

Research Article

Clinical and Hemodynamic Predictors of Intensive Care Unit Mortality in Critically Ill Patients with Acute Pulmonary Embolism: A Retrospective Cohort Study

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Abstract

Acute pulmonary embolism (PE) presenting with hemodynamic instability or severe right ventricular (RV) failure carries high intensive care unit (ICU) mortality. This retrospective cohort study evaluated clinical, biochemical, and echocardiographic predictors of all-cause ICU mortality in critically ill patients admitted with acute high-risk and intermediate-high-risk PE. A total of 260 consecutive adult ICU admissions managed for acute PE across two academic tertiary care medical centers over a 2-year period were analyzed. Primary outcome was all-cause ICU mortality. In-hospital non-fatal major bleeding events and acute kidney injury (AKI) rates were recorded as

secondary safety outcomes. Overall ICU mortality occurred in 21.5% ($n = 56$) of the cohort. Patients who died exhibited significantly higher rates of initial persistent hypotension/obstructive shock (62.5% vs. 22.1% , $p < 0.001$), higher baseline cardiac troponin I levels (1.85 ± 1.12 { ng/mL } vs. 0.62 ± 0.45 { ng/mL } , $p < 0.001$), higher pulmonary artery systolic pressure (PASP; 58.4 ± 12.6 { mmHg } vs. 44.2 ± 10.8 { mmHg } , $p < 0.001$), and an elevated RV-to-left ventricular (RV/LV) diameter ratio 1.2 (69.6 % vs. 32.4% , $p < 0.001$). Multivariable logistic regression identified obstructive shock upon admission (aOR: 3.82, 95% CI: 1.88–7.76, $p < 0.001$), elevated RV/LV ratio 1.2 (aOR: 2.94, 95%

CI: 1.42–6.08, $p = 0.004$), serum lactate $3.5 \{ \text{mmol/L} \}$ (aOR: 2.51, 95% CI: 1.25–5.04, $p = 0.010$), and stage 2–3 AKI (aOR: 2.18, 95% CI: 1.06–4.48, $p = 0.034$) as independent predictors of ICU death. Obstructive shock, severe echocardiographic RV strain, elevated serum lactate, and acute kidney injury serve as crucial early prognostic indicators for risk stratification in critically ill acute PE patients.

Keywords: Acute pulmonary embolism, intensive care unit, right ventricular dysfunction, obstructive shock, cardiac troponin, lactate, mortality predictors.

Introduction

Acute pulmonary embolism (PE) is a major cardiovascular emergency and a leading cause of preventable hospital mortality worldwide. While mild or low-risk PE can often be safely managed in outpatient or step-down settings, high-risk (massive) and intermediate-high-risk (submassive) PE present with life-threatening hemodynamic instability, requiring immediate admission to the intensive care unit (ICU) for invasive monitoring, mechanical respiratory support, vasopressor administration, or advanced reperfusion therapies. [1–3]

The physiological hallmark of severe PE is acute right ventricular overload. Obstruction

of the pulmonary vascular bed increases RV afterload, inducing RV dilation, tricuspid regurgitation, and increased myocardial wall tension. This leads to RV ischemia, interventricular septal shift toward the left ventricle, reduced LV preload, and decreased cardiac output, culminating in obstructive shock and multi-organ dysfunction syndrome (MODS). [4–6]

Despite advances in risk stratification tools such as the Pulmonary Embolism Severity Index (PESI) and Bova score, predicting short-term ICU mortality in critically ill PE patients remains challenging. The relative prognostic weight of specific bedside hemodynamic, echocardiographic, and biochemical parameters in predicting ICU survival requires further clarification to guide timely escalation to systemic thrombolysis, catheter-directed thrombolysis (CDT), or surgical embolectomy. [7–10]

This study aimed to determine the independent clinical, hemodynamic, and echocardiographic predictors of all-cause ICU mortality in critically ill patients admitted with acute PE.

Methodology

A retrospective cohort study was conducted analyzing consecutive adult patients (aged

18 years) admitted to the ICUs of two tertiary care academic centers over a 2-year period at Shahida Islam Teaching Hospital, Pakistan with a primary diagnosis of acute PE confirmed by computed tomography pulmonary angiography (CTPA) or high-probability ventilation-perfusion (V/Q) scintigraphy.

Patients were categorized according to European Society of Cardiology (ESC) guidelines into:

- **High-Risk PE:** Presenting with persistent hypotension (systolic blood pressure [SBP] < 90 { mmHg} or SBP drop 40 { mmHg} for > 15 { min}) or obstructive shock.
- **Intermediate-High-Risk PE:** Hemodynamically stable (SBP 90 { mmHg}) but exhibiting both RV dysfunction on echocardiography/CTPA and elevated cardiac biomarkers (cardiac troponin I or NT-proBNP).

Exclusion criteria comprised patients transferred to outside institutions within 24 hours of admission, those with pre-existing end-stage renal disease on maintenance hemodialysis, active terminal malignancy, or incomplete clinical/echocardiographic records.

