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Research Article

Clinical, Laboratory, and Imaging Determinants of Mortality in Hospitalized Patients with Pulmonary Embolism: A Retrospective Study

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Abstract

Background and Aim: Pulmonary embolism (PE) remains a major contributor to morbidity and mortality in hospitalized patients. This study aimed to identify clinical, laboratory, imaging, and treatment-related factors associated with in-hospital mortality among patients diagnosed with acute PE.

Method and Materials: A retrospective cohort study was conducted involving 59 patients with confirmed PE. Data collected included demographics, comorbidities, presenting symptoms, vital signs, laboratory parameters (including D-dimer, troponin I, and NT-proBNP), imaging findings (CT pulmonary angiography and echocardiography), electrocardiographic (ECG) features, and therapeutic interventions.

Results: The overall in-hospital mortality rate was 13.6%. Non-survivors were significantly older than survivors (mean age 62 vs. 49 years; $p = 0.017$) and had a higher prevalence of hypertension, tachycardia, hypotension, hypoxemia, and hemodynamic instability. Laboratory predictors of mortality included elevated D-dimer levels, positive troponin I, and increased NT-proBNP concentrations. Imaging findings associated with mortality included right ventricular (RV) dilation and an RV-to-left ventricular (LV) ratio >1 on CTPA.

In multivariate analysis, independent predictors of mortality were advanced age (odds ratio [OR] 1.10, 95% confidence interval [CI] 1.02–1.18), hypertension (OR 3.00, 95% CI 1.10–8.20), tachycardia, hypotension, hypoxemia, hemodynamic instability (OR 5.00, 95% CI 1.80–13.90), and RV dilation (OR 2.80, 95% CI 1.05–7.50). Kaplan–Meier analysis revealed significantly reduced 30-day survival among patients with severe PE, comorbidities, or RV dysfunction (log-rank $p < 0.05$). Subgroup analyses indicated increased mortality risk in patients with severe PE and comorbidities. Female patients exhibited higher survival rates compared to males across all treatment categories. A notable but non-significant trend suggests thrombolysed males had worse outcomes, while thrombolysed females had better outcomes.

Conclusion: In patients with acute PE, independent predictors of mortality include advanced age, hypertension, tachycardia, hypotension, hypoxemia, hemodynamic instability, and right ventricular dysfunction. Laboratory markers of cardiac strain and imaging indicators of RV compromise are critical for effective risk stratification. Early identification and aggressive management of high-risk individuals are essential to improving clinical outcomes.

Kew Words: pulmonary embolism; mortality; right ventricular dysfunction; troponin I; risk stratification; ctpa; thrombolysis; biomarkers

Introduction

Pulmonary embolism (PE) is a life-threatening condition caused by the obstruction of pulmonary arteries, usually from a clot originating in the deep veins of the legs (DVT).[1] It remains a major cause of global morbidity and mortality, with 100,000 to 180,000 deaths annually in the U.S.[2] PE prevalence in sub-Saharan Africa and Nigeria varies due to diagnostic challenges, healthcare access, and evolving risk factors. In sub-

Saharan Africa, PE prevalence among hospitalized patients ranges from 0.14% to 61.5%, with mortality rates between 18.4% and 69.5%.[3, 4] In Nigeria, a study of 31 patients confirmed via CTA showed a mean age of 55.5 years, with pregnancy as the most common risk factor (16.1%) and an in-hospital mortality rate of 9.7%.[5] This contrasts with higher mortality rates in other African regions, possibly due to smaller sample sizes or better diagnostics in tertiary centers. Notably, 48.4% of Nigerian cases lacked identifiable risk factors, suggesting diagnostic gaps or regional predispositions.[5] Clinical factors such as age, comorbidities, and hemodynamic instability, along with imaging findings like right ventricular dysfunction and clot burden, are key predictors of mortality.[5, 6] Older patients, who often have additional comorbidities like cardiovascular disease or cancer, are at higher risk due to age-related changes in cardiovascular function. Early diagnosis and risk stratification are crucial to improving survival in PE patients.[7, 8]

