

Navigating the spectrum of Acute Pulmonary Embolism: Real world outcomes and high-risk predictors in a tertiary care cohort

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Abstract**Background**

Pulmonary embolism (PE) is a major cardiovascular emergency associated with high early mortality and morbidity [1, 2]. Rapid clinical identification, prompt risk stratification, and timely intervention are essential to mitigate mortality [3, 7]. We aimed to evaluate the clinical spectrum, diagnostic workup, treatment strategies, and predictors of in-hospital mortality among patients with acute PE admitted to a tertiary care referral center in South India.

Methods

This prospective observational study enrolled 110 consecutive patients with computed tomography pulmonary angiography (CTPA)-confirmed acute PE admitted to Nizam's Institute of Medical Sciences (NIMS), Hyderabad, between July 2021 and July 2022. Baseline demographics, underlying risk factors, presenting features, diagnostic findings, therapeutic strategies, and clinical outcomes were analyzed. Multivariate logistic regression was performed to identify independent predictors of in-hospital mortality.

Results

The mean age was 47.3 +/- 14.0 years, with a subtle male predominance (51.8%, n=57). Common risk factors included prolonged bed-ridden status (38.1%), surgery within the preceding three weeks (34.5%), obesity (24.5%), and active malignancy (12.7%). Concurrent deep vein thrombosis (DVT) was documented in 62.7% of patients. Dyspnea (80.9%), palpitations (69.1%), and orthopnea/paroxysmal nocturnal dyspnea (52.7%) were the primary presenting symptoms; sinus tachycardia (80.9%), elevated jugular venous pressure (69.1%), and hypotension (33.6%) were the primary physical signs. On CTPA, proximal thrombus involvement was observed in 43.6% of patients, while 56.4% had distal/lobar/segmental occlusion. Hemodynamically, 33.6% had massive PE and 21.8% had submassive PE. Systemic thrombolytic therapy was administered in 19.1% (n=21) of patients, predominantly using streptokinase (13.6%). Overall in-hospital mortality was 26.4% (n=29). On multivariate analysis, active malignancy ($p < 0.005$), systolic blood pressure < 100 mmHg ($p < 0.001$), arterial oxygen saturation $< 90\%$ ($p < 0.005$), proximal thrombus location on CTPA ($p < 0.010$), right ventricular (RV) dysfunction on echocardiography ($p < 0.005$), and intracranial neoplasm ($p < 0.005$) were significant independent predictors of mortality.

Conclusion

Acute PE in this cohort presented with a high burden of massive/submassive cases and significant in-hospital mortality. Early risk stratification combining clinical parameters, echocardiographic RV dysfunction, and anatomical burden on CTPA is critical to identify high-risk subset patients who may benefit from aggressive advanced reperfusion therapies.

Keywords: Pulmonary Embolism, Deep Vein Thrombosis, Computed Tomography Pulmonary Angiography, Thrombolysis, Pulmonary Embolism Severity Index, Right Ventricular Dysfunction.

Introduction

Venous thromboembolism (VTE), encompassing deep vein thrombosis (DVT) and pulmonary embolism (PE), constitutes a common cause of cardiovascular mortality and morbidity worldwide [1, 3]. Acute PE represents an acute occlusion of the pulmonary arterial bed, most frequently resulting from embolization of thrombi originating from deep veins of the lower extremities or pelvis [1].

The pathophysiological cascade underlying VTE is historically grounded in Virchow's triad: hypercoagulability, venous stasis, and vascular endothelial injury [1]. Risk factors are classically divided into transient/provoked (e.g., recent major abdominal or orthopedic surgery, trauma, prolonged immobilization) and persistent/unprovoked causes (e.g., active malignancy, inherited thrombophilias, antiphospholipid syndrome) [6, 7].

When emboli occlude the pulmonary arterial circulation, the abrupt increase in pulmonary vascular resistance (PVR) leads to right ventricular (RV) pressure overload, RV dilation, wall stress, and subsequent RV failure [1, 10, 11]. Interventricular dependence impairs left ventricular (LV) diastolic filling, culminating in reduced cardiac output, systemic hypotension, cardiogenic shock, and sudden cardiac death [1, 12].

