 Saturation (% at RA)	90.47 ± 4.79
RR (/min)	25.95 ± 3.76
HR (/min)	106.36 ± 19.01
SBP (mmhg)	119 ± 20.7
DBP (mmhg)	74.76 ± 10.17

Table 2: Clinical Presentation at admission:

Symptom	Percentage (%)	n
Dyspnea	85.7%	36
Chest Pain	33.3%	14
Cough	28.6%	12
Hemoptysis	14.3%	6
Giddiness	16.7%	7
Loss of Consciousness	4.8%	2
Palpitations	54.8%	23
Lower Limb Pain	28.6%	12
Back Pain	7.1%	3

Table 3: Co-morbidities and Risk Factors:

Condition/Risk Factor	Percentage (%)	n
Systemic Hypertension	50.0%	21
Type 2 Diabetes Mellitus	31.0%	13
Smokers	52.4%	22
Malignancy	16.7%	7
Immobilisation	26.2%	11
Previous Pulmonary Thromboembolism (PTE)	9.5%	4
Previous Deep Vein Thrombosis (DVT)	4.8%	2
Coronary Artery Disease	16.7%	7
Obesity	7.1%	3
Post Surgery	9.5%	4
Trauma	9.5%	4
Oral Contraceptive Pill (OCP) Intake	2.4%	1
Pregnancy	2.4%	1
Polycythemia	7.1%	3
Factor V Mutations	9.5%	4
Protein C, S Deficiency	9.5%	3

Table 4: Well's score: (Three Tier Model)

Risk Category	Percentage (%)	N
Low Risk	9.5%	4
Moderate Risk	54.8%	23
High Risk	35.7%	15

Electrocardiography and Imaging findings in patients with Acute Pulmonary Embolism:

Table 4: Electrocardiographic Assessment:

Parameter	Percentage (%)	N
Rhythm Atrial Fibrillation	7.1	3
Sinus Tachycardia	76.1	32
Non-specific ST Changes	19	8
S1Q3T3 Pattern	4.7	2
Incomplete Right Bundle Branch Block (RBBB)	4.7	2
Complete Right Bundle Branch Block (RBBB)	2.3	1
Right Axis Deviation	30.9	13

Table 5: Echocardiographic Assessment:

Parameter	Percentage (%)	N
Right Atrium/Right Ventricle Dilatation	59.5	25
Right Atrial Thrombus	4.7	2
Pulmonary Hypertension - Mild	26.2	11
Pulmonary Hypertension - Moderate	19	8
Pulmonary Hypertension - Severe	7.1	3
Tricuspid Regurgitation - Trace	9.5	4
Tricuspid Regurgitation - Grade I	28.5	12
Tricuspid Regurgitation - Grade II	57.1	24

Table 6: USG Lower Limb Doppler:

Parameter	Percentage (%)	N
DVT Present	61.9	26
DVT Absent	38.1	16

Table 7: Computed Tomography Pulmonary Angiography

Parameter	Percentage (%)	N
Central Thrombi	33.3	14
Peripheral Thrombi	66.7	28
Bilateral Involvement	57.1	24
Pulmonary Trunk	7.1	3

Table 8: Laboratory Investigations

Parameter	Value
D-Dimer	2375.38 ± 1656.56
Troponin-I	215.35 ± 142.18
NLR	5.69 ± 4.28
PLR	196.46 ± 118.72

Table 9: ESC Risk Stratification

Parameter	Value (%)	N
High Risk	33.3	4
Intermediate High Risk	66.7	17
Intermediate Low Risk	57.1	10
Low Risk	7.1	11

Table 10: Inhospital Management in Patients with Acute Pulmonary Embolism:

Parameter	Percentage (%)	N
Thrombolysis	33.3	14
- Streptokinase	28.5	12
- Alteplase	4.7	2
Anti-Coagulants	61.9	26
- Heparin	61.5	16
- Enoxaparin	38.4	10
Mechanical Thrombectomy	4.8	2

Dyspnea (85.7%) and palpitations (54.8%) were the most frequently observed clinical symptoms at presentation. The mean systolic and diastolic blood pressures were 119 ± 20.7 mmHg and 74.76 ± 10.17 mmHg, respectively. The average heart rate, respiratory rate, and oxygen saturation were 106.36 ± 19.01 bpm, 25.95 ± 3.76 breaths/min, and 90.47 ± 4.79%, respectively.

Smoking (52.4%) followed by Immobilisation (26.2%) was the most common risk factor. Systemic Hypertension (50%) was the most common comorbidity. USG Doppler studies showed DVT in 26 cases (61.9%). The most common ECG finding was Sinus Tachycardia (76.1%) followed by non-specific ST-T changes (19%). The most common Echocardiography finding was Tricuspid Regurgitation (95.2%) followed by RA/RV dilatation (59.5%). On CT-Pulmonary Angiography, Central Thrombi was noted in 33.3%, Peripheral Thrombi in 66.7% while bilateral involvement was noted in 57.1% and Pulmonary Trunk involvement was noted in 7.1%. Pulmonary Hypertension was noted in 22 patients (52.3%). Thrombolysis was carried out in 33.3% while the rest were treated with anti-coagulants (of which 2 cases had contra-indication to thrombolysis). Patients and their attendants were contacted via the hospital database to assess their status 90 days post-admission- Mortality was seen in 8 patients (19%) while survival noted in 34 patients (81%).