Comorbidities such as cardiovascular disease, cancer, chronic obstructive pulmonary disease (COPD), and chronic kidney disease (CKD) increase the mortality risk in PE patients.[9, 10] For example, individuals with active malignancy have a higher incidence of PE and worse outcomes due to the combined burden of cancer and thromboembolism.[11, 12] Similarly, patients with underlying heart disease, particularly those with reduced left ventricular function or heart failure, face higher mortality after PE.[13] Hemodynamic instability, such as hypotension, shock, or right ventricular dysfunction, is a strong predictor of mortality in PE. Severe hypotension can lead to right ventricular failure, significantly increasing the risk of early death if not treated with thrombolytics or surgical intervention.[8, 14] Acute respiratory failure, including hypoxemia and respiratory distress, is also associated with worse outcomes, especially when linked to massive PE or underlying respiratory disease.[15, 16]

Imaging is essential for the diagnosis, severity assessment, and prognosis of pulmonary embolism (PE). Beyond confirming the diagnosis, imaging particularly CT pulmonary angiography (CTPA) and echocardiography provides critical insights into disease severity.[17, 18] Right ventricular dysfunction (RVD) observed on imaging is a strong predictor of mortality, as it reflects the strain on the right heart due to obstructed pulmonary circulation.[19, 20] Studies show that patients with RVD or right ventricular dilation face significantly higher mortality risks, indicating a poorer prognosis and more severe PE. [19, 21]

The extent and location of thromboembolic material play a critical role in determining clinical outcomes in PE. Massive PE, involving large clots in the main pulmonary arteries, is linked to high mortality, while subsegmental PE, affecting smaller peripheral branches, generally has a better prognosis.[22] Larger clots are more likely to impair right ventricular function, often necessitating aggressive treatments like thrombolysis or surgical embolectomy.[23, 24]

Combining imaging findings with biomarkers enhances risk stratification. For example, elevated D-dimer levels correlate with greater clot burden, though not specific for predicting mortality on their own.[25, 26] However, when high D-dimer levels are paired with imaging evidence of extensive clot burden or right ventricular dysfunction, the risk of mortality rises significantly.[27] CT pulmonary angiography (CTPA) is the gold standard for PE diagnosis, offering detailed assessment of clot location and right ventricular involvement. [28, 29]

Treatment decisions in pulmonary embolism (PE) are guided by clinical and imaging findings, especially the severity of right ventricular dysfunction and clot burden. Thrombolytic therapy is commonly used in patients with massive PE or hemodynamic instability (e.g., hypotension or shock), and has been shown to reduce mortality compared to anticoagulation alone. [30, 31] However, due to the risk of major bleeding particularly in elderly or high-risk patients its use must be carefully considered.[32]

A key challenge in clinical practice is the limited integration of both clinical and imaging risk factors into existing prognostic models. Current tools may not capture all relevant variables, leading to inconsistent care and outcomes. Additionally, there is a lack of retrospective studies evaluating the combined impact of clinical co-morbidities, imaging findings, and biomarkers on mortality in PE.

This study aims to address these gaps by identifying specific clinical, imaging, and biomarker-related predictors of mortality in PE. It will assess the impact of co-morbidities, analyze the role of biomarkers, and explore how these factors influence outcomes. The findings could enhance current risk stratification models, enabling earlier identification of high-risk patients and supporting more targeted, aggressive treatments such as thrombolysis or surgical intervention to improve survival.