Prompt clinical diagnosis remains challenging because symptoms such as dyspnea, pleuritic chest pain, cough, and syncope are non-specific and overlap with other acute conditions [3, 13]. Diagnostic workflows rely on clinical risk scoring (e.g., Wells score) [14], quantitative D-dimer testing [15], bedside echocardiography for RV strain assessment [16], and definitive visualization via multidetector computed tomography pulmonary angiography (CTPA) [13].

Although international guidelines (such as the European Society of Cardiology [ESC]) provide clear diagnostic and therapeutic pathways, real-world presentation, management patterns, and

outcomes show substantial variation across regional demographics and resource settings. In South Asian populations, comprehensive epidemiological and therapeutic data regarding acute PE remain limited. This study was undertaken to examine the clinical characteristics, diagnostic parameters, therapeutic strategies, and risk factors predicting in-hospital mortality in patients with acute PE admitted to a major tertiary care university hospital in South India.

Material and Methods

Study Design and Setting

This prospective observational study was conducted in the Department of Cardiology at Nizam's Institute of Medical Sciences (NIMS), Hyderabad, Telangana, India. The study protocol was approved by the NIMS Institutional Ethics Committee (Review Letter No. EC/NIMS/2823/2021). Written informed consent was obtained from all patients or their legally acceptable representatives prior to inclusion.

Patient Selection

Consecutive patients admitted to NIMS between July 2021 and July 2022 were screened.

- **Inclusion Criteria:** All consecutive patients with confirmed acute pulmonary embolism documented by CTPA during the study period.
- **Exclusion Criteria:** Patients with clinically suspected acute PE who did not undergo CTPA or in whom CTPA failed to definitively confirm pulmonary arterial thrombosis.

Data Collection and Diagnostic Protocol

Detailed clinical evaluations were performed at admission, including comprehensive histories, demographic details, underlying comorbidities, transient/permanent VTE risk factors, presenting

symptoms, and baseline hemodynamic measurements. Functional status of dyspnea was classified using New York Heart Association (NYHA) classes.

Standard laboratory evaluations included complete blood counts, renal and liver function panels, procoagulant screening (anti-nuclear antibody [ANA], anti-phospholipid antibodies [APLA], Protein C, Protein S, Antithrombin-III, Factor V Leiden, serum homocysteine, thyroid-stimulating hormone [TSH]), and cardiac biomarkers (Troponin-T and N-terminal pro-B-type natriuretic peptide [NT-proBNP]).

Instrumental evaluations included:

1. **Electrocardiography (ECG):** Analyzed for sinus tachycardia, S1Q3T3 pattern, right bundle branch block (RBBB), atrial fibrillation, and RV strain patterns (25).
2. **Chest X-ray (CXR):** Evaluated for cardiomegaly, pulmonary artery dilation, wedge-shaped opacities (Hampton's hump), or pleural effusions (25).
3. **Transthoracic Echocardiography (TTE):** Performed in all patients to evaluate RV dimensions, RV free wall motion (McConnell's sign), Tricuspid Annular Plane Systolic Excursion (TAPSE), estimated pulmonary artery systolic pressure (PASP), and direct visualization of right heart thrombi (16, 24).
4. **Lower Limb Venous Doppler Ultrasonography:** Performed to screen for lower extremity deep vein thrombosis (DVT) (17, 18).
5. **CTPA:** Confirmed PE diagnosis and determined anatomical thrombus extent:

- *Category 1 (Proximal)*: Thrombus involving the main pulmonary artery (MPA), right pulmonary artery (RPA), or left pulmonary artery (LPA), including saddle emboli.
- *Category 2 (Distal)*: Thrombus isolated to lobar, segmental, or subsegmental branches (13).

