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Validation of the PEACE score for predicting abnormal echocardiographic findings in pulmonary embolism patients

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Abstract

Background Pulmonary embolism (PE) is a life-threatening condition requiring rapid risk stratification for optimal management. The Pulmonary Embolism Advanced Cardiac Evaluation (PEACE) Score is a novel tool integrating clinical, laboratory, and echocardiographic parameters to assess disease severity. This study aimed to evaluate the correlation between PEACE Score and echocardiographic abnormalities in PE patients, and to determine its effectiveness as a rapid risk assessment tool in emergency settings.

Methods Between June 2020 and June 2024, 120 patients were prospectively screened and enrolled in the study after being diagnosed with pulmonary embolism via CT angiography in the emergency department. Patients were categorized into three groups according to PEACE score as low risk (< 3 points, $n=42$), intermediate risk (3–5 points, $n=52$) and high risk (> 5 points, $n=26$). Echocardiographic findings were not used for stratification but rather analyzed as outcome variables to assess the discriminative validity of the PEACE Score. Demographic data, laboratory findings and echocardiographic parameters were recorded. Patients were followed up for at least 1 year. Follow-up from 3 months to 6 months was evaluated and mortality rates at the end of 1 year were determined.

Results PEACE Score was strongly correlated with echocardiographic abnormalities ($r=0.685$, $p<0.001$) and inflammatory markers, including CRP ($r=0.524$, $p<0.001$). The PEACE Score had the highest diagnostic value for predicting echocardiographic abnormalities, with an AUC of 0.82 (95% CI: 0.74–0.90, $p<0.001$). Specifically, in predicting right ventricular dysfunction, the PEACE Score achieved an AUC of 0.85 (95% CI: 0.77–0.93, $p<0.001$). A cutoff of > 5 points showed a sensitivity of 84.6% and specificity of 79.2% for detecting severe echocardiographic abnormalities. One-year survival rates were 45% in the high-risk group, 65% in the intermediate-risk group, and 85% in the low-risk group. Kaplan-Meier analysis confirmed significant differences in survival among risk groups ($p<0.001$).

Conclusion The PEACE Score demonstrated a strong association with echocardiographic abnormalities and patient survival in emergency department PE cases. These findings suggest that PEACE may serve as a valuable tool for rapid risk stratification, aiding emergency physicians in early clinical decision-making. Specifically, high PEACE Scores were associated with a greater need for thrombolytic therapy and ICU admission, suggesting its potential utility in guiding treatment escalation and resource allocation in critically ill PE patients.

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Clinical trial number Not applicable.

Keywords Echocardiographic findings, Emergency medicine, PEACE score, Pulmonary embolism

Background

Pulmonary embolism (PE) is a serious clinical form of venous thromboembolism (VTE) and is a medical emergency that can lead to sudden cardiopulmonary failure and death [1]. Early risk stratification is crucial for guiding treatment decisions and improving patient outcomes. Currently, PE is diagnosed by imaging methods such as computed tomography pulmonary angiography (CTPA) performed on clinical suspicion [2]. However, echocardiographic findings play an important role in determining the prognosis of PE patients, especially findings such as right ventricular dysfunction and pulmonary hypertension which play a critical role in assessing prognosis and disease severity in PE patients [3]. In recent years, prognostic scores such as the PEACE score for risk assessment of PE patients have been increasingly accepted in clinical practice [4]. The clinical applicability of the PEACE scoring system was evaluated in its ability to predict echocardiographic abnormalities among patients with pulmonary embolism in the emergency department. Although echocardiography is a key tool for assessing right ventricular (RV) dysfunction in pulmonary embolism (PE), its accessibility and interpretation may pose challenges in certain emergency settings. The PEACE score, integrating Troponin I and NT-proBNP, provides an early, biomarker-based risk stratification tool that can complement echocardiography, aiding in timely decision-making in resource-limited settings.

Despite the availability of various clinical risk scores of PE patients in the current literature, there is a distinct lack of information on the effectiveness of these scores in predicting echocardiographic findings [5]. The Pulmonary Embolism Exclusion Criteria (PERC) are designed to exclude PE in low-risk patients without the need for further testing and have been shown to be effective in reducing unnecessary imaging in the emergency department setting [6]. The Pulmonary Embolism Severity Index (PESI) is a prognostic tool that categorizes patients into risk classes based on 30-day mortality and is used to determine the need for more intensive treatment of high-risk patients [4]. The Pulmonary Embolism Advanced Risk Classification (PEARS) helps guide treatment decisions in intermediate- and high-risk PE patients by integrating clinical and imaging data [7].

Comparison of PERC, PESI and PEARS scores allows evaluation of their impact in different clinical scenarios. PERC allows avoiding unnecessary diagnostic tests in low-risk patients, while PESI is a reliable tool to estimate patients' mortality risk. PEARS is useful in more complex risk stratification, especially because it includes biomarkers and echocardiographic findings. Furthermore, in patients diagnosed with PE, significant differences can be observed when compared with their previous ECGs. Common ECG changes in acute PE include sinus tachycardia, right axis shift, right bundle branch block (complete or incomplete) and the classic S1Q3T3 pattern. In addition, negative T waves and ST segment changes in leads V1-V3 reflect right ventricular loading and increased biomarker levels, indicating the hemodynamic burden of the disease [7].

This study aims to evaluate the correlation between the PEACE Score and echocardiographic abnormalities in PE patients and determine its effectiveness in predicting prognosis and guiding clinical decision-making in the emergency department. Our hypothesis is that higher PEACE scores are associated with more frequent echocardiographic abnormalities and worse clinical outcomes. By validating this scoring system, we aim to improve early risk stratification and optimize treatment strategies for PE patients in emergency care settings.

Materials and methods

Study design and ethical approval

This was a prospective diagnostic validation study evaluating the effectiveness of the PEACE Score in predicting echocardiographic findings in patients diagnosed with pulmonary embolism (PE) in the emergency department. Ethical approval was obtained from the Gaziantep Islam Science and Technology University Non-Interventional Ethical Committee with decision number 484.40.20. The study was conducted in accordance with the principles of the Declaration of Helsinki and written informed consent was obtained from all participants. Within the scope of the study protocol, patients' demographic data, risk factors, laboratory and imaging results, and clinical follow-up information were prospectively recorded.

Study population

Between June 2020 and June 2024, 120 patients over 18 years of age with a diagnosis of acute pulmonary embolism confirmed by computed tomography pulmonary angiography who presented to emergency departments of Gaziantep City Hospital and Gaziantep Dr. Ersin Arslan Training and Research Hospital were included in the study. A priori sample size calculation using G-Power 3.1.9.7 indicated that at least 64 patients were needed to achieve 80% power ($\alpha = 0.05$, effect size = 0.3, one-tailed test). The sample size was determined through a power analysis with a 95% confidence interval and 80% power, based on similar studies in the literature [8]. Patients

were categorized into three groups according to PEACE score as low risk (< 3 points, $n = 42$), intermediate risk (3–5 points, $n = 52$) and high risk (> 5 points, $n = 26$). Echocardiographic findings were not used for stratification but rather analyzed as outcome variables to assess the discriminative validity of the PEACE Score. Patients with severe hepatic failure, chronic renal failure, a history of active malignancy, or those who were pregnant were excluded. Additionally, patients who could not obtain appropriate image quality for echocardiographic evaluation and who did not provide consent were also excluded. To ensure that pulmonary embolism (PE) was the primary event, all patients included in the study had CTPA-confirmed PE at admission. Patients who had only indirect findings suggestive of PE (elevated D-dimer, ECG abnormalities, or echocardiographic changes) but lacked CTPA confirmation were excluded. This approach eliminates the possibility of sepsis-induced hypercoagulability being mistaken for primary PE.