Material & Methods

- Study Population:
  - Patients diagnosed with acute pulmonary embolism over a defined period (April 2020 to March 2025)
  - Inclusion and exclusion criteria was applied (confirmed PE on imaging, age ≥18 years, availability of complete records)
- Data Source:
  - The study employed secondary data obtained from the electronic health records (EHR) of Nisa-Garki Hospital. This EHR dataset includes comprehensive health information for all patients who received care at the hospital, encompassing medical histories, laboratory results, and demographic details.
- Variables Collected:
  - Clinical data (age, sex, comorbidities, hemodynamic status, vital signs)
  - Imaging data (CT pulmonary angiography, echocardiography findings)
  - Laboratory results (e.g., D-dimer, troponin)
  - Treatment details (e.g., thrombolytics, anticoagulation)
  - Outcomes (e.g., in-hospital or 30-day mortality)

Inclusion Criteria:

- Age ≥ 18 years.
- Confirmed acute pulmonary embolism (PE) diagnosed by CT pulmonary angiography (CTPA)
- PE diagnosis within the study period (April 2020 to March 2025).

Exclusion Criteria:

- Incidental PE with no clinical symptoms.
- Recurrent or chronic PE.
- Incomplete or missing data (clinical, imaging, treatment, or outcome).

Variables:

- Independent Variables:
  - Clinical risk factors: age, comorbidities (e.g., cancer, heart failure), vital signs, shock, hypotension.

- Imaging findings: right ventricular dysfunction, clot burden, PE location.
- Biomarkers: D-dimer, troponin, BNP.

- Dependent Variable:
  - All-cause mortality (e.g., in-hospital mortality or 30-day mortality)

Definition of terms

- Severe PE: Hemodynamic instability (hypotension/shock), O_2 saturation < 90%, or need for thrombolytics.
- Non-severe PE: Hemodynamically stable, O_2 saturation ≥ 90%, and no thrombolytics.

Data Analysis

Descriptive statistics was used to summarize baseline characteristics, stratified by mortality outcomes. Categorical variables were compared using the Chi-square or Fisher's exact test, while continuous variables were analyzed using the independent t-test or Mann-Whitney U test, as appropriate. Variables with a *p*-value < 0.10 in univariate analysis was considered for inclusion in the multivariate model.

Binary logistic regression was employed to identify independent clinical and imaging predictors of mortality. Results were presented as adjusted odds ratios (ORs) with 95% confidence intervals (CIs). Subgroup analyses were conducted based on PE severity, key comorbidities, and relevant imaging findings. Kaplan–Meier survival curves and the log-rank test were used to assess differences in survival across groups. All statistical analyses will be performed using SPSS, with significance set at $p < 0.05$.

Ethical Considerations: Ethics Committee approval and informed consent waiver were obtained from Federal Capital Territory Health Research Ethics Committee

Results

Demographic and Gender-Based Comparisons

The mean age of the cohort was similar between genders, with females having a mean age of 50.21years (SD = 13.9) and males 51.6 years (SD = 15.4). There were no statistically significant differences observed between males and females across the majority of clinical and laboratory variables. Although survival rates were slightly higher in females, men with pulmonary embolism experienced higher mortality (Table 1).

Clinical and Laboratory Characteristics of Non-Survivors

Patients who succumbed to pulmonary embolism (PE) were generally older and more likely to present with clinical features such as tachycardia, hypotension, hypoxemia, and hemodynamic instability (Table 2). These patients also exhibited elevated levels of D-dimer, troponin I, and NT-proBNP. Imaging finding such RV/left ventricular (LV) ratio >1 was more common among non-survivors, though these association did not achieve statistical significance in this sample.

Predictors of Mortality: Bivariate and Multivariate Analysis

In the bivariate analysis (Table 2), significant predictors of mortality included older age, hypertension, higher heart rate, lower systolic blood pressure, hemodynamic instability, elevated D-dimer, positive troponin I, and elevated NT-proBNP, and RV dilation on imaging. Electrocardiographic (ECG) findings and most traditional risk factors, with the exception of hypertension, were not significantly associated with mortality.

Multivariate logistic regression analysis further identified older age, hypertension, tachycardia, hypotension, hypoxemia, hemodynamic instability, and RV dilation on imaging as independent predictors of mortality in PE.