Table 11: P-values from Statistical Analysis

Test/Comparison	p-value
Risk Stratification (ESC) vs. 90-day Mortality	0.079
Malignancy vs. 90-day Mortality	**0.005**
Immobilisation vs. 90-day Mortality	0.932
Previous PTE vs. 90-day Mortality	0.308
Previous DVT vs. 90-day Mortality	0.482
Obesity vs. 90-day Mortality	0.513
Post Surgery vs. 90-day Mortality	0.75
Trauma vs. 90-day Mortality	0.097
OCP Intake vs. 90-day Mortality	0.623
Pregnancy vs. 90-day Mortality	0.623
Polycythemia vs. 90-day Mortality	0.383

Factor V Mutation vs. 90-day Mortality	0.308
Wells' Score vs. 90-day Mortality	**0.033**
2D-ECHO vs. 90-day Mortality	0.176
RV Dysfunction vs. 90-day Mortality	**0.009**
Pulmonary Hypertension vs. 90-day Mortality	0.726
Thrombosis Location vs. 90-day Mortality	0.266

Table 12:

Test/Comparison	**p-value**
D-Dimer vs. Risk Stratification (ESC)	**0.050**
Trop-I vs. Risk Stratification (ESC)	**0.001**
NLR vs. Risk Stratification (ESC)	0.292
PLR vs. Risk Stratification (ESC)	0.081
D-Dimer vs. 90-day Mortality	0.116
Trop-I vs. 90-day Mortality	0.018
NLR vs. 90-day Mortality	0.116
PLR vs. 90-day Mortality	**0.018**

A logistic regression model was built to evaluate the potential risk factors associated with 90-day mortality and ESC Risk Stratification (dependant variables) including NLR, PLR (Table 11 & 12). Malignancy and RV Dysfunction were found to have significant association with 90-day mortality (p-value <0.001). Well's score, Thrombi location on CT-PA and RV dysfunction were found to have significant association with ESC Risk Stratification criteria. ECHO findings and Troponin-I were significantly associated with CT Severity index (p-value<0.05). There is a moderate and statistically significant positive correlation ($p < 0.05$) between D-Dimer and PLR in DVT Present patients (Figure 1).

Figure 1: Correlation between D-Dimer and PLR in Patients with DVT:

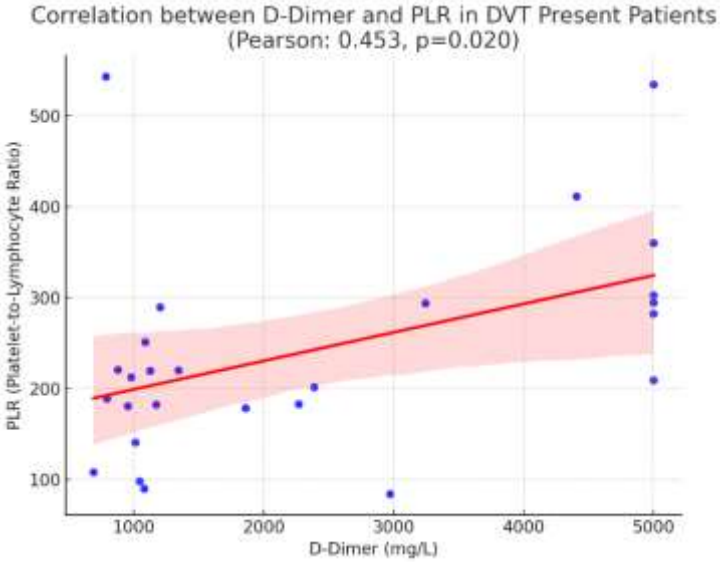
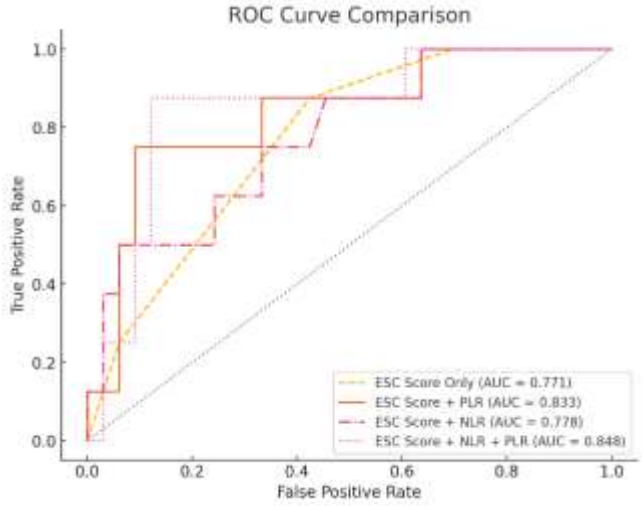