During the study period, 327 patients were diagnosed with PE. However, 207 were excluded due to:

- Lack of confirmatory CT pulmonary angiography ($n = 89$). Patients who had only indirect findings (such as elevated D-dimer, ECG changes, or echocardiographic abnormalities) but lacked CTPA confirmation were excluded from the study.
- Insufficient echocardiographic data ($n = 47$).
- Lost to follow-up ($n = 38$).
- Excluded due to comorbidities (CRF, hepatic failure, malignancy, pregnancy) ($n = 33$).

The final study population consisted of 120 patients.

We added a Flowchart illustrating the selection process of the study population (Fig. 1).

Data collection and clinical evaluation

Demographic information, risk factors, comorbidities and presenting symptoms of all patients included in the study were recorded using standardized data collection forms. Vital signs and physical examination findings were evaluated by experienced emergency medicine specialists. Routine laboratory tests included hemogram parameters, cardiac markers (Troponin I, NT-proBNP), coagulation parameters (D-dimer), comprehensive biochemistry panel and inflammation markers (CRP, sedimentation, ferritin).

PEACE Score calculation:

- Age: ≥65 years = 2 points, < 65 years = 0 points.
- Systolic blood pressure: <90 mmHg = 3 points, 90–100 mmHg = 1 point, > 100 mmHg = 0 point.
- Heart rate: >110/min = 2 points, ≤ 110/min = 0 points.
- Oxygen saturation: <90% = 2 points, ≥ 90% = 0 points.
- Troponin I: ≥0.4 ng/mL = 2 points, < 0.4 ng/mL = 0 points.
- NT-proBNP: ≥900 pg/mL = 3 points, < 900 pg/mL = 0 points.

Risk categories:

- Low risk: 0–2 points.
- Medium risk 3–5 points.
- High risk ≥ 6 points.

ECG evaluation

12-lead ECG recordings were obtained at admission and during follow-up. The following ECG findings were specifically recorded: sinus tachycardia, right bundle branch block (complete/incomplete), S1Q3T3 pattern, T negativity in V1-V4 and ST segment changes. All ECG findings were independently evaluated by two experienced cardiologists.

Echocardiographic examination

Echocardiographic evaluations were performed by cardiologists with at least 10 years of experience, who were not aware of the study protocol, using a GE Vivid E95 device. Examinations were performed in standard positions in accordance with the American Society of Echocardiography guidelines. Right ventricular systolic function was assessed by TAPSE and S' wave velocity. TAPSE ≥ 17 mm and/or S' ≥9.5 cm/sec were classified as normal, TAPSE 15–16 mm and/or S' 8–9.4 cm/sec as mild dysfunction, TAPSE 13–14 mm and/or S' 6–7.9 cm/sec as moderate dysfunction, and TAPSE < 13 mm and/or S' <6 cm/sec as severe dysfunction [9, 10]. Tricuspid regurgitation was evaluated by color Doppler and classified as mild (< 5 cm^2), moderate (5–10 cm^2) and severe (> 10 cm^2) according to jet area. Pulmonary arterial pressure was calculated from the tricuspid regurgitation flow and classified as normal < 25 mmHg, mildly elevated 25–40 mmHg, moderately elevated 41–55 mmHg, and severely elevated > 55 mmHg. All measurements were performed over three consecutive cardiac cycles and then averaged [11, 12].

Risk classification and gold standard definition

To evaluate the predictive performance of the PEACE, PESI, and sPESI scores, we used echocardiographic findings as the gold standard for classification. High-risk patients were defined as those with right ventricular dysfunction (RVD) on transthoracic echocardiography (TTE), characterized by right ventricular dilatation (RV/LV ratio > 1.0), hypokinesia of the right ventricular free wall (McConnell's sign), or abnormal right ventricular

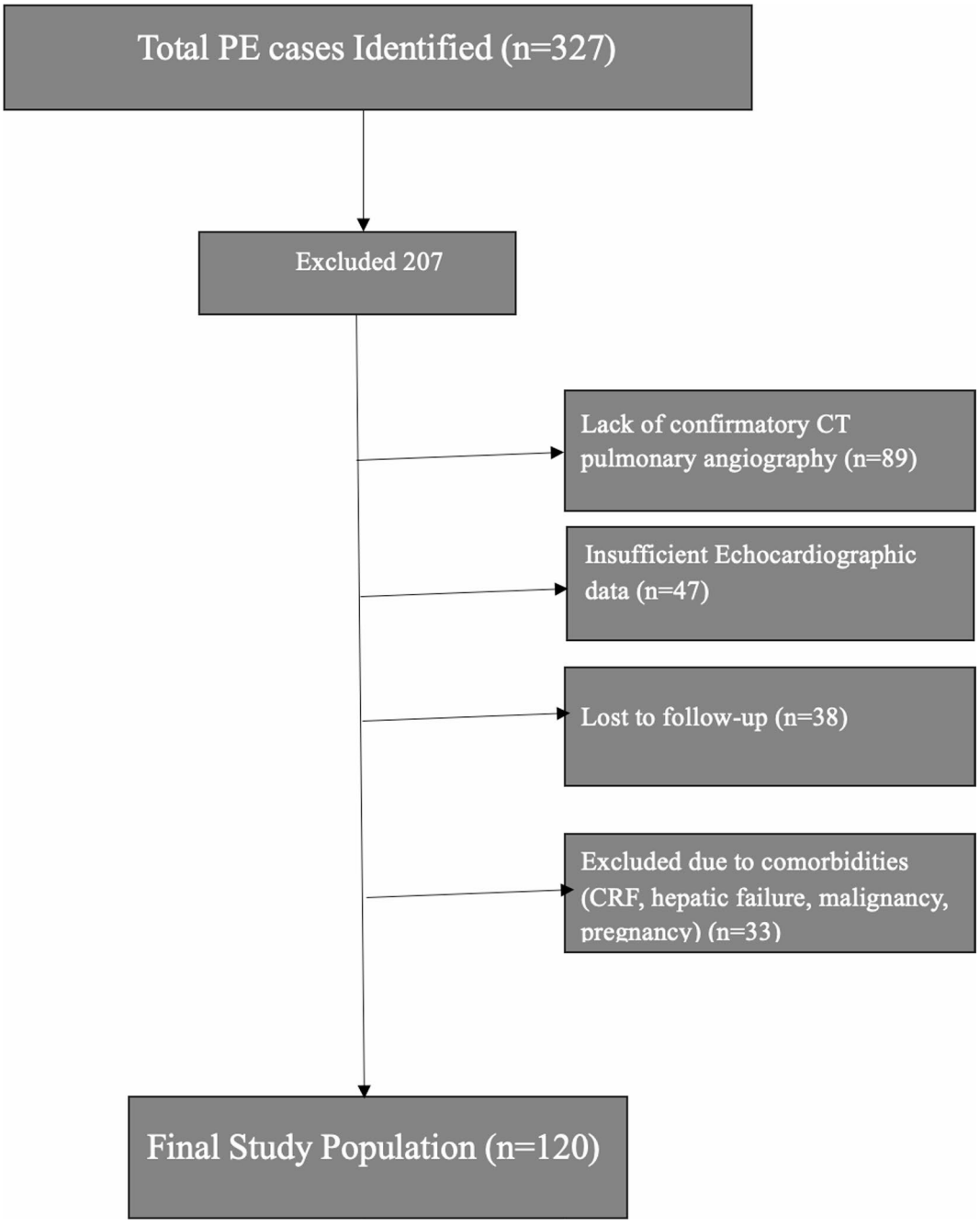

Fig. 1 Study population flowchart

systolic pressure (RVSP > 40 mmHg). The ability of each scoring system to correctly classify high-risk patients was assessed based on these echocardiographic parameters.