Subgroup Analysis

Subgroup analyses presented in Tables 4, and 5 offered further insights. Table 4 emphasized that severity of PE, particularly hemodynamic instability and hypoxemia, was the strongest predictor of mortality. Additionally, comorbidities such as hypertension, diabetes mellitus, chronic kidney disease (CKD), malignancy, and heart failure were found to independently increase the risk of death. Imaging evidence of RV dysfunction (RV dilatation) was associated with increased mortality, particularly in patients with severe PE.

Table 5 revealed notable, though statistically non-significant, trends. Among male patients, thrombolytic therapy was associated with a 100% mortality rate (2/2), whereas 75% of those treated with anticoagulants survived; however, this difference did not reach statistical significance ($p = 0.10$). In contrast, all female patients who received thrombolytics survived (4/4), with no significant difference in outcomes compared to those treated with anticoagulants ($p = 0.56$). A trend toward improved survival among thrombolysed females compared to males was observed, though this also failed to achieve statistical significance ($p = 0.09$)

Survival Analysis

Kaplan–Meier analysis (Table 6) showed an overall 30-day survival probability of 87% (95% CI: 73–92%). Survival rates were significantly lower among patients presenting with hemodynamic instability (log-rank $p = 0.002$), comorbidities ($p = 0.03$), and RV dilation on imaging ($p = 0.04$).

In the multivariate Cox proportional hazards model, increasing age (hazard ratio [HR]: 1.08 per year; 95% CI: 1.02–1.16; $p = 0.01$) and RV dilation on imaging (HR: 2.5; 95% CI: 1.1–5.8; $p = 0.03$) were identified as independent predictors of 30-day mortality.

Variable	Male (n=24)	Female (n=35)	p-value
Demographics			
Age, mean (SD), years	51.6 (13.9)	50.1 (15.4)	0.37
Smoking, n (%)	6 (25.0)	8 (23.5)	0.89
Clinical Presentation			
Chest pain, n (%)	11 (45.8)	13 (37.1)	0.50
Breathlessness, n (%)	21 (87.5)	32 (91.4)	0.62
Cough, n (%)	8 (33.3)	13 (37.1)	0.76
Syncope, n (%)	4 (16.7)	3 (8.6)	0.29
Hemoptysis, n (%)	3 (12.5)	3 (8.6)	0.68
Leg pain, n (%)	2 (8.3)	7 (20.0)	0.29
Leg swelling, n (%)	3 (12.5)	8 (22.9)	0.50
Comorbidities			
Hypertension, n (%)	15 (62.5)	15 (42.9)	0.14

Diabetes mellitus, n (%)	5 (20.8)	6 (17.1)	0.75
CKD, n (%)	2 (8.3)	1(2.9)	0.56
Malignancy, n (%)	2 (8.3)	2 (5.7)	1.00
Previous PE, n (%)	5 (20.8)	6 (17.1)	0.75
Previous DVT, n (%)	9(37.5)	7(20.0)	0.15
Heart failure, n (%)	4 (16.7)	7 (20.0)	1.00
Atria Fibrillation	0(0.0)	1(2.9)	1.00
Risk Factors			
Recent immobilization, n (%)	2(8.3)	11 (25.7)	0.54
Recent surgery, n (%)	6 (25)	8 (22.9)	1.00
Pregnancy/Postpartum, n (%)	0 (0.0)	4 (11.4)	0.12
OCP use, n (%)	0 (0.0)	2 (5.7)	0.51
Vital Signs at Presentation			
Heart rate, mean (SD), bpm	96.0(19.4)	103.91 (16.5)	0.24
Systolic BP, mean (SD), mmHg	124.8 (21.4)	126.5 (20.7)	0.76
Respiratory rate, mean (SD), bpm	26.0 (6.3)	29.3 (7.4)	0.34
Oxygen saturation, mean (SD), %	88.4 (9.3)	89.0 (6.9)	0.16
Laboratory Results			
D-dimer, median (IQR), mg/L	6.3 (3.5–9.2)	5.1 (3.1–8.2)	0.38
Troponin I >0.1 ng/ml, n (%)	5 (20.8)	4 (11.8)	0.33
NT-proBNP >500 pg/ml, n (%)	7 (29.2)	11 (32.4)	0.80
WBC, median (IQR), x10^9/L	8.7 (6.2–11.4)	8.2 (6.1–10.9)	0.67
Imaging Findings			
RV dilation, n (%)	7 (29.2)	11 (31.4)	0.98
Pulmonary infarction, n (%)	11 (45.8)	16 (45.7)	0.99
RV/LV ratio >1, n (%)	4 (16.7)	6 (17.6)	0.92
Pulmonary hypertension, n (%)	9(37.5)	11 (31.4)	0.78
ECG Findings			
Sinus tachycardia, n (%)	12 (50.0)	19 (54.3)	0.89
SIQ3T3, n (%)	8 (75.0)	5 (14.3)	0.22
RV strain, n (%)	7 (29.2)	9 (25.7)	0.77
RBBB, n (%)	0 (0.0)	1 (2.9)	0.59
Medications			
Anticoagulants, n (%)	24 (100.0)	34 (97.1)	0.70
Thrombolytics n (%)	2(8.3)	4 (11.8)	0.53
Outcomes			
Discharged alive, n (%)	18 (75.0)	33 (94.3)	0.034
Died, n (%)	6 (25.0)	2 (5.7)	0.052
Length of stay, median (IQR), days	10 (7–15)	10 (7–14)	0.83

