ORIGINAL RESEARCH

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The Association of the Wells Score with CT-derived RV/LV Ratio and Mortality

Wells Skorunun BT'den Elde Edilen RV/LV Oranı ve Mortalite ile İlişkisi

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Abstract

Objective: Pulmonary thromboembolism (PTE) is the third most common cause of cardiovascular mortality worldwide. The aim of this study was to investigate the relationship between the clinical Wells score and the right ventricle/left ventricle (RV/LV) ratio measured by computed tomography pulmonary angiography (CTPA), and to evaluate the predictive value of these parameters for 30-day mortality in patients diagnosed with PTE.

Method: In this retrospective cross-sectional study, 150 patients diagnosed with acute PTE in the emergency department were included. Wells scores were calculated for all patients, and RV and LV diameters were measured on CTPA images to determine the RV/LV ratio. Parameters associated with mortality were evaluated using univariable and multivariable logistic regression analyses.

Results: Compared with survivors, non-survivors had significantly higher Wells scores (7.06 vs. 4.88; p=0.011), larger RV diameters (45.8 mm vs. 42.4 mm; p=0.034), and higher troponin-I levels (0.52 ng/mL vs. 0.21 ng/mL; p=0.024). Mortality was 23.4% in patients with a Wells score ≥7, whereas no deaths occurred in the low-risk group. Receiver operating characteristic analysis demonstrated an area under the curve of 0.69 for the Wells score and 0.67 for troponin-I in predicting mortality. No significant correlation was found between the Wells score and the CTPA-derived RV/LV ratio (p=0.90). In multivariable analysis, advanced age [odds ratio (OR) 1.13], high Wells score (OR 1.49), and elevated troponin-I levels (OR 2.01) were identified as independent predictors of mortality.

Öz

Amaç: Pulmoner tromboemboli (PTE), dünya genelinde kardiyovasküler mortalitenin üçüncü en sık nedenidir. Bu çalışmanın amacı, PTE tanısı alan hastalarda klinik Wells skoru ile bilgisayarlı tomografi pulmoner anjiyografide (BTPA) ölçülen sağ ventrikül/sol ventrikül (RV/LV) oranı arasındaki ilişkiyi ve bu parametrelerin 30 günlük mortalite üzerindeki öngörücü değerini araştırmaktır.

Yöntem: Retrospektif kesitsel tasarımlı bu çalışmaya, acil serviste akut PTE tanısı alan 150 hasta dahil edilmiştir. Hastaların Wells skorları hesaplanmış, BT görüntülerinden RV ve LV çapları ölçülerek oranlanmıştır. Verilerin analizinde, sürekli değişkenlerin Wells skor grupları arasındaki karşılaştırmaları için tek yönlü varyans analizi kullanılmış; mortalite ile ilişkili parametreler ise univaryant ve multivaryant lojistik regresyon analizleri ile değerlendirilmiştir.

Bulgular: Otuz günlük takipte mortalite oranı %10,6 (n=16) olarak saptanmıştır. Ölen hastalarda Wells skoru (7,06'ya karşı 4,88; p=0,011), sağ ventrikül çapı (45,8 mm'ye karşı 42,4 mm; p=0,034) ve troponin-I düzeyleri (0,52 ng/mL'ye karşı 0,21 ng/mL; p=0,024) sağ kalanlara göre anlamlı derecede yüksektir. Wells skoru ≥7 olan grupta mortalite %23,4 iken, düşük riskli grupta ölüm izlenmemiştir. Alıcı çalışma karakteristiği analizinde Wells skorunun mortaliteyi öngörmede eğri altında kalan alanı 0,69, troponin-I'in ise 0,67 olarak saptanmıştır. Ayrıca malignite varlığı ölen grupta belirgin şekilde fazladır (%25'e karşı %8,3; p=0,048). Wells skoru ile BT pulmoner anjiyografi kaynaklı RV/LV oranı arasında anlamlı bir korelasyon bulunmamıştır (p=0,90). Multivaryant analizde yüksek Wells skoru [olasılık oranı (OR) 1,49],

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Abstract

Conclusion: The Wells score and troponin-I levels are critical indicators for identifying increased short-term mortality risk in patients with PTE. Although the Wells score does not show a correlation with the CTPA-derived RV/LV ratio, it is significantly associated with mortality. Troponin-I independently predicts mortality. Our findings suggest that an integrated, multilayered risk stratification approach combining clinical scoring systems and biomarkers may facilitate early identification and optimized management of high-risk PTE patients.