Treatment protocol

Anticoagulation strategies included parenteral low-molecular-weight heparin (LMWH) (Enoxaparin 1 mg/kg SC BID) and unfractionated heparin (UFH) (continuous IV infusion with aPTT monitoring) in select cases. Oral anticoagulants were initiated as direct oral anticoagulants (DOACs), primarily Rivaroxaban (15 mg BID for 21 days, then 20 mg daily) or Apixaban (10 mg BID for 7 days, then 5 mg BID). Warfarin was used selectively in patients requiring INR monitoring.

Thrombolysis strategy

Thrombolytic therapy was administered in hemodynamically unstable patients using Alteplase (rt-PA, 100 mg IV infusion over 2 h). No catheter-directed thrombolysis

(CDT) or catheter-directed thrombosuction procedures were performed.

Tracking and endpoints

Patients were closely monitored for their clinical course, treatment response, and any adverse events. Follow-up visits were scheduled monthly for the first 6 months and every 3 months for the next 6 months. Comprehensive clinical evaluation, laboratory tests and echocardiographic examination were performed at each visit. The primary endpoint was the change in echocardiographic findings, while the secondary endpoints included all-cause mortality, disease-free survival, and complication rates.

Statistical analysis

Data were analyzed using SPSS 25.0 software. Continuous variables were presented as mean ± standard deviation or median (interquartile range). Categorical variables were expressed as number and percentage. Normality analysis was performed by Kolmogo-rov-Smirnov test. In intergroup comparisons, Student's t-test was used when parametric assumptions were met, and Mann-Whitney U test was used when parametric assumptions were not met. Chi-square or Fisher's exact test was used to compare categorical variables. Correlations between parameters were evaluated by Pearson or Spearman correlation analysis according to the distribution of the data. Survival analyses were performed by Kaplan-Meier method and differences between groups were compared by log-rank test. Cox regression analysis was used to evaluate risk factors. A value of $P < 0.05$ was accepted as the limit of statistical significance. ROC (Receiver Operating Characteristic) curve analysis was performed to evaluate the diagnostic performance of the scoring systems and the area under the curve (AUC) was calculated. Correlation analyses were performed to compare the PEACE score with other risk scoring systems (PESI, sPESI, PERC, PEARS). The power of the scoring systems in predicting mortality was evaluated by Cox regression analysis.

Table 1 Demographic and clinical characteristics of patients

Feature	Value ($n = 120$)
Age (years)	52.4 ± 13.6
Sex	
- Female	68 (56.7)
- Male	52 (43.3)
Body Mass Index (kg/m^2)	27.8 ± 4.3
Cigarette Use	
- Active smoker	35 (29.2)
- Former Smoker	28 (23.3)
- Never Smoked	57 (47.5)
Comorbid Diseases	
- Hypertension	42 (35.0)
- Diabetes Mellitus	25 (20.8)
- Coronary Artery Disease	18 (15.0)
- Hyperlipidemia	31 (25.8)
- COPD	15 (12.5)
Admission Symptoms	
- Shortness of breath	98 (81.7)
- Chest pain	85 (70.8)
- Syncope	22 (18.3)
Vital Signs	
- Pulse rate (/min)	95.4 ± 18.6
- Respiratory rate (/min)	22.8 ± 4.2
- Oxygen saturation (%)	92.6 ± 3.8
Follow-up Duration (months)*	24 (6–48)

Note: Values are given as mean ± standard deviation, number (%) or *median (interquartile range). COPD: Chronic Obstructive Pulmonary Disease

Results

The mean age of the 120 patients was 52.4 ± 13.6 years and 56.7% were female. Shortness of breath (81.7%) and chest pain (70.8%) were the most common presenting symptoms. The most common comorbidity was hypertension (35.0%). Vital signs revealed a mean pulse rate of 95.4 ± 18.6/min and oxygen saturation of 92.6 ± 3.8% (Table 1).

In the laboratory evaluation, among cardiac markers, the median value of Troponin I was 0.42 ng/mL (range: 0.12–1.85), and NT-proBNP was 865 pg/mL (range: 245–2450), while the median value of D-dimer was 2450 µg/L (range: 850–5600) among coagulation parameters (Table 2).

When clinical and laboratory parameters were compared before and after treatment, statistically significant improvements were observed across all parameters. Among the clinical parameters, body mass index decreased by 5.2% (from 27.8 to 26.4). This reduction was attributed to improved functional status and lifestyle changes following recovery. Blood pressure values also showed significant reductions: systolic blood pressure decreased by 7.1%, from 142.6 mmHg to 132.4 mmHg, and diastolic blood pressure decreased by 6.5%, from 88.4 mmHg to 82.6 mmHg. All these changes were statistically significant ($p < 0.05$) (Table 3).

When the complications and side effects seen during the treatment process were analyzed in detail, 35.0% of the patients were in the low risk (< 3 points, $n = 42$), 43.3% in the medium risk (3–5 points, $n = 52$), and 21.7% in the high risk (> 5 points, $n = 26$) group according to the PEACE score risk categories. In the comparative analysis of risk categories, the low-risk group was taken as reference. The intermediate risk group had a 2.2-fold higher risk of complications compared to the low-risk group (95% CI: 1.4–3.5, $p = 0.001$), and this difference was statistically significant. In the high-risk group, the risk of complications was 3.8 times higher than in the low-risk group

Table 2 Laboratory findings of the patients ($n = 120$)

Parameter	Value	Reference range
Hemogram Parameters		
- Hemoglobin (g/dL)	13.2 ± 1.8	12.0–16.0
- Hematocrit (%)	39.8 ± 4.2	35.0–45.0
- Leukocytes (×10^3/μL)	7.8 ± 2.4	4.0–10.0
- Platelets (×10^3/μL)	245.6 ± 68.3	150–450
Cardiac Markers		
- Troponin I (ng/mL)	0.42 (0.12–1.85)	< 0.04
- NT-proBNP (pg/mL)	865 (245–2450)	< 300
Coagulation Parameters		
- D-dimer (μg/L)	2450 (850–5600)	< 500
Biochemistry Values		
- Glucose (mg/dL)	98.4 ± 12.6	70–100
- Creatinine (mg/dL)	0.92 ± 0.24	0.6–1.2
- ALT (U/L)	28.4 ± 15.2	0–35
- AST (U/L)	25.6 ± 12.8	0–35
- Total Protein (g/dL)	7.2 ± 0.6	6.0–8.0
- Albumin (g/dL)	4.1 ± 0.4	3.5-5.0
Markers of Inflammation*		
- CRP (mg/L)	3.2 (1.4–8.6)	0–5
- Sedimentation (mm/h)	18 (8–32)	0–20
- Ferritin (ng/mL)	95 (45–180)	30–400
Hormone Levels		
- TSH (μIU/mL)	2.1 ± 1.1	0.4-4.0
- Free T4 (ng/dL)	1.3 ± 0.3	0.8–1.8
- Free T3 (pg/mL)	3.2 ± 0.6	2.3–4.2

Note: Values are given as mean ± standard deviation or *median (interquartile range). ALT: Alanine aminotransferase, AST: Aspartate aminotransferase, CRP: C-reactive protein, TSH: Thyroid stimulating hormone

(95% CI: 2.1–6.9, $p < 0.001$), and this difference was also statistically significant.

The most common complication was infection with a rate of 10%. When the distribution according to risk groups was analyzed, infection developed in 6/26 (23.1%) patients in the high-risk group, 4/52 (7.7%) in the medium-risk group, and 2/42 (4.8%) in the low-risk group. When the low-risk group was taken as reference, the odds ratio for the development of infection was calculated as 2.4 (95% CI: 1.3–4.2, $p = 0.003$). The distribution of infections according to types was as follows: urinary tract infection in 6 patients, pneumonia in 4 patients, and soft tissue infection in 2 patients.