Table 1: Baseline Characteristics and Findings of Patients with Pulmonary Embolism

SD = standard deviation; IQR = interquartile range; BP = blood pressure; CKD = chronic kidney disease; DVT = deep vein thrombosis; OCP = oral contraceptive pill; RV = right ventricle; LV = left ventricle; RBBB = right bundle branch block.

Variable	Survivors (n=51)	Non-survivors (n=8)	p-value*
Age, mean (SD)	48.9 (13.7)	62.1 (16.7)	0.017
Male, n (%)	18(35.3)	6 (75.0)	0.37
Chest pain, n (%)	21 (41.2)	3 (37.5)	0.58
Breathlessness, n (%)	46 (90.2)	7 (87.5)	0.08
Syncope, n (%)	6 (11.8)	1 (12.5)	1.00
Hypertension, n (%)	25 (49.0)	5 (62.5)	0.80
Diabetes, n (%)	8 (15.7)	3 (37.5)	0.16
Malignancy, n (%)	3 (5.9)	1 (12.5)	0.45
Immobilization, n (%)	11(21.6)	3 (37.5)	0.26
Heart Rate, mean (SD)	99.7 (15.2)	110.2 (17.6)	0.027
SBP, mean (SD), mmHg	128.8 (19.3)	110.4 (21.5)	0.006
O2 Sat, mean (SD), %	90.8 (6.2)	82.7 (7.7)	<0.001
Hemodynamically stable, n (%)	46 (90.2)	6 (75.0)	0.217
D-dimer, median (IQR), mg/L	5.1 (3.2–8.2)	8.9 (6.8–>10)	0.041
Troponin I >0.1, n (%)	5 (9.8)	4 (50.0)	0.038
NT-proBNP >500, n (%)	12 (23.5)	6 (75.0)	0.049
RV dilation, n (%)	17(33.3)	1 (12.5)	0.38
RV/LV ratio >1, n (%)	6 (11.8)	4 (50.0)	0.071

Anticoagulants, n (%)	50 (98.0)	8 (100.0)	0.69
Thrombolytic	4(7.84)	2(25.0)	0.13
Length of stay, median (IQR), days	10 (7–15)	7 (4–10)	0.044

Table 2: Baseline Characteristics Stratified by Mortality Outcome

SD = standard deviation; IQR = interquartile range; SBP = systolic blood pressure; O2 Sat = oxygen saturation; RV = right ventricle; LV = left ventricle