Keywords: Mortality, pulmonary embolism, RV/LV ratio, Wells score

Öz

ileri yaş (OR 1,13) ve troponin-I yüksekliği (OR 2,01) mortaliteyi öngören bağımsız risk faktörleri olarak tanımlanmıştır.

Sonuç: Wells skoru ve troponin-I düzeyi, PTE hastalarında kısa dönem mortalite riskinin artışının belirlenmesinde kritik göstergelerdir. Klinik Wells skoru, BTPA tabanlı RV/LV oranı ile doğrudan korelasyon göstermese de mortalite ile anlamlı ilişkilidir. Troponin-I myokardiyal hasarı yansıtmakla kalmayıp çalışmamızda mortaliteyi de anlamlı derecede öngörmüştür. Çalışmamız klinik skorlama ve biyobelirteçlerin birleştirildiği entegre, çok katmanlı bir risk stratifikasyonu yaklaşımın, yüksek riskli hastaların erken tespiti ve yönetimi için yeni bir ufuk açabileceğini göstermektedir.

Anahtar kelimeler: Mortalite, pulmoner emboli, RV/LV oranı, Wells skoru

Introduction

Pulmonary thromboembolism (PTE) is a life-threatening cardiovascular condition that requires urgent diagnosis and treatment, resulting from the acute obstruction of the main pulmonary artery or its branches by thrombus formation (1,2). PTE is the third most common cause of cardiovascular mortality worldwide, following ischemic heart disease and stroke (3,4). In untreated situation of masive PTE, mortality rates can increase to 25-30% (4,5). The clinical severity of PTE largely depends on the development of acute right ventricular (RV) dysfunction (6,7). In high-risk (massive) PTE cases accompanied by shock, mortality rates can reach 50% (8).

The diagnostic approach to PTE involves clinical pretest probability scoring systems, D-dimer testing, and imaging algorithms (9). The most widely used clinical prediction rules are the Wells score and the revised Geneva score; both assign points based on patients' symptoms, clinical signs, and risk factors to stratify PTE probability into low, intermediate, and high categories (10). The Wells score is the only clinical prediction rule specifically validated in hospitalized patients and was first described by Wells et al. (11), due to its practicality, it is widely used in the initial evaluation of patients with suspected PTE (11). In a 2015 meta-analysis including 11 studies, the Wells score was shown to have superior diagnostic accuracy compared with the revised Geneva score for predicting pulmonary embolism in patients with suspected disease (12). Use of clinical probability assessment tools such as the Wells score allows risk-based application of D-dimer testing and imaging strategies, which may reduce unnecessary computed tomography use (13). These models aim to minimize unnecessary use of computed tomography pulmonary angiography (CTPA) in low-risk patients while ensuring that high-risk PTE cases are not missed (14).

Current guidelines classify PTE into three major risk categories: High, intermediate, and low risk (15). High-risk PTE is defined by the presence of hemodynamic instability, such as hypotension, shock, or cardiac arrest, and represents approximately 5% of cases and is associated with a 30-day mortality of 15-30% (8). In intermediate-high-risk PTE, patients are normotensive but exhibit RV dysfunction and/or positive cardiac injury biomarkers, including troponin and B-type natriuretic peptide (BNP). This subgroup represents approximately 20-25% of all PTE cases and is associated with a 30-day mortality rate of around 5-10%. In contrast, patients with low-risk PTE show no evidence of hemodynamic instability or right ventricular dysfunction (RVD); early mortality in this group is approximately 1%, and outpatient management may be safely considered. (4).

RVD is a key determinant of risk stratification in PTE (15). RVD is defined by an RV/left ventricle (LV) diameter ratio >1.0 on CTPA and/or elevated cardiac biomarkers such as troponin and BNP (16). An increased RV/LV ratio is associated with worsening cardiac output and higher mortality. Patients with RV/LV >1.0 have an approximately fourfold increase in 30-day mortality. In initially stable patients with RVD, mortality may rise substantially if hemodynamic deterioration occurs (17,18). Therefore, early recognition of RV overload is essential to guide the timely escalation of treatment in PT.

While the Wells score has been primarily validated and widely used as a diagnostic tool for assessing the probability of PTE, its role as a prognostic indicator, particularly in relation to RVD and short-term mortality, remains less well established. This study, therefore, aims not only to examine

the diagnostic utility of the Wells score but also to evaluate its potential prognostic value in predicting mortality and its correlation with imaging markers of RV involvement in patients with acute PTE.