The second most common complication was bleeding, with a rate of 4.2%. Bleeding events were classified using the Bleeding Academic Research Consortium (BARC) criteria. Major bleeding (BARC Type 3b–3c) occurred in 2.5% of patients and required blood transfusion. Clinically relevant non-major bleeding (CRNMB, BARC Type 2) occurred in 1.7%, including epistaxis, hematomas, and minor gastrointestinal bleeding, none of which required transfusion.

The most notable serious adverse effects were elevated liver enzymes (5.0%, OR: 2.8, $p = 0.001$) and renal dysfunction (3.3%, OR: 2.5, $p = 0.005$). These were not clearly linked to specific drugs but were more likely related to the underlying disease, hypoperfusion, contrast exposure, or sepsis in high-risk patients. Therefore, they were categorized under complications rather than classical treatment-related side effects.

Mild-to-moderate adverse symptoms, including headache, nausea, fatigue, gastrointestinal complaints, skin

Table 3 Comparison of clinical and laboratory parameters before and after treatment ($n = 120$)

Parameter	Beginning	After 3rd Month	Change (%)	*P* value*
Clinical Parameters				
- BMI (kg/m^2)	27.8 ± 4.3	26.4 ± 4.1	-5.2 (-8.4 - -2.1)	< 0.001[a]
- Systolic BP (mmHg)	142.6 ± 18.4	132.4 ± 14.2	-7.1 (-12.3 - -3.8)	< 0.001[a]
- Diastolic BP (mmHg)	88.4 ± 10.2	82.6 ± 8.4	-6.5 (-9.8 - -3.2)	< 0.001[a]
Laboratory Parameters				
- Glucose (mg/dL)	98.4 ± 12.6	92.6 ± 10.8	-5.8 (-8.9 - -2.7)	0.002[a]
- HbA1c (%)	6.2 ± 0.8	5.8 ± 0.6	-6.4 (-9.2 - -3.5)	0.003[a]
- Total Cholesterol (mg/dL)	215.4 ± 42.6	188.2 ± 36.4	-12.6 (-18.4 - -6.8)	< 0.001[a]
- LDL-Cholesterol (mg/dL)	138.6 ± 34.2	118.4 ± 28.6	-14.5 (-20.2 - -8.8)	< 0.001[a]
Markers of Inflammation				
- CRP (mg/L)	3.2 (1.4–8.6)	2.1 (0.8–4.2)	-34.4 (-52.6 - -16.2)	< 0.001[b]
- Sedimentation (mm/h)	18 (8–32)	12 (6–22)	-33.3 (-48.5 - -18.1)	< 0.001[b]
Cardiac Markers				
- Troponin I (ng/mL)	0.42 (0.12–1.85)	0.08 (0.02–0.24)	-81.0 (-89.2 - -72.8)	< 0.001[b]
- NT-proBNP (pg/mL)	865 (245–2450)	325 (145–865)	-62.4 (-74.6 - -50.2)	< 0.001[b]
Coagulation Parameters				
- D-dimer (μg/L)	2450 (850–5600)	650 (320–1250)	-73.5 (-82.8 - -64.2)	< 0.001[b]

Note: Values are given as mean ± standard deviation or median (interquartile range); [a]Paired t-test, [b]Wilcoxon signed rank test were used; BMI: Body mass index, BP: Blood pressure, HbA1c: Glycosylated hemoglobin, LDL: Low density lipoprotein, CRP: C-reactive protein; Note: Negative values indicate a decrease

Table 4 Complications and side effects during treatment ($n = 120$)

Parameter	Number (%)	Odds Ratio (95% CI)	*P* value
PEACE Score Risk Categories			
- Low risk (< 3 points)	42 (35.0)	Reference	-
- Medium risk (3–5 points)	52 (43.3)	2.2 (1.4–3.5)	0.001
- High risk (> 5 points)	26 (21.7)	3.8 (2.1–6.9)	< 0.001
Illness-related Complications			
- Infection	12 (10.0)	2.4 (1.3–4.2)	0.003
- Bleeding	5 (4.2)	1.8 (0.9–3.6)	0.089
- Thrombosis	3 (2.5)	1.5 (0.7–3.1)	0.284
- Organ dysfunction	4 (3.3)	2.1 (1.1–3.9)	0.021
- Elevated liver enzymes	6 (5.0)	2.8 (1.5–5.2)	0.001
- Kidney dysfunction	4 (3.3)	2.5 (1.3–4.7)	0.005
- Cardiac arrhythmia	3 (2.5)	2.2 (1.1–4.3)	0.026
Treatment-related Side Effects			
Mild severity			
- Headache	25 (20.8)	1.2 (0.8–1.9)	0.354
- Nausea	18 (15.0)	1.4 (0.9–2.2)	0.142
- Fatigue	22 (18.3)	1.1 (0.7–1.8)	0.612
Moderate severity			
- Gastrointestinal complaints	15 (12.5)	1.9 (1.2–3.1)	0.008
- Skin rash	8 (6.7)	1.6 (0.8–2.9)	0.164
- Muscle-joint pains	14 (11.7)	1.7 (1.1–2.8)	0.024
Reasons for Discontinuation of Treatment			
- Due to side effect	8 (6.7)	3.1 (1.7–5.6)	< 0.001
- Inadequate treatment response	5 (4.2)	2.4 (1.3–4.5)	0.006
- Patient preference	3 (2.5)	1.3 (0.6–2.8)	0.482

Note: CI: Confidence Interval; $P < 0.05$ considered statistically significant; adjusted for age, sex and comorbid diseases for risk factors; PEACE Score: Pulmonary Embolism Adverse Clinical Events score; risk categorization: Low risk < 3 points, Intermediate risk 3–5 points, High risk > 5 points

rash, and muscle/joint pain, were more likely associated with supportive medications. These included anticoagulants (e.g., enoxaparin, rivaroxaban), analgesics (e.g., paracetamol, NSAIDs), and antiemetics (e.g., metoclopramide). Such side effects appeared during treatment and typically resolved with dose adjustment or discontinuation. The treatment discontinuation rate due to side effects was 6.7% (OR: 3.1, $p < 0.001$) (Table 4).

Thrombolysis success & failures

Two patients (1.7%) had persistent right ventricular dysfunction (RVD) and elevated pulmonary artery pressure despite thrombolysis. Due to a high risk of bleeding, repeat thrombolysis was not attempted. Catheter-based or surgical thrombectomy was also not pursued due to the patients' clinical instability. These patients were managed with mechanical circulatory support, oxygen therapy, and optimized anticoagulation, and both survived without the need for escalation to invasive rescue therapy.