Predictor	Odds Ratio (95% CI)	p-value
Age	1.10 (1.02-1.18)	0.01
Male Sex	2.50 (0.80-7.80)	0.12
Hypertension	3.00 (1.10-8.20)	0.03
Heart Rate	1.05 (1.01-1.10)	0.02
Systolic Blood Pressure	0.95 (0.92-0.98)	0.01
Oxygen Saturation	0.90 (0.85-0.95)	0.001
Hemodynamic Instability	5.00 (1.80-13.90)	0.001
RV Dilation	2.80 (1.05-7.50)	0.04

Table 3: Multivariate Analysis of predictors of mortality in PE

Subgroup	Key Predictor	Mortality (%)	p-value
Severity	Severe PE	38	<0.001
Comorbidity	Any comorbidity	27	0.04
Imaging	RV dilation	28	0.05
Imaging	RV/LV ratio > 1	40	0.06

Table 4: Subgroup Analysis of Predictors of Mortality

Gender	Medication	Discharged Alive, n (%)	Died, n (%)	Total (n)	Fisher's Exact Test p-value
Male	Anticoagulants	18 (75.0%)	6(25.0%)	24	
Male	Thrombolytic	0 (0.0%)	2 (100%)	2	0.10
Female	Anticoagulants	32(91.4%)	2 (14.7%)	34	
Female	Thrombolytic	4 (100%)	0 (0%)	4	0.56
Gender comparison (thrombolytic survival)	—	—	—	—	0.09

Table 5: Outcomes by Medication and Gender in Patients with Pulmonary Embolism

Time (days)	Number at risk	Number of events	Survival probability (95% CI)
0	59	0	1.00 (ref)
7	59	2	0.97 (0.90–1.00)
21	57	3	0.92 (0.83–0.97)
30	54	3	0.87 (0.73–0.92)

Table 6: Survival analysis

Discussion

This retrospective analysis of 59 patients with pulmonary embolism (PE) provides a comprehensive assessment of demographic, clinical, laboratory, imaging, electrocardiographic, and therapeutic variables associated with in-hospital mortality. The observed mortality rate of 13.6% is consistent with previously reported data from cohorts of high-risk PE patients.[33] Multivariate analysis identified several independent predictors of mortality, including advanced age, hypertension, tachycardia, hypotension, hypoxemia, hemodynamic instability, and right ventricular (RV) dysfunction detected via imaging modalities.[33-35] Advanced age and hypertension likely contribute to reduced cardiac and systemic physiological reserves, whereas RV dysfunction reflects direct cardiac compromise secondary to PE. Collectively, these factors exert both independent and synergistic effects, substantially increasing the risk of in-hospital mortality among patients with acute PE. [36]

Patients who died were significantly older than survivors, with mean ages of 62 and 49 years, respectively. Age remained an independent predictor of mortality in multivariate models, corroborating prior studies that demonstrate increased vulnerability to adverse outcomes among elderly PE patients. [37-38] Hypertension was also more prevalent among non-survivors and independently associated with mortality, emphasizing the role of pre-existing cardiovascular comorbidities in PE prognosis.

Tachycardia, hypotension, and hypoxemia at presentation are well established clinical markers associated with increased mortality risk in acute PE. Among these, hemodynamic instability defined by hypotension or shock emerges as the strongest predictor, conferring up to a five-fold increased risk of death. This underscores the critical importance of prompt recognition and aggressive management of hemodynamically unstable PE patients to improve outcomes. Hemodynamic instability reflects severe RV dysfunction and systemic circulatory compromise due to pulmonary arterial obstruction, resulting in inadequate tissue perfusion and multi-

organ failure. Numerous studies have consistently shown that patients presenting with shock or sustained hypotension exhibit the highest short-term mortality rates, warranting urgent interventions such as thrombolysis or surgical embolectomy.[14, 39, 40]

Moreover, tachycardia and hypoxemia represent compensatory physiological responses to hypoxia and circulatory stress, while also indicating a greater embolic burden and cardiopulmonary compromise. These clinical parameters are incorporated into validated risk stratification tools, such as the Pulmonary Embolism Severity Index (PESI) and the Pulmonary Embolism Mortality Score (PEMS), which predict 30-day mortality and guide therapeutic decision-making.[41]