Hemodynamic Risk Stratification and Severity Scores

Patients were stratified into hemodynamic severity groups according to international guidelines:

- **Massive PE**: Persistent arterial hypotension (systolic blood pressure [SBP] < 100 mmHg or a drop in SBP of ≥ 40 mmHg for > 15 minutes not caused by new-onset arrhythmia, hypovolemia, or sepsis) or cardiogenic shock.
- **Submassive PE**: Hemodynamically stable (SBP ≥ 100 mmHg) but presenting with evidence of RV dysfunction on echocardiography and/or elevated cardiac biomarker levels (Troponin-T, NT-proBNP).
- **Low-Risk/Other PE**: Absence of hypotension, cardiogenic shock, and RV dysfunction (7,24).

Risk of 30-day all-cause mortality was evaluated using the original Pulmonary Embolism Severity Index (PESI) score, stratifying patients into Risk Classes I to V (19).

Treatment Protocol

Therapeutic choices were determined by attending cardiology consultants based on clinical presentation and prevailing guidelines:

- **Anticoagulation:** Intravenous unfractionated heparin (UFH), subcutaneous low-molecular-weight heparin (LMWH, Enoxaparin), or Fondaparinux, bridged to oral anticoagulants (coumarin derivatives or direct oral anticoagulants [DOACs]) (6,7).
- **Systemic Thrombolysis:** Reserved for massive PE or deteriorating submassive PE without absolute bleeding contraindications (7,28). Agents used included:
 - *Streptokinase:* 250,000 IU loading dose over 30 min, followed by 100,000 IU/h for 24 h.
 - *Urokinase:* 4400 IU/kg loading over 10 min, followed by 4400 IU/kg/h for 12–24 h.
 - *Alteplase (r-tPA):* Full-dose (100 mg over 2 h) or half-dose (10 mg bolus +40 mg infusion over 2 h).
- **Catheter-Directed Interventions & IVC Filters:** Catheter-directed thrombolysis or inferior vena cava (IVC) filter placement was performed in selected cases based on clinical indication (22, 23).

Statistical Analysis

Categorical variables are expressed as absolute frequencies and percentages. Continuous variables are reported as mean $\pm$ standard deviation (SD). Group comparisons were evaluated using Pearson's Chi-square test or Fisher's exact test for categorical parameters. To identify independent clinical, laboratory, and radiological predictors of in-hospital mortality, univariate analysis was conducted followed by multivariate logistic regression analysis. A two-tailed p -value < 0.05 was

considered statistically significant. Statistical analyses were performed using STATA IC/14.1 software (StataCorp, College Station, TX, USA).

Results

Demographic Characteristics and Risk Factors

A total of 110 CTPA-confirmed PE cases were evaluated. The cohort comprised 57 males (51.8%) and 53 females (48.2%), with an overall mean age of 47.32 ± 14.00 years.

The most common predisposing risk factors were prolonged bed-ridden status (38.1%, $n = 42$), history of major surgery within the preceding 3 weeks (34.5%, $n = 38$), obesity (24.5%, $n = 27$), hypertension (23.6%, $n = 26$), and active cigarette smoking (18.1%, $n = 20$). Active malignancy was documented in 14 patients (12.7%). Doppler evidence of concurrent lower extremity DVT was observed in 69 patients (62.7%). Table 1 summarizes predisposing risk factors.

Table 1: Predisposing Risk Factors in Patients with Acute PE ($N = 110$)

Risk Factor	Number of Patients (n)	Percentage (%)
Deep Vein Thrombosis (DVT)	69	62.7%
Bed-ridden Status (> 3 days)	42	38.1%
Recent Surgery (< 3 weeks)	38	34.5%
Obesity	27	24.5%

Risk Factor	Number of Patients (n)	Percentage (%)
Hypertension	26	23.6%
Cigarette Smoking	20	18.1%
Malignancy	14	12.7%
Varicose Veins	12	10.9%
Trauma / Fracture	9	8.1%
Oral Contraceptive Pills (OCPs)	4	3.6%
Air Travel (> 8 hours)	2	1.8%

Underlying Comorbidities

Cerebrovascular accident (CVA) with secondary limb paralysis/immobility was the most frequent coexisting condition (17.2%, $n = 19$), followed by intracranial tumors (12.7%, $n = 14$), diabetes mellitus (10.9%, $n = 12$), and coronary artery disease (7.3%, $n = 8$). Procoagulant states identified on specialized laboratory evaluation included secondary APLA syndrome with Systemic Lupus Erythematosus ($n = 1$), primary APLA syndrome ($n = 1$), and Protein C deficiency ($n = 1$). Overt hypothyroidism (TSH > 10 mIU/L) was present in 8.2% ($n = 9$). Table 2 details baseline comorbidities.