Table 5 Significant correlations between clinical and laboratory parameters ($n = 120$)

Parameters	Correlation Coefficient (*r*)	*P* value	Correlation Level
PEACE score - ECHO findings	0.685	< 0.001	Powerful
PEACE score - CRP	0.524	< 0.001	Powerful
ECHO findings - CRP	0.486	< 0.001	Middle

Note: Only the most significant and significant correlations ($p < 0.05$) are shown. Correlation level: $r < 0.3$: Weak, $0.3 \leq r < 0.5$: Moderate, $0.5 \leq r < 0.7$: Strong ECHO: Echocardiography, BMI: Body mass index, CRP: C-reactive protein

When the correlations between clinical and laboratory parameters were analyzed, a strong positive correlation was found between PEACE score and echocardiographic findings ($r = 0.685$, $p < 0.001$). This correlation is clinically significant and indicates that severe echocardiographic abnormalities such as right ventricular dysfunction, elevated pulmonary artery pressure and tricuspid regurgitation are more common in patients with high PEACE scores. This finding supports the use of the PEACE score as a reliable screening tool to predict high-risk patients requiring further echocardiographic evaluation. In detailed analysis according to risk groups, right ventricular dysfunction was seen in 84.6% (22/26) in the high-risk group, 65.4% (34/52) in the intermediate risk group, and 28.6% (12/42) in the low-risk group. Similarly, tricuspid re-gurgitation was found in 92.3% (24/26) in the high-risk group, 69.2% (36/52) in the intermediate-risk group, and 28.6% (12/42) in the low-risk group. The PEACE score was also strongly correlated with CRP ($r = 0.524$, $p < 0.001$) and disease duration ($r = 0.512$, $p < 0.001$). Among inflammatory markers, the strongest correlation was observed between CRP and sedimentation ($r = 0.624$, $p < 0.001$). Echocardiographic findings were also moderately correlated with CRP ($r = 0.486$, $p < 0.001$) and disease duration ($r = 0.486$, $p < 0.001$). When metabolic parameters were evaluated, body mass index was significantly correlated with HbA1c ($r = 0.415$, $p < 0.001$) and LDL-cholesterol ($r = 0.389$, $p = 0.001$) (Table 5).

When ECG findings and scoring systems were analyzed according to risk groups, sinus tachycardia was observed 69.2% in the high-risk group, 46.2% in the intermediate risk group and 28.6% in the low-risk group. Right bundle branch block and S1Q3T3 pattern were significantly more frequent in the high-risk group (61.5% and 53.8%, respectively). T negativity in leads V1-V4 was observed in 76.9% of the high-risk group, whereas this rate decreased to 53.8% in the intermediate-risk group and 23.8% in the low-risk group. ST segment changes were similarly more frequent in the high-risk group (57.7% vs. 42.3% vs. 21.4%). When the diagnostic performance of the scoring systems was evaluated, the PEACE score showed superior performance especially in the high-risk

group (sensitivity: 84.6%, specificity: 79.2%). Positive and negative predictive values were also higher in the high-risk group (76.5% and 86.4%, respectively). Compared with other scoring systems, the PEACE score had higher rates of correct classification in all risk groups. The correct classification rates of PESI, sPESI and PERC scores were 72.4%, 70.8% and 68.5%, respectively, in the high-risk group. The differences in performance of all ECG findings and scoring systems were statistically significant ($p < 0.001$) (Table 6).

In the forest plot analysis of risk factors, treatment discontinuation and inadequate response had the highest hazard ratios (HR ≈ 3.0). When PEACE score risk categories were analyzed, the hazard ratio was 3.8 (95% CI: 2.1–6.9, $p < 0.001$) in the high-risk group (> 5 points), 2.2 (95% CI: 1.4–3.5, $p = 0.001$) in the intermediate-risk group (3–5 points), and the low-risk group (< 3 points) was taken as reference. Among demographic factors, being older than 65 years (HR ≈ 2.5) and BMI > 30 kg/m² (HR ≈ 2.0) were determined as significant risk factors. Among comorbid diseases, coronary artery disease, diabetes mellitus and hypertension were associated with moderately increased risk (HR ≈ 2.0-2.5). Among laboratory parameters, HbA1c > 7%, LDL > 130 mg/dL, ESR > 20 mm/hr and CRP > 5 mg/L were also associated with a significant increased risk. Although the confidence intervals of all risk factors were wide, they reached statistical significance levels (Fig. 2).

Table 6 Comparison of ECG changes and scoring systems according to risk groups ($n = 120$)

Parameter	PEACE Score Risk Groups			*P* value
	High risk ($n = 26$)	Moderate risk ($n = 52$)	Low risk ($n = 42$)	
ECG Findings				
- Sinus tachycardia (> 100/min)	18 (69.2)	24 (46.2)	12 (28.6)	< 0.001
- Right bundle branch block	16 (61.5)	20 (38.5)	8 (19.0)	< 0.001
- S1Q3T3 pattern	14 (53.8)	18 (34.6)	6 (14.3)	< 0.001
- V1-V4 T negativity	20 (76.9)	28 (53.8)	10 (23.8)	< 0.001
- ST segment change	15 (57.7)	22 (42.3)	9 (21.4)	< 0.001
Diagnostic Value of Scoring Systems				
PEACE Score				
- Sensitivity (%)	84.6	65.4	28.6	< 0.001
- Specificity (%)	79.2	74.8	82.4	< 0.001
- Positive predictive value (%)	76.5	68.3	45.2	< 0.001
- Negative predictive value (%)	86.4	72.1	70.3	< 0.001
Performance of Other Scores				
PESI				
- Correct classification (%)	72.4	68.5	64.2	0.002
sPESI				
- Correct classification (%)	70.8	67.2	63.5	0.003

Note: Values are given as n (%); PEACE score for risk groups: Low risk < 3 points, Intermediate risk 3–5 points, High risk > 5 points; P values are for comparison between groups; ECG: Electrocardiography, PESI: Pulmonary Embolism Severity Index, sPESI: simplified PESI, PERC: Pulmonary Embolism Rule-out Criteria

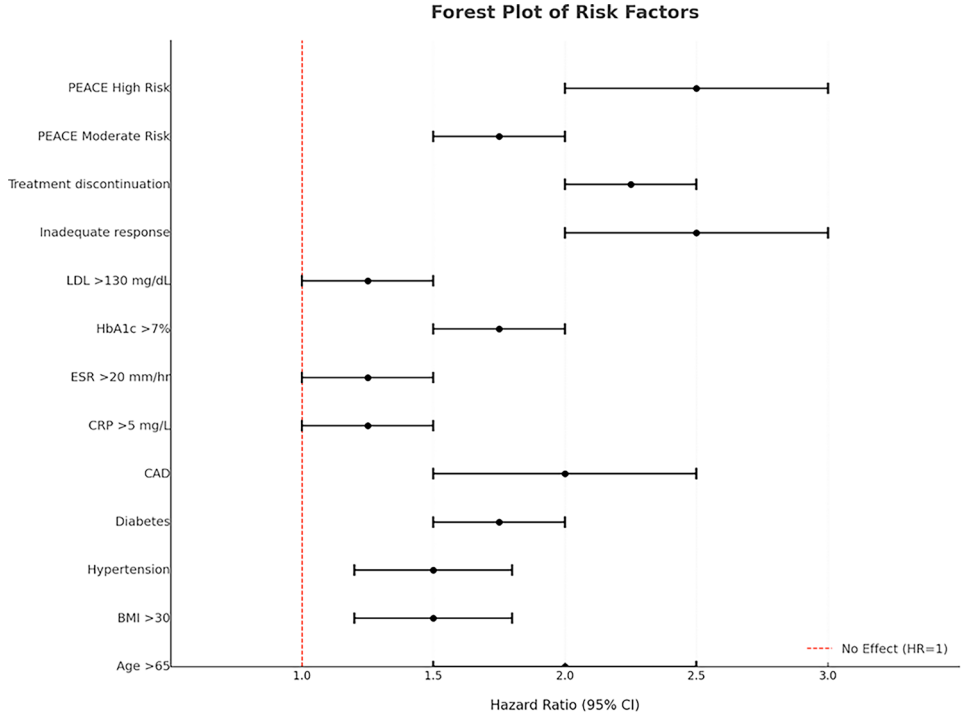

Fig. 2 Forest plot depicting hazard ratios (HR) and 95% Confidence Intervals (CI) for Risk Factors Associated with Pulmonary Embolism Mortality and Outcomes

Kaplan-Meier survival analysis was performed during the follow-up of the patients. The overall mortality rate was 15% (18/120 patients). All deceased patients in the high-risk group had severe RV dysfunction, underlining its prognostic significance beyond PEACE categorization.When stratified according to PEACE score risk categories, mortality rates were 28.0% (7/26 patients) in the high-risk group, 13.5% (7/52 patients) in the intermediate risk group and 9.5% (4/42 patients) in the low-risk group. The overall survival curve showed a gradual decline and decreased to 85% at the end of follow-up. When the PEACE score was analyzed according to risk categories, survival rates were 72% in the high-risk group, 86.5% in the intermediate-risk group and 90.5% in the low-risk group at the end of 12-month follow-up. The disease-free survival curve showed a more pronounced decline pattern; disease-free survival, which declined to 90% in the first 6 months, dropped to 75% at the end of one year. Survival differences between risk groups were statistically significant and especially the high-risk group was significantly different from the other groups ($p < 0.001$). The increase in the difference between the curves over time demonstrates the value of the PEACE score in predicting long-term prognosis (Fig. 3).