Figure 2: ROC Analysis between ESC with NLR, PLR, NLR+PLR



Receiver Operating Characteristic (ROC) Curve Analysis

ROC analysis (Figure 2) was conducted to assess the predictive performance of the ESC risk stratification score alone and with PLR and NLR. The AUC for ESC alone was **0.771**, while adding PLR increased it to **0.833**, and adding NLR increased it to **0.778**. The highest AUC (**0.848**) was observed when both PLR and NLR were included with ESC, indicating the best predictive capability. These findings suggest that integrating both biomarkers with ESC stratification significantly enhances prognostic accuracy, supporting their potential role in risk assessment and clinical decision-making.

DISCUSSION:

Our study highlights the potential of neutrophil-to-lymphocyte ratio (NLR) and platelet-to-lymphocyte ratio (PLR) as effective biomarkers for risk stratification and predicting 90-day mortality in patients with acute pulmonary embolism (APE). The results align with existing literature, demonstrating that elevated NLR and PLR levels are associated with poorer outcomes, including higher mortality and increased disease severity (Kayrak et al., 2014; Kundi et al., 2019). These biomarkers, easily derived from routine complete blood count (CBC) tests, provide a cost-effective and accessible method to enhance current risk stratification models, such as those outlined by the European Society of Cardiology (ESC) (Konstantinides et al., 2019).

NLR, a known marker of inflammation, was significantly elevated in our patient cohort, reflecting the intense inflammatory response and immune dysregulation often seen in APE. Neutrophils contribute to endothelial damage and microvascular obstruction, while lymphocytopenia indicates impaired immune response (Zahorec, 2001; Sincer et al., 2019). Our findings support previous studies that have identified NLR as an independent predictor of both short and long-term mortality in APE patients (Akgüllü et al., 2015; Li et al., 2020). Similarly, PLR, which reflects a prothrombotic and proinflammatory state, was also elevated in our study. Elevated PLR levels have been linked to increased pulmonary artery pressure, right ventricular strain, and adverse clinical outcomes, further underscoring its value as a prognostic tool (Çetin et al., 2018; Öz et al., 2019).

The inclusion of PLR in the ESC risk stratification model improved its predictive accuracy for 90-day mortality, suggesting that PLR can complement existing risk assessment tools (Liao et al., 2021). This is particularly important for identifying high-risk patients who may benefit from aggressive interventions, such as thrombolysis or embolectomy (Jiménez et al., 2010; Konstantinides et al., 2019). Our study also identified malignancy and right ventricular

dysfunction as significant predictors of 90-day mortality, consistent with prior research (Barco et al., 2019; Çetin et al., 2018). These findings highlight the importance of combining clinical data with biomarker analysis to optimize risk stratification and patient outcomes.
This study has certain limitations. Being a single-center study with a small sample size, the findings may not be generalizable to broader populations. The retrospective design carries inherent risks of selection bias and missing data. Additionally, the study focuses on 90-day mortality, limiting insight into long-term prognostic implications. Follow-up NLR and PLR could have helped predict the outcome better. While NLR and PLR showed significant predictive value, standardized cut-off values remain undefined. Future large-scale, multicenter, prospective studies are needed to validate these findings (Kundi et al., 2019; Aujesky et al., 2021). Additionally, investigating the interplay between NLR, PLR, and other inflammatory biomarkers could provide deeper insights into the pathophysiology of APE.

Conclusion:

NLR and PLR can be used as tools for risk stratification, providing dynamic insights into patient outcomes [18]. Despite their promise, further research is needed to establish standardized cutoff values and validate their utility across diverse patient populations [19].
In conclusion, NLR and PLR incorporated with ESC have emerged as valuable indicators for mortality prediction in APE, offering insights into the inflammatory and thrombotic processes underlying the disease [20]. Their incorporation into clinical practice may improve risk stratification, facilitate timely interventions, and ultimately enhance patient outcomes [21]. This review examines the existing evidence on NLR and PLR as markers of risk stratification and mortality prediction in APE, emphasizing their clinical significance and potential applications in routine practice.

Authors Contribution:

Conceptualization : Dr Ramya Priya Anbalagan, Dr Vigneshwaran J, Dr Sridhar R, Dr Kowsika R
Methodology : Dr Muthukumaran L, Dr Meenakshi N, Dr Ramya Priya Anbalagan, Dr Kowsika R
Data Curation : Dr Kowsika R, Dr Anudeep Aveleli
Formal Analysis : Dr Kowsika R, Dr Vigneshwaran J
Writing – Original Draft : Dr Kowsika R, Dr Muthukumaran L, Dr Sridhar R
Writing – Review & Editing : Dr Kowsika R, Dr Muthukumaran L, Dr Sridhar R
Supervision : Dr Sridhar R, Dr Meenakshi N

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