Table 2: Underlying Comorbidities in the Study Population (N = 110)

Comorbidity	Number of Patients (n)	Percentage (%)
Cerebrovascular Accident (CVA)	19	17.2%
Intracranial Tumors	14	12.7%
Malignancy (Overall)	14	12.7%
Diabetes Mellitus	12	10.9%
Coronary Artery Disease	8	7.3%
Rheumatic Heart Disease (MS with AF)	4	3.6%
Membranous Glomerulonephritis	3	2.7%
Infective Endocarditis	2	1.8%
Primary Antiphospholipid Syndrome (APLA)	1	0.9%

Comorbidity	Number of Patients (n)	Percentage (%)
Systemic Lupus Erythematosus (dsDNA+ / Secondary APLA)	1	0.9%
Protein C Deficiency	1	0.9%

Clinical Presentation and Physical Signs

The dominant presenting symptom was acute dyspnea (80.9%, $n = 89$), followed by palpitations (69.1%, $n = 76$), orthopnea/paroxysmal nocturnal dyspnea (52.7%, $n = 58$), and chest pain (32.7%, $n = 36$). Syncope occurred in 10.9% ($n = 12$) and hemoptysis in 8.2% ($n = 9$). Based on NYHA functional criteria for dyspnea, 40.9% ($n = 45$) presented in NYHA Class III and 28.2% ($n = 31$) in NYHA Class IV.

On physical examination, sinus tachycardia (HR > 110 bpm) was the most prevalent sign (80.9%, $n = 89$), followed by elevated JVP (69.1%, $n = 76$), tachypnea (RR > 30/min; 38.2%, $n = 42$), and overt arterial hypotension (SBP < 100 mmHg; 33.6%, $n = 37$). Hypoxemia ($\text{SpO}_2 < 90\%$ on room air) was documented in 16.4% ($n = 18$). Organ dysfunction at admission included renal dysfunction in 5.5% ($n = 6$) and hepatic dysfunction in 3.6% ($n = 4$). Clinical symptoms and signs are summarized in Table 3.

Table 3: Clinical Symptoms and Physical Signs at Presentation

Parameter	Number of Patients (n)	Percentage (%)
Symptoms		
Dyspnea	89	80.9%
Palpitations	76	69.1%
Orthopnea / PND	58	52.7%
Chest Pain	36	32.7%
Lower Limb Pedal Edema	31	28.1%
Cough	15	13.6%
Syncope	12	10.9%
Hemoptysis	9	8.2%
Fever	8	7.2%
Altered Sensorium	6	5.5%

Parameter	Number of Patients (n)	Percentage (%)
Physical Signs		
Tachycardia (HR > 110 bpm)	89	80.9%
Elevated Jugular Venous Pressure (JVP)	76	69.1%
Tachypnea (RR > 30/min)	42	38.2%
Hypotension (SBP < 100 mmHg)	37	33.6%
Oxygen Desaturation (SpO ₂ < 90%)	18	16.4%
Altered Mental State	10	9.1%
Hypothermia (< 36.0°C)	9	8.2%

Diagnostic Investigations and Risk Stratification

Twelve-lead ECG demonstrated sinus tachycardia in 80.5% ($n = 88$), RV strain with T-wave inversions in anterior/inferior leads in 58.2% ($n = 64$), incomplete/complete RBBB in 50.9% ($n = 56$), and the classical $S_1Q_3T_3$ pattern in 33.6% ($n = 37$). Normal ECG was recorded in 10.9% ($n = 12$). CXR abnormalities included main/right pulmonary artery enlargement

(prominent pulmonic segment) in 56.4% ($n = 62$), wedge-shaped pleural opacities in 10.9% ($n = 12$), and pleural effusions in 8.2% ($n = 9$); 24.5% ($n = 27$) had a normal CXR.