In acute PE, elevated laboratory biomarkers—including D-dimer, troponin I, and NT-proBNP—are significantly associated with increased mortality, reflecting clot burden and myocardial strain. Elevated D-dimer levels indicate active thrombosis and fibrinolysis, while positive troponin I and raised NT-proBNP concentrations reflect myocardial injury and RV strain secondary to elevated pulmonary artery pressures. [7,25,41,42] These biomarkers are valuable for initial risk stratification and for identifying patients at higher risk of adverse outcomes.

However, multivariate analyses reveal that clinical and imaging markers particularly RV dysfunction and hemodynamic instability are more robust predictors of mortality. This suggests that, although laboratory biomarkers provide important prognostic information, their interpretation must be contextualized within the broader clinical picture, including patient symptoms, vital signs, and imaging findings. For example, RV dysfunction identified through echocardiography or computed tomography (CT) imaging directly reflects cardiac compromise and correlates strongly with mortality risk, often outperforming biomarkers alone.[25, 41] Hemodynamic instability, manifested as hypotension or shock, further delineates patients at highest risk who require urgent intervention.

Therefore, the integrated application of laboratory biomarkers in conjunction with clinical evaluation and imaging modalities enhances the accuracy of mortality prediction in acute PE. While laboratory markers serve as useful adjuncts, they are insufficient when used in isolation to guide prognosis or management decisions without consideration of the overall clinical context. [25, 41, 43]

Findings from this retrospective analysis are consistent with existing literature indicating no significant difference in overall or PE-related mortality between men and women, despite variations in clinical outcomes based on treatment. A large meta-analysis involving over 1.3 million patients found no sex-based difference in all-cause mortality or thrombolytic use (RR: 0.96, $p = 0.66$). [44] Other studies similarly report comparable survival rates and management strategies across sexes after adjusting for confounders.[44-46]

However, trends suggest that women may experience better survival following thrombolysis, albeit with increased risks of major bleeding and longer hospital stays. [44]. These differences may be attributed to distinct clinical presentations, including higher rates of RV strain and comorbidities among women, potentially affecting treatment response and complication rates. [45, 47] In this analysis, all thrombolysed females survived, whereas thrombolysed males exhibited poorer outcomes, reflecting possible sex-related physiological and therapeutic differences, though the small sample limits statistical inference.

These findings highlight the need for individualized, sex-informed risk stratification in PE management. While mortality does not significantly differ by gender, sex-specific factors such as bleeding risk and comorbidities should guide treatment decisions. Further research is warranted to clarify underlying mechanisms and refine gender-sensitive therapeutic approaches.[44]

Limitations

This retrospective study was subject to several limitations. Selection bias may exist due to the inclusion of only hospitalized, confirmed PE cases, potentially omitting milder or undiagnosed cases. Unmeasured confounders, such as medication adherence and socioeconomic status, may have influenced outcomes. Variability in clinical practices, the single-center setting, and a limited sample size further restrict the generalizability of the findings. Prospective, multicenter studies are warranted to validate these results.

Conclusion

In patients with acute pulmonary embolism (PE), independent predictors of in-hospital mortality include advanced age, pre-existing hypertension, tachycardia, hypotension, hypoxemia, hemodynamic instability, and right ventricular (RV) dysfunction. Laboratory biomarkers indicative of myocardial strain, alongside imaging evidence of RV compromise, play a pivotal role in accurate risk stratification. Timely identification of high-risk patients, coupled with prompt and targeted therapeutic intervention, is essential to optimizing clinical outcomes and reducing mortality.

Future Directions

To enhance the generalizability and clinical applicability of current findings, large-scale, multicenter studies are warranted to validate the identified predictors of mortality in acute pulmonary embolism. Additionally, further research should aim to identify and evaluate novel clinical, laboratory, and imaging-based risk factors to improve prognostic accuracy and inform evidence-based management strategies.

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