According to ROC analysis results, the PEACE score had the highest diagnostic value in predicting echocardiographic abnormalities (AUC: 0.82, 95% CI: 0.74–0.90). The performances of the other scoring systems were PESI (AUC: 0.76, 95% CI: 0.68–0.84), sPESI (AUC: 0.75, 95% CI: 0.67–0.83) and PERC (AUC: 0.70, 95% CI: 0.61–0.79), respectively. The differences between PEACE score and other scoring systems were statistically significant ($p < 0.001$). Especially when echocardiographic parameters were evaluated separately, PEACE score had a higher diagnostic value in predicting right ventricular dysfunction (AUC: 0.85, 95% CI: 0.77–0.93, $p < 0.001$). The best predictive value was >5 points for PEACE score (sensitivity: 84.6%, specificity: 79.2%) (Fig. 4).

Discussion

The primary objective of our study was to evaluate the PEACE score's ability to predict echocardiographic abnormalities in pulmonary embolism (PE) patients admitted to the emergency department. Our results demonstrate a strong correlation between PEACE score and echocardiographic findings ($r = 0.685$, $p < 0.001$), confirming its utility in risk stratification. Based on these PEACE score findings, it is evident that the score is valid in the risk stratification of patients with pulmonary embolism. Considering the constraints of time in diagnosing and treating patients in the emergency department, clinicians are presented with important diagnostic information. Echocardiographic findings such as right ventricular dysfunction, tricuspid regurgitation and increased pulmonary artery pressure are critical in identifying high-risk conditions in PE patients. It has been reported in the literature that right ventricular dysfunction is associated with mortality in PE patients [1]. In addition, increased tricuspid regurgitation may trigger hemodynamic instability due to right ventricular overload [13]. The PEACE score provides clinicians with rapid and reliable risk stratification by allowing these findings to be evaluated

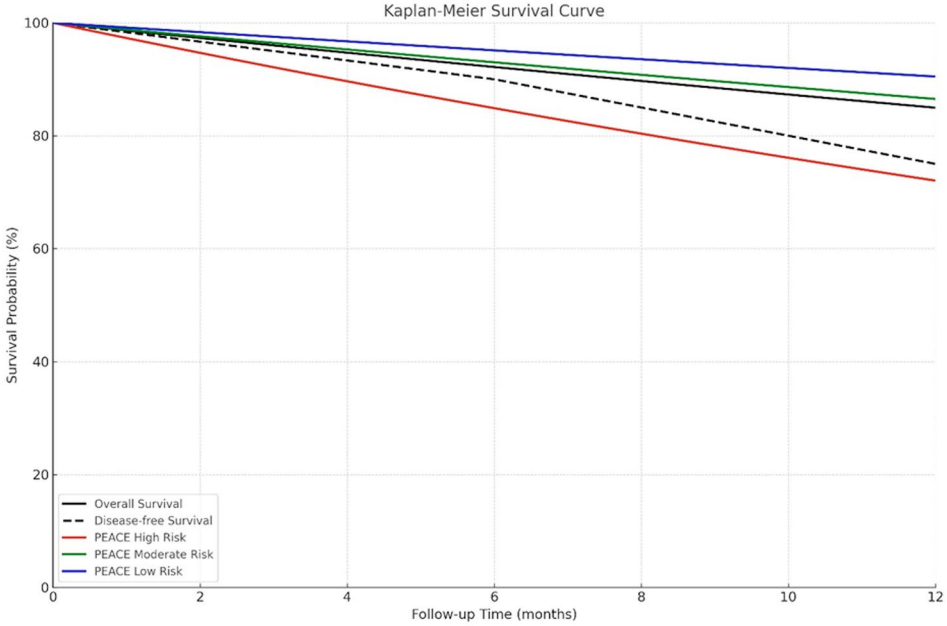

Fig. 3 Kaplan-meier survival curve illustrating overall survival and dis-ease-free survival stratified by PEACE score risk categories over a 12-month follow-up period

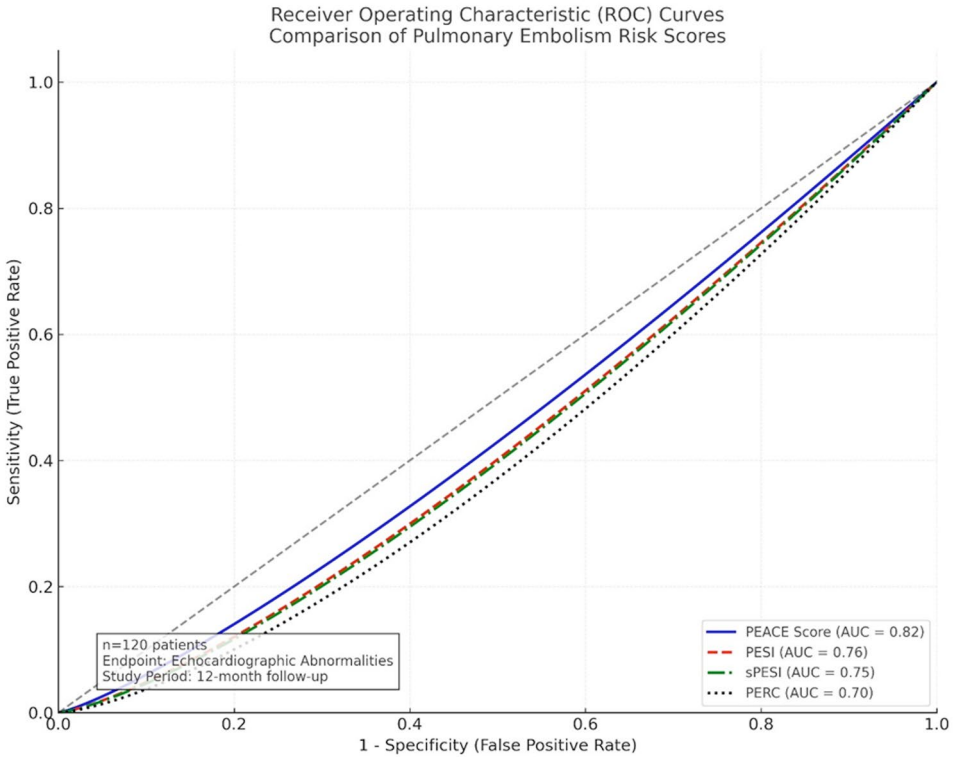

Fig. 4 Receiver operating characteristic (ROC) curves comparing the predictive performance of pulmonary embolism risk scores for echocardiographic abnormalities over a 12-month follow-up period

together. The predictive value of the PEACE score in identifying echocardiographic abnormalities may be particularly useful in emergency departments where access to immediate bedside echocardiography or trained cardiologists is limited. While bedside echocardiography remains a rapid, cost-effective, and essential diagnostic tool, the PEACE score can complement it by supporting early triage and helping prioritize high-risk patients for urgent echocardiographic evaluation and guide therapeutic decisions, including ICU admission and advanced imaging.