Materials and Methods

This single-center study was a retrospective cross-sectional analysis. We reviewed the medical records of patients aged ≥18 years who were diagnosed with acute PTE and admitted to the emergency department before 2018. Patients were eligible if pulmonary artery thromboembolism was confirmed by CTPA and complete clinical and laboratory data were available from archival records. Patients were excluded for chronic pulmonary hypertension, pregnancy, trauma recent major surgery requiring prophylactic anticoagulation, concomitant severe cardiac disease such as advanced left heart failure or incomplete data. The study protocol was approved by the Ethics Committee of Bezmialem Vakıf University (approval number: E-540224451-050.04-216335, date: 31.10.2025).

Demographic characteristics (age, sex, and comorbidities), clinical findings at presentation, vital signs (blood pressure, heart rate, and oxygen saturation), and laboratory data were obtained through a review of patient medical records and digital archive systems. Venous blood samples were collected from the antecubital vein using vacuum tubes containing EDTA for complete blood count, serum-separator tubes for biochemical and cardiac biomarker analyses, and 3.2% citrate tubes for D-dimer measurement. Biochemical parameters were analyzed using the Siemens Diagnostics ADVIA 1800 system. Cardiac troponin I was measured with the Siemens Diagnostics ADVIA Centaur XP immunoassay analyzer, and D-dimer levels were determined using an enzyme-linked immunosorbent assay (VIDAS ELISA).

For each patient, the Wells clinical prediction score was calculated using data obtained at presentation. The score components included clinical signs of deep vein thrombosis (3 points); tachycardia, defined as a heart rate >100 beats/min (1.5 points); immobilization or recent major surgery (1.5 points); history of PTE or deep vein thrombosis (1.5 points); hemoptysis (1 point); active malignancy (1 point); and absence of an alternative diagnosis more likely than PTE (3 points). Based on the total score, patients were classified into three probability categories: Low (0-1 points), intermediate (2-6 points), and high (≥7 points).

CTPA images obtained at the time of diagnosis were retrospectively reviewed by two independent radiologists who were blinded to the clinical data. RV and LV diameters were measured on axial images representing the four-chamber view, from the inner wall to the inner wall. Measurements were preferentially performed during the diastolic phase of the cardiac cycle, when ventricular dimensions are maximal, and the largest diameters were recorded in millimeters. The RV/LV diameter ratio was calculated for each patient. In accordance with previously established thresholds, the presence of RV dilatation on CTPA was assessed using an RV/LV ratio ≥1.0. (18-20).

CTPA reports were reviewed to determine localization of thrombus within the pulmonary arterial tree and to document ancillary findings, including cardiac chamber enlargement, interventricular septal deviation, and inferior vena cava distension. When available, echocardiographic findings at initial presentation and cardiac biomarker levels (troponin I and BNP) were retrieved from archival records and recorded as markers of RVD.

The echocardiographic data were evaluated retrospectively. Records of transthoracic echocardiography (TTE) examinations, performed as part of routine clinical care within the first 24 hours after hospital admission, were analyzed. Examinations were performed using a cardiac ultrasound system (Epiq 7 Philips Ultrasound System, Amsterdam, the Netherlands) in accordance with the recommendations of the American Society of Echocardiography (ASE). RV dimension was assessed by measuring the basal RV diameter from the apical four-chamber view. RV systolic function was evaluated using tricuspid annular plane systolic excursion (TAPSE). Left ventricular ejection fraction (LVEF) was calculated using the biplane Simpson method from apical two- and four-chamber views. For analyses involving biomarkers and echocardiographic parameters, only patients with available data for the respective variables were included. No imputation was performed due to the retrospective nature of the study.

Statistical Analysis

Statistical analyses were performed using SPSS software (version 22.0). Continuous variables were presented as mean ± standard deviation or median (interquartile range) depending on data distribution, and categorical variables were expressed as counts and percentages. Normality of continuous data was assessed using the Shapiro-Wilk test.

Comparisons between two groups were performed using the independent samples t-test or Mann-Whitney U test, as appropriate. For comparisons among more than two groups, One-Way ANOVA or Kruskal-Wallis tests were used. Categorical variables were compared using the chi-square test or Fisher's exact test, as appropriate. Correlations between continuous variables were evaluated using Spearman's rank correlation coefficient. Receiver operating characteristic (ROC) curve analyses were performed to determine the predictive ability of the Wells score and troponin-I for 30-day mortality, and the area under the curve (AUC), sensitivity, and specificity were calculated. Univariable logistic regression analyses were conducted to identify variables associated with mortality; those with p<0.05 were subsequently included in a multivariable logistic regression model to determine independent predictors. A two-sided p-value <0.05 was considered statistically significant.