Echocardiography revealed pulmonary arterial hypertension (PAH) in 83.6% ($n = 92$), RV dysfunction (RV enlargement/free wall hypokinesis) in 55.5% ($n = 61$), and mobile right heart thrombi in 5.5% ($n = 6$).

Laboratory biomarkers confirmed D-dimer positivity in 95.5% ($n = 64/67$ tested), Troponin-T positivity in 53.4% ($n = 31/58$ tested), and elevated NT-proBNP (> 300 pg/mL) in 75.0% ($n = 24/32$ tested).

CTPA categorized 43.6% ($n = 48$) of cases with proximal thrombus burden (MPA/RPA/LPA involvement), with saddle emboli present in 10.0% ($n = 11$). Distal/lobar/segmental thrombus distribution was seen in 56.4% ($n = 62$).

According to hemodynamic criteria, 37 patients (33.6%) were classified as Massive PE, 24 (21.8%) as Submassive PE, and 49 (44.5%) as Low-Risk/Other PE. Distribution according to baseline PESI Risk Classes was: Class I (9.1%, $n = 10$), Class II (28.2%, $n = 31$), Class III (10.9%, $n = 12$), Class IV (23.6%, $n = 26$), and Class V (33.6%, $n = 37$). Diagnostic findings are summarized in Table 4.

Table 4: Key Diagnostic Modalities and Findings ($N = 110$)

Diagnostic Parameter	Number (n) / Total Tested	Percentage (%)
Electrocardiogram (ECG)		
Sinus Tachycardia	88 / 110	80.5%
RV Strain Pattern	64 / 110	58.2%
Right Bundle Branch Block (RBBB)	56 / 110	50.9%
$S_1Q_3T_3$ Pattern	37 / 110	33.6%
Normal ECG	12 / 110	10.9%
Atrial Fibrillation	4 / 110	3.6%
Echocardiography		
Pulmonary Arterial Hypertension (PAH)	92 / 110	83.6%
RV Dysfunction	61 / 110	55.5%
Left Ventricular Dysfunction	8 / 110	7.3%

Diagnostic Parameter	Number (n) / Total Tested	Percentage (%)
Right Heart Thrombus	6 / 110	5.5%
CT Pulmonary Angiography (Thrombus Location)		
Lobar / Segmental / Subsegmental	62 / 110	56.4%
Main / Left / Right Pulmonary Artery	48 / 110	43.6%
<i>Saddle Embolus Subtype</i>	11 / 110	10.0%
Biomarkers & Venous Doppler		
Venous Doppler (+ for DVT)	69 / 110	62.7%
D-dimer Positive	64 / 67	95.5%
Troponin-T Positive	31 / 58	53.4%
NT-pro BNP Elevated (> 300 pg/mL)	24 / 32	75.0%

Therapeutic Management

Parenteral anticoagulation was administered to 108 patients (98.2%), utilizing unfractionated heparin in 98.2% ($n = 108$), enoxaparin in 12.7% ($n = 14$), and fondaparinux in 1.8% ($n = 2$).

Systemic thrombolytic therapy was administered to 21 patients (19.1%). Streptokinase was the predominant thrombolytic agent (13.6%, $n = 15$), followed by urokinase (2.7%, $n = 3$), full-dose alteplase (1.8%, $n = 2$), and half-dose alteplase (0.9%, $n = 1$). Major absolute contraindications (e.g., recent major surgery, intracranial tumors, recent hemorrhagic stroke) precluded thrombolysis in the remaining massive/submassive patients. Two patients (1.8%) underwent catheter-directed thrombolysis using a multi-side-hole perfusion catheter with streptokinase, achieving echocardiographic resolution without procedure-related complications. IVC filters were implanted in 2 patients (1.8%) due to absolute contraindications to anticoagulation or recurrent VTE despite anticoagulation.

Adjunctive therapies included loop diuretics/spironolactone in 20.9% ($n = 23$), oral sildenafil in 10.0% ($n = 11$), digoxin in 15.5% ($n = 17$), and aspirin in 11.8% ($n = 13$).