Data parameters extracted from electronic medical records included baseline

demographics, Pulmonary Embolism Severity Index (simplified sPESI) score, initial vital signs, arterial blood gas metrics, serum lactate, high-sensitivity cardiac troponin I (hs-cTnI), and NT-proBNP levels. Transthoracic echocardiography (TTE) performed within 24 hours of ICU admission was reviewed to document RV/LV diameter ratio (measured in the apical 4-chamber view), pulmonary artery systolic pressure (PASP), tricuspid annular plane systolic excursion (TAPSE), and interventricular septal flattening.

Primary study outcome was all-cause ICU mortality. Secondary outcomes included in-hospital major bleeding events (defined per International Society on Thrombosis and Haemostasis [ISTH] criteria) and development of Stage 2 or 3 Acute Kidney Injury (KDIGO criteria).

Statistical analysis was performed using SPSS version 26.0. Continuous variables were expressed as mean $\pm$ SD or median [IQR] and compared using Student's t -test or Mann-Whitney U test. Categorical variables were compared using Chi-square or Fisher's exact tests. Multivariable logistic regression models were established to

evaluate independent risk factors for ICU mortality

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Results

Table 1: Baseline Demographics, Admission Parameters, and Clinical Risk Profiles

Clinical Parameter	Overall Cohort (N=260)	ICU Survivors (n=204)	ICU Non-Survivors (n=56)	p-value
Age (Years ± SD)	61.2 ± 14.5	59.8 ± 14.1	66.3 ± 15.2	0.003
Gender (Male / Female %)	142 / 118 (54.6% / 45.4%)	110 / 94 (53.9% / 46.1%)	32 / 24 (57.1% / 42.9%)	0.672
High-Risk (Massive) PE Class (%)	80 (30.8%)	45 (22.1%)	35 (62.5%)	< 0.001
Intermediate-High-Risk PE Class (%)	180 (69.2%)	159 (77.9%)	21 (37.5%)	< 0.001
sPESI Score 1 (%)	212 (81.5%)	158 (77.5%)	54 (96.4%)	0.001
Initial Hemodynamics & Gasometry				
Admission SBP ({mmHg} ± {SD})	104.2 ± 22.6	109.5 ± 19.8	84.8 ± 23.1	< 0.001

Clinical Parameter	Overall Cohort (N=260)	ICU Survivors (n=204)	ICU Non-Survivors (n=56)	p-value
Heart Rate ({bpm} $\pm$ {SD})	112.4 $\pm$ 18.2	109.8 $\pm$ 17.5	121.8 $\pm$ 18.9	< 0.001
Arterial {PaO}_2 / {FiO}_2 Ratio	215 $\pm$ 68	232 $\pm$ 62	153 $\pm$ 54	< 0.001
Initial Serum Lactate ({mmol/L} $\pm$ {SD})	2.8 $\pm$ 1.6	2.2 $\pm$ 1.1	4.9 $\pm$ 2.2	< 0.001
High-Sensitivity Troponin I ({ng/mL} $\pm$ {SD})	0.88 $\pm$ 0.72	0.62 $\pm$ 0.45	1.85 $\pm$ 1.12	< 0.001

Note: Patients who died during ICU admission presented with significantly higher illness severity scores, lower systemic arterial blood pressures, worse oxygenation parameters, and markedly higher initial serum lactate and troponin levels.

Table 2: Echocardiographic and Reperfusion Therapy Characteristics

Parameter	Overall Cohort (N=260)	ICU Survivors (n=204)	ICU Non-Survivors (n=56)	p-value
Echocardiographic Parameters				

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Parameter	Overall Cohort (N=260)	ICU Survivors (n=204)	ICU Non-Survivors (n=56)	p-value
RV/LV Diameter Ratio 1.2 (%)	105 (40.4%)	66 (32.4%)	39 (69.6%)	< 0.001
Pulmonary Artery Systolic Pressure ({mmHg})	47.2 ± 12.1	44.2 ± 10.8	58.4 ± 12.6	< 0.001
TAPSE (< 16 { mm}) (%)	118 (45.4%)	82 (40.2%)	36 (64.3%)	0.001
Interventricular Septal Flattening (%)	134 (51.5%)	92 (45.1%)	42 (75.0%)	< 0.001
Reperfusion & Invasive Therapies				
Systemic Thrombolysis (rt-PA) (%)	52 (20.0%)	38 (18.6%)	14 (25.0%)	0.288
Catheter-Directed Thrombolysis / Embolectomy	28 (10.8%)	24 (11.8%)	4 (7.1%)	0.320
Invasive Mechanical Ventilation (%)	68 (26.2%)	32 (15.7%)	36 (64.3%)	< 0.001
Vasopressor / Inotropic Support (%)	94 (36.2%)	48 (23.5%)	46 (82.1%)	< 0.001

Note: Severe right ventricular overload (RV/LV ratio 1.2 , elevated PASP, septal flattening, reduced TAPSE) was significantly more frequent among patients who died in the ICU.