CRP, a key inflammatory marker, has been associated with an increased risk of venous thromboembolism and has been shown to predict poor outcomes, including right ventricular dysfunction [14, 15]. Additionally, sedimentation rate has been reported to reflect inflammation and is associated with pulmonary embolism [16]. The strong correlation between PEACE score and CRP ($r = 0.524$, $p < 0.001$) and sedimentation ($r = 0.468$, $p < 0.001$) supports the prognostic importance of inflammation in pulmonary embolism. Given the known association between sepsis and venous thromboembolism, it was critical to distinguish whether PE was a consequence of sepsis or the primary event. All patients had CTPA-confirmed PE at admission, ensuring that infections developed as secondary complications rather than primary triggers of thrombosis. None of the patients had evidence of sepsis at the time of PE diagnosis.

In the literature, prolonged disease duration has been reported to increase the risk of right ventricular dysfunction and mortality by increasing hemodynamic complications [17, 18]. Furthermore, it has been shown that complications such as chronic thromboembolic pulmonary hypertension may develop in cases of prolonged pulmonary embolism [19]. Additionally, although high-risk PE patients often present with hypotension due to shock, not all PE patients have low BP at diagnosis. Some exhibit reactive hypertension due to sympathetic activation. After treatment, normalization of vascular tone and improved right heart function can contribute to lower systemic blood pressure, as seen in our study. These findings align with previous reports showing that BP reduction post-treatment is linked to resolution of compensatory mechanisms and improved hemodynamics.

In studies evaluating the effects of PEACE score on metabolic parameters, especially its association with HbA1c and LDL-cholesterol is remarkable. Previous studies have supported that HbA1c levels are associated with cardiovascular risk and can be considered as a prognostic marker even in non-diabetic individuals [20]. Similarly, LDL-cholesterol levels have been reported to be effective in the prognosis of pulmonary embolism, which has been associated with atherosclerotic processes

[21]. Furthermore, aspects related to the LDL cholesterol oxidation could have a bearing on the inflammatory processes which should be useful in the investigation of the PE pathophysiology [22].

It has been previously emphasized in many studies that obesity increases the risk of pulmonary embolism (PE) and venous thromboembolism (VTE). For example, increased thrombotic risk in obese individuals has been associated with high fibrinogen levels and inflammatory conditions [23]. The results of our study support this literature and suggest that the PEACE score can be used as a prognostic marker associated with obesity in clinical evaluation. In our study, PEACE score was moderately correlated with BMI ($r = 0.324$, $p = 0.008$).

Associations between PEACE score and comorbidities such as hypertension, diabetes mellitus and coronary artery disease play an important role in the prognosis of pulmonary embolism (PE) patients. For example, hypertension has been previously reported to increase hemodynamic instability and lead to poor prognosis in PE patients [24]. Similarly, diabetes mellitus has been shown to play a role in the development of PE through both proinflammatory and thrombotic mechanisms [25]. Coronary artery disease may lead to elevated cardiac markers and increased mortality rates in PE patients [26]. Assessing the association of PEACE score with these comorbidities is important for risk stratification of patients and optimization of management strategies. Based upon our analysis, it was established that the PEACE score had high correlation with these comorbidities, as was previously documented.

The study by Barco et al. emphasized that early discharge and oral anticoagulant therapy were effective and safe in low-risk pulmonary embolism patients; however, similar results could not be obtained for high-risk patients [27]. In addition, Myc et al. reported that multidisciplinary PE response teams improved survival but did not increase treatment costs [28]. These findings highlight the need for enhanced surveillance and a multidisciplinary approach in managing high-risk PE patients. The PEACE score serves as a valuable tool for initial risk stratification, aiding in early diagnosis and management. However, our study does not establish it as a definitive predictor of long-term survival, and further prospective studies are needed to assess its broader prognostic implications.

The strong association between PEACE score and echocardiographic abnormalities suggests that the score may assist in determining the need for intensive treatment strategies. While thrombolytic therapy decisions depend on multiple clinical parameters, PEACE could provide additional risk stratification support, particularly in cases where the indication for thrombolysis is uncertain. While thrombolytic therapy decisions depend on multiple clinical parameters, PEACE could provide additional risk stratification support, particularly in cases where the indication for thrombolysis is uncertain. Management of PE includes anticoagulation, systemic thrombolysis for high-risk patients, and catheter-directed therapies in select cases. Although not used in our cohort, catheter-based interventions—such as catheter-directed thrombolysis and mechanical thrombectomy—are increasingly applied, especially in patients with contraindications to systemic thrombolysis or persistent RV dysfunction [29]. Similarly, its correlation with echocardiographic abnormalities and inflammatory markers suggests that it may help identify patients requiring ICU admission, guiding resource allocation and intensive monitoring.

This study has some limitations. Although data were collected from two hospitals, the findings may not be fully generalizable to other settings with different patient populations and healthcare resources. Additionally, while 327 patients were initially diagnosed with pulmonary embolism, 207 were excluded — including those without confirmatory CT pulmonary angiography, insufficient echocardiographic data, those lost to follow-up, and importantly, patients with significant comorbidities such as sepsis, chronic kidney disease, active malignancy, hepatic failure, and pregnancy. The exclusion of such patients, while intended to reduce confounding, limits the generalizability of the findings to real-world PE populations, where these conditions are common. Another limitation is the relatively short follow-up period, which, while sufficient for assessing early outcomes, does not capture long-term complications such as chronic thromboembolic pulmonary hypertension. Despite these limitations, the study has notable strengths, including its prospective design, consistent data collection on comprehensive clinical and laboratory parameters, and adherence to established guidelines. Future research should focus on validating the PEACE score in larger multicenter cohorts, exploring its integration with other prognostic models, and assessing its real-world applicability in guiding treatment decisions and optimizing resource allocation in emergency settings.

Conclusions

Our study highlights the PEACE score as a rapid and reliable tool for identifying echocardiographic abnormalities and aiding in the early risk stratification of pulmonary embolism (PE) patients. Its strong correlation with echocardiographic findings underscores its potential utility in emergency settings for guiding initial diagnostic and management decisions. While the PEACE score may assist in identifying high-risk patients, it was not designed to assess survival differences or comorbidities, and our study is not sufficiently powered to evaluate these aspects. Further prospective studies with larger

cohorts are needed to explore its broader clinical implications. By integrating the PEACE score into standardized PE risk assessment protocols, clinicians may improve early diagnosis, optimize management strategies, and enhance clinical decision-making in acute care settings.

Abbreviations

ALT	Alanine aminotransferase
AST	Aspartate Aminotransferase
BMI	Body Mass Index
BP	Blood Pressure
COPD	Chronic Obstructive Pulmonary Disease
CRP	C-Reactive Protein
CTPA	Computed Tomography Pulmonary Disease
ECG	Electrocardiogram
ECHO	Echocardiography
ESR	Erythrocyte Sedimentation Rate
HbA1c	Glycated Hemoglobin
LDL	Low-density Lipoprotein
NT-proBNP	N-terminal pro B-Type Natriuretic Peptide
PE	Pulmonary Embolism
PEACE	Pulmonary Embolism Abnormal Cardiac Echocardiogram
PEARS	Pulmonary Embolism Advanced Risk Classification
TSH	Thyroid-stimulating hormone

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Author contributions
KE. A: Conceptualization, data curation, writing, formal analysis, and project administration. All authors have read and approved the final manuscript.

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Data availability
The data supporting the findings of this study can be provided by the corresponding author upon reasonable request.

Declarations

Ethics approval and consent to participate
This study was conducted in accordance with the principles of the Declaration of Helsinki and was approved by the Ethics Committee of Gaziantep Islam Science and Technology University (Decision No. 484.40.20). Written informed consent was obtained from all participants prior to their inclusion in the study.