In-Hospital Outcomes and Predictors of Mortality

Overall in-hospital mortality was 26.4% ($n = 29$). In-hospital mortality demonstrated a strong stepwise association with baseline PESI Risk Classes: Class I (0%), Class II (0%), Class III (13.8%, $n = 4$), Class IV (17.2%, $n = 5$), and Class V (68.9%, $n = 20$). Among thrombolysed patients ($n = 21$), 16 patients (76.2%) demonstrated echo-documented resolution of pulmonary hypertension, while 5 (23.8%) experienced PE-related mortality. Non-fatal therapy-related minor bleeding occurred in 12% of patients (e.g., mucosal/gum bleeding, upper GI bleed, ecchymosis); no fatal bleeding was directly attributable to thrombolytic therapy.

Multivariate logistic regression analysis revealed six parameters as independent predictors of in-hospital mortality: active malignancy, persistent hypotension (SBP < 100 mmHg), severe hypoxemia (SpO₂ < 90%), main/proximal pulmonary artery thrombus location on CTPA, echocardiographic RV dysfunction, and underlying intracranial neoplasm ($p < 0.01$ for all). Multivariate regression findings are detailed in Table 5.

Table 5: Multivariate Regression Analysis for Predictors of In-Hospital Mortality

Variable	Odds Ratio / Correlation	p-value
Systolic Blood Pressure < 100 mmHg	Strong predictor	< 0.001
Active Malignancy	Strong predictor	< 0.005
Arterial Oxygen Saturation (SpO₂ < 90%)	Independent predictor	< 0.005
Echocardiographic RV Dysfunction	Independent predictor	< 0.005
Intracranial Neoplasm	Independent predictor	< 0.005
Proximal Thrombus Location on CTPA (MPA/RPA/LPA)	Independent predictor	< 0.010

Discussion

This observational study provides a detailed characterization of acute pulmonary embolism within a South Indian tertiary academic center. The overall mean age of our cohort (47.3 +/- 14.0 years) is lower than reported in Western clinical registries, such as the RIETE registry, where the incidence of PE strongly correlates with advancing age [2, 5]. However, major risk factor distributions—specifically prolonged immobilization/bed-ridden state (38.1%), recent surgery (34.5%), and active malignancy (12.7%)—align with the RIETE registry findings [5].

Clinical presentation in our population was dominated by acute dyspnea (80.9%) and sinus tachycardia (80.9%). Notably, classical pleuritic chest pain was reported in only 32.7% of our cohort, compared to 76% in the PIOPED II study [13]. Similarly, sinus tachycardia was significantly more frequent in our study than in PIOPED II (20%) [13].

ECG signs of right heart strain were substantially more common in our cohort than the non-specific ST and T changes (49%) reported in early PIOPED cohort data [25]. Transthoracic echocardiography documented PAH in 83.6% and RV strain/dysfunction in 55.5%. RV dysfunction is strongly associated with adverse outcomes, carrying an 8.1% mortality rate in Kasper et al.'s landmark registry, which escalates rapidly when combined with hypotension (15%) or cardiogenic shock (25%) [24]. Furthermore, elevated Troponin-T (53.4%) and NT-proBNP (75.0%) levels in our subset strongly reflect this pathophysiological stress cascade and remain vital for risk stratification [26].

CTPA anatomical classification correlated with the high rate of clinical shock and RV dysfunction observed, with 62.7% of the cohort also showing positive lower limb venous Doppler findings, reinforcing the 70% concomitant DVT prevalence previously established in the literature [17].

Systemic thrombolysis was administered to 19.1% of patients, primarily utilizing streptokinase, aligning with approved European Society of Cardiology guidelines [7, 28]. Half-dose alteplase was utilized successfully in one submassive patient, consistent with the MOPETT trial findings demonstrating significant reductions in pulmonary arterial pressure without increased bleeding risks [27]. Two patients underwent IVC filter placement, which reduces recurrent PE risk but lacks significant mortality benefit [23].