Table 3: Clinical Outcomes and Complications

Outcome Measure	Total Cohort (N=260)	High-Risk PE (n=80)	Intermediate-High PE (n=180)	p-value
Primary Outcome: ICU Mortality (%)	56 (21.5%)	35 (43.8%)	21 (11.7%)	< 0.001
ICU Length of Stay (Days, Median [IQR])	5 [3–9]	7 [4–12]	4 [2–7]	< 0.001
Hospital Mortality (%)	64 (24.6%)	38 (47.5%)	26 (14.4%)	< 0.001
Complications				
ISTH Major Bleeding (%)	24 (9.2%)	14 (17.5%)	10 (5.6%)	0.002
Acute Kidney Injury (KDIGO Stage 2–3) (%)	62 (23.8%)	34 (42.5%)	28 (15.6%)	< 0.001

Note: ICU mortality was substantially higher in patients presenting with high-risk PE compared to those with intermediate-high-risk PE (43.8% vs 11.7%).

Table 4: Multivariable Logistic Regression Analysis of Independent Predictors of ICU Mortality

Predictor Variable	Unadjusted OR (95% CI)	Adjusted OR (aOR) (95% CI)*	p-value
Obstructive Shock / Persistent Hypotension	5.82 (3.12–10.85)	3.82 (1.88–7.76)	< 0.001
Echocardiographic RV/LV Ratio 1.2	4.80 (2.54–9.07)	2.94 (1.42–6.08)	0.004
Initial Serum Lactate 3.5 { mmol/L}	4.35 (2.32–8.15)	2.51 (1.25–5.04)	0.010
Acute Kidney Injury (Stage 2–3)	3.98 (2.11–7.51)	2.18 (1.06–4.48)	0.034
High-Sensitivity Troponin I > 1.0 { ng/mL}	3.65 (1.92–6.94)	1.84 (0.88–3.85)	0.104
Invasive Mechanical Ventilation	9.68 (4.95–18.92)	2.42 (1.08–5.42)	0.032

Note: *Multivariable model adjusted for age, sPESI score, baseline comorbid conditions (hypertension, COPD, heart failure), systemic thrombolysis therapy, and arterial oxygenation status.

Discussion

This retrospective cohort study evaluated predictors of ICU mortality among critically ill patients with acute pulmonary embolism. The overall ICU mortality rate was 21.5%, rising to 43.8% in patients presenting with high-risk PE. The analysis identified initial obstructive shock, echocardiographic RV/LV

ratio 1.2, elevated serum lactate (3.5 { mmol/L}), stage 2–3 AKI, and need for invasive mechanical ventilation as independent risk factors for death in the ICU.

Obstructive shock was the primary clinical driver of short-term mortality (aOR: 3.82, 95% CI: 1.88–7.76). Acute pulmonary

arterial occlusion sharply increases right ventricular afterload, causing acute RV dilation, wall strain, and increased myocardial oxygen demand. As RV wall stress increases, coronary perfusion pressure falls, precipitating RV ischemia and acute failure. The resulting leftward septal shift impairs LV filling, severely dropping stroke volume and cardiac index. [11–13]

Echocardiographic evidence of severe RV overload—specifically an RV/LV diameter ratio 1.2—was strongly associated with mortality (aOR: 2.94). While mild RV dilation (RV/LV > 0.9) is common in acute PE, a ratio exceeding 1.2 reflects significant volume and pressure overload, correlating with severe loss of RV stroke volume and high risk of hemodynamic collapse. [14, 15]

Biochemical markers provided important prognostic insight. Initial serum lactate 3.5 { mmol/L} served as an independent predictor of mortality (aOR: 2.51). Elevated lactate in acute PE reflects systemic tissue hypoperfusion, anaerobic metabolism, and acute organ dysfunction secondary to cardiogenic/obstructive shock. Furthermore, development of stage 2–3 AKI was associated with a more than 2-fold increase in the odds of death (aOR: 2.18), highlighting the downstream impact of persistent renal venous congestion and systemic arterial hypotension on end-organ perfusion. [16–18]

Limitations of this study include its retrospective design, potential for unmeasured confounding, and variability in the timing of advanced reperfusion therapies (systemic thrombolysis vs. catheter-directed interventions) across treating ICU teams.

In conclusion, early risk stratification combining hemodynamic parameters (obstructive shock), echocardiographic markers (RV/LV ratio 1.2), and perfusion

biomarkers (serum lactate, renal clearance) is essential for identifying high-risk PE patients in the ICU. Timely identification of these risk factors may help guide aggressive therapeutic decisions, including early advanced reperfusion therapies or mechanical circulatory support (e.g., VA-ECMO).

Conclusion

ICU mortality among critically ill patients with acute pulmonary embolism remains substantial, particularly in those presenting with high-risk PE. Obstructive shock, severe echocardiographic RV strain (RV/LV ratio 1.2), elevated admission lactate, and acute kidney injury serve as key independent predictors of mortality. Integrating these parameters upon ICU admission allows for rapid identification of deteriorating patients who may benefit from early escalation of care.

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