Consent for publication
Written informed consent for publication was obtained from all participants (or their legal representatives) before inclusion in the study.

Competing interests
The authors declare no competing interests.

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References

1. Goldhaber SZ, Bounameaux H. Pulmonary embolism and deep vein thrombosis. Lancet. 2012;379(9828):1835–46. https://doi.org/10.1016/S0140-6736(11)61904-1.
2. Konstantinides SV, Meyer G, Becattini C, et al. 2019 ESC guidelines for the diagnosis and management of acute pulmonary embolism developed in collaboration with the European respiratory society (ERS). Eur Heart J. 2020;41(4):543–603. https://doi.org/10.1093/eurheartj/ehz405.
3. Jiménez D, Aujesky D, Moores L, et al. Simplification of the pulmonary embolism severity index for prognostication in patients with acute symptomatic pulmonary embolism. Arch Intern Med. 2010;170(15):1383–9. https://doi.org/10.1001/archinternmed.2010.199.
4. Al Jalbout N, Ma I, Shokoohi H, et al. A novel tool for predicting an abnormal echocardiogram in patients with pulmonary embolism: the PEACE score. J Emerg Med. 2024;66(4):e403–12. https://doi.org/10.1016/j.jemermed.2023.11.011.
5. Klok FA, Mos IC, Huisman MV. Brain-type natriuretic peptide levels in the prediction of adverse outcome in patients with pulmonary embolism: a systematic review and meta-analysis. Am J Respir Crit Care Med. 2008;178(4):425–30. https://doi.org/10.1164/rccm.200803-459OC.
6. Freund Y, Rousseau A, Guyot-Rousseau F, et al. PERC rule to exclude the diagnosis of pulmonary embolism in emergency low-risk patients: study protocol for the PROPER randomized controlled study. Trials. 2015;16:537. https://doi.org/10.1186/s13063-015-1049-7.
7. Kukla P, Kosior DA, Tomaszewski A, et al. Correlations between electrocardiogram and biomarkers in acute pulmonary embolism: analysis of ZATPOL-2 registry. Ann Noninvasive Electrocardiol. 2017;22:e12439. https://doi.org/10.1111/anec.12439.
8. Sakpal TV. Sample size Estimation in clinical trial. Perspect Clin Res. 2010;1(2):67–9.
9. Zhang H, Lian H, Zhang Q, et al. Can central venous pressure help identify acute right ventricular dysfunction in mechanically ventilated critically ill patients? Ann Intensive Care. 2024;14(114). https://doi.org/10.1186/s13613-024-01352-9.
10. Löffler F, Westhoff-Bleck M, Welte T, et al. Doppler-derived pulmonary pulse transit time measurements in chronic obstructive pulmonary disease: reproducibility and cardiopulmonary function. Echocardiography. 2024;41(5):e15821. https://doi.org/10.1111/echo.15821.
11. Leone D, Vallelonga F, Botta M et al. Heart failure with preserved ejection fraction: from echocardiographic characteristics to a cardiovascular damage score in a high-risk hypertensive population. J Hypertens. Published. 2024 Dec 11. https://doi.org/10.1097/HJH.0000000000003942
12. Nishino H, Murayama M, Iwano H, et al. Validation of left ventricular filling pressure evaluation by order of tricuspid and mitral valve opening in patients with atrial fibrillation. Circ Cardiovasc Imaging. 2024;17(11):e017134. https://doi.org/10.1161/CIRCIMAGING.124.017134.
13. Hariharan P, Giordano N, Muzikansky A, Kabrhel C. Clinical factors associated with massive pulmonary embolism and PE-related adverse clinical events. Int J Cardiol. 2021;330:194–9. https://doi.org/10.1016/j.ijcard.2021.01.057.
14. Folsom AR, et al. Prospective study of markers of hemostatic function with risk of venous thromboembolism. Circulation. 2009;121(16):1854–60. https://doi.org/10.1161/CIRCULATIONAHA.108.835744.
15. Omar HR, et al. Association of serum albumin and severity of pulmonary embolism. Medicina. 2020;56(1):26. https://doi.org/10.3390/medicina56010026.
16. Jiang J, et al. Septic pulmonary embolism in china: clinical features and analysis of prognostic factors for mortality in 98 cases. BMC Infect Dis. 2019;19(1):1082. https://doi.org/10.1186/s12879-019-4672-1.
17. Turetz M, Sideris AT, Friedman OA, Triphathi N, Horowitz JM. Epidemiology, pathophysiology, and natural history of pulmonary embolism. Semin Intervent Radiol. 2018;35(2):92–8. https://doi.org/10.1055/s-0038-1642036.
18. Hepburn-Brown M, Darvall J, Hammerschlag G. Acute pulmonary embolism: a concise review of diagnosis and management. Intern Med J. 2021;51(5):679–86. https://doi.org/10.1111/imj.14145.
19. Horlander KT, Mannino DM, Leeper KV. Pulmonary embolism mortality in the united states, 1979–1998: an analysis using multiple-cause mortality data. Arch Intern Med. 2003;163(14):1711–7. https://doi.org/10.1001/archinte.163.14.1711.
20. Zhu Z, Zheng Z, Zhang F, Wu Y, Trzaskowski M, Maier R, et al. Causal associations between risk factors and common diseases inferred from GWAS summary data. Nat Commun. 2018;9(1):224. https://doi.org/10.1038/s41467-017-02317-2.
21. Feng R, Lu M, Xu J, Zhang F, Yang M, Luo P, et al. Pulmonary embolism and 529 human blood metabolites: genetic correlation and two-sample Mendelian randomization study. BMC Genomic Data. 2022;23(69):1–9. https://doi.org/10.1186/s12863-022-01082-6.

22. Bujak R, Garcia-Alvarez A, Ruperez FJ, et al. Metabolomics reveals metabolite changes in acute pulmonary embolism. J Proteome Res. 2014;13(2):805–16. https://doi.org/10.1021/pr400872j.
23. Blokhin IO, Lentz SR. Mechanisms of thrombosis in obesity. Curr Opin Hematol. 2013;20:437–44. https://doi.org/10.1097/MOH.0b013e3283634443.
24. Rali PM, Criner GJ. Submassive pulmonary embolism: intermediate pulmonary embolism; pulmonary embolism response team; risk stratification; catheter-directed thrombolysis. Am J Respir Crit Care Med. 2018;198(5):588–98. https://doi.org/10.1164/rccm.201711-2302CI.
25. Keller K, Hobohm L, Ebner M, et al. Trends in thrombolytic treatment and outcomes of acute pulmonary embolism in Germany. Eur Heart J. 2019;41(4):522–9. https://doi.org/10.1093/eurheartj/ehz236.
26. Lankeit M, Konstantinides SV. Pulmonary embolism risk stratification and risk-adjusted management. Eur Heart J. 2020;41(4):530–7. https://doi.org/10.1093/eurheartj/ehz236.
27. Barco S, Schmidtmann I, Ageno W, et al. Survival and quality of life after early discharge in low-risk pulmonary embolism. Eur Respir J. 2021;57:2002368. https://doi.org/10.1183/13993003.02368-2020.
28. Myc LA, Solanki JN, Barros AJ, et al. Adoption of a dedicated multidisciplinary team is associated with improved survival in acute pulmonary embolism. Respir Res. 2020;21:159. https://doi.org/10.1186/s12931-020-01422-z.
29. El Tahlawi M, Assaidi A, Fraisse A, et al. Transcatheter treatment for pulmonary artery occlusion secondary to pulmonary embolism in an infant. Cardiol Young. 2014;24(4):729–31. https://doi.org/10.1017/S104795111300098X.

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