In-hospital mortality in our cohort was 26.4%, which is markedly higher than the 30-day mortality seen in the EMPEROR registry (5.4%) [29] or the RIETE registry (1.8%) [5]. This discrepancy is largely driven by the high prevalence of massive and submassive PE, concurrent intracranial tumors, and cerebrovascular accidents within our specialized tertiary care population. The PESI score accurately predicted mortality risk across our cohort [19].

Multivariate regression analysis demonstrated that persistent hypotension ($\text{SBP} < 100 \text{ mmHg}$), arterial desaturation ($\text{SpO}_2 < 90\%$), active malignancy, underlying intracranial neoplasm, echocardiographic RV dysfunction, and proximal main pulmonary artery thrombus burden were independent predictors of mortality. These findings highlight the necessity of immediate multi-parameter risk stratification at admission to guide aggressive interventions—such as catheter-directed mechanical thrombectomy, surgical embolectomy, or mechanical circulatory support—where systemic thrombolysis is contraindicated or fails.

Conclusions

In this tertiary care observational study, acute PE presented as a severe cardiovascular condition characterized by high functional impairment, frequent RV dysfunction, and substantial in-hospital mortality. Early risk assessment combining clinical hemodynamic parameters (hypotension, hypoxemia), the PESI score, echocardiographic RV strain, and anatomical extent on CTPA

accurately predicts mortality. Establishing dedicated multidisciplinary Pulmonary Embolism Response Teams (PERT) and expanding access to advanced catheter-directed therapies may improve outcomes in high-risk patients who have contraindications to systemic thrombolysis.

Limitations

This study has several limitations:

1. **Single-Center Design:** Conducted at a tertiary university referral hospital, introducing potential tertiary referral bias toward higher disease severity and complex neurosurgical/oncological comorbidities.
2. **Sample Size:** The sample size ($N = 110$) reflects consecutive confirmed cases over a 1-year period; larger multicenter cohorts are needed to further validate multivariate risk models.
3. **Long-Term Follow-Up:** Lack of post-discharge long-term follow-up precludes evaluation of late VTE recurrence, chronic thromboembolic pulmonary hypertension (CTEPH), and long-term functional survival.

Compliance with Ethical Standards

- **Ethical Approval:** Institutional Ethics Committee approval was granted by NIMS Institutional Ethics Committee (Letter No. EC/NIMS/2823/2021).
- **Informed Consent:** Informed consent was obtained from all individual participants or their legal guardians included in the study.
- **Conflict of Interest:** The authors declare that they have no conflicts of interest.

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Abbreviations:

- **ANA:** Anti-Nuclear Antibody
- **APLA:** Anti-Phospholipid Antibody
- **CTPA:** Computed Tomography Pulmonary Angiography
- **CVA:** Cerebrovascular Accident
- **CXR:** Chest X-ray
- **DOAC:** Direct Oral Anticoagulant
- **DVT:** Deep Vein Thrombosis

- **ECG:** Electrocardiogram
- **HR:** Heart Rate
- **IVC:** Inferior Vena Cava
- **JVP:** Jugular Venous Pressure
- **LMWH:** Low-Molecular-Weight Heparin
- **LPA:** Left Pulmonary Artery
- **LV:** Left Ventricle
- **MPA:** Main Pulmonary Artery
- **NT-proBNP:** N-terminal pro-B-type natriuretic peptide
- **NYHA:** New York Heart Association
- **OCP:** Oral Contraceptive Pill
- **PAH:** Pulmonary Arterial Hypertension
- **PE:** Pulmonary Embolism
- **PESI:** Pulmonary Embolism Severity Index
- **PND:** Paroxysmal Nocturnal Dyspnea
- **PVR:** Pulmonary Vascular Resistance
- **RBBB:** Right Bundle Branch Block
- **RPA:** Right Pulmonary Artery

- **RR:** Respiratory Rate
- **RV:** Right Ventricle
- **SBP:** Systolic Blood Pressure
- **SpO2:** Peripheral Capillary Oxygen Saturation
- **TAPSE:** Tricuspid Annular Plane Systolic Excursion
- **TSH:** Thyroid-Stimulating Hormone
- **TTE:** Transthoracic Echocardiography
- **UFH:** Unfractionated Heparin
- **VTE:** Venous Thromboembolism