Results

A total of 150 patients were included in the study. During the 30-day follow-up period, 16 patients (10.6%) died. The clinical, demographic, laboratory, and radiological characteristics of the patients are summarized in Table 1. The mean age of the study population was 63.5±16.1 years.

Compared with survivors, patients in the mortality group were significantly older (77 vs. 61 years, p<0.001) and had a higher proportion of female patients (54%). In addition, the mortality group had a significantly larger RV end-diastolic diameter (45.8 mm vs. 42.4 mm, p=0.034). Troponin-I levels were also significantly higher in non-survivors than in survivors (0.52 ng/mL vs. 0.21 ng/mL, p=0.024). Similarly, the RV/LV ratio was higher in the mortality group. Moreover, the Wells score was significantly higher in patients who died (7.06 vs. 4.88, p=0.011), and the prevalence of malignancy was greater among patients who died (25% vs.

Table 1. Distribution of demographic characteristics, vital signs, laboratory findings, imaging parameters, and comorbidity prevalence in the overall study population, survivors, and non-survivors

Parameters	All patients (n=150)	Survive (n=134)	Non-survive (n=16)	p-value
Age	63.5	61.85	77.31	**<0.001**
Female	68	61	7	0.92
Male	82	73	9	0.92
Height (cm)	166.77	166.89	166.06	0.826
Weight (kg)	78.4	79.11	74.0	0.306
BMI	26.88	27.36	24.73	0.072
Pulse	92.9	91.3	104.38	0.054
Temperature	36.69	36.69	36.68	1
Respiratory rate	22.12	21.97	23.12	0.846
Troponin-I	0.25	0.21	0.52	0.024
LV (mm) measured by CT	47.38	47.31	47.98	0.695
RV (mm) measured by CT	42.78	42.41	45.84	0.034
RV/LV measured by CT	0.92	0.91	0.99	0.023
Wells score	5.15	4.88	7.06	**0.011**
DM %	27.6	25.4	43.8	0.142
HT %	45.6	47.5	31.2	0.289
CAD %	11.9	11.9	12.5	1
CKD %	6.0	5.1	12.5	0.244
CVD %	8.2	6.8	18.8	0.127
CHF %	12.6	12.6	12.5	1
Malignancy %	10.3	8.3	25.0	**0.048**
RVd (mm) measured by ECHO	36.8	35.9	42.1	**<0.001**
TAPSE (mm)	18.2	18.9	14.6	**<0.001**
LVEF (%)	57.4	57.8	55.9	0.21

Data are presented as mean ± standard deviation or n (percentage). Statistically significant p-values are shown in bold. BMI: Body mass index, LV: Left ventricle, RV: Right ventricle, RV/LV: Right ventricle/left ventricle diameter ratio, DM: Diabetes mellitus, HT: Hypertension, CAD: Coronary artery disease, CKD: Chronic kidney disease, CVA: Cerebrovascular accident, CHF: Congestive heart failure, LVEF: Left ventricular ejection fraction, TAPSE: Tricuspid annular plane systolic excursion

8.3%, p=0.048). On echocardiographic assessment, non-survivors had a significantly larger RV basal diameter (42.1 mm vs. 35.9 mm, p<0.001) and lower TAPSE values (14.6 mm vs. 18.9 mm, p<0.001), while no significant difference was observed in LVEF (55.9% vs. 57.8%, p=0.21).

In the overall cohort, 27.6% of patients had diabetes mellitus, 45.6% had hypertension, 27.7% were current smokers, 32.3% had a history of immobilization, and 36% had deep vein thrombosis.

When patients were stratified into low-, intermediate-, and high-risk groups according to the Wells score, the demographic, clinical, laboratory, and radiological parameters were summarized in Table 2. No significant differences were observed among the groups in age, height, body mass index, systolic and diastolic blood pressure, troponin-I levels, or RV/LV ratio measured on CTPA (all p>0.05). The mean Wells score for the entire cohort was 5.1, whereas it was 7.1 in patients who died and 4.9 in survivors.

Mortality increased significantly with increasing Wells score. No deaths occurred in the low-risk group, whereas mortality was 7.2% in the intermediate-risk (3-6) group and 23.4% in the high-risk (≥7) group. This difference was statistically significant (p=0.011) (Figure 1).

Using a Wells score cut-off value of ≥7, the sensitivity and specificity for predicting 30-day mortality in patients with PTE were 68.8% and 68.4%, respectively. ROC curve analysis demonstrated an AUC of 0.69 [95% confidence interval (CI): 0.56-0.81; p=0.006] (Figure 2a). The cut-off value for troponin-I was set at 0.014 ng/mL, and 34.7% of the patients had troponin levels >0.014 ng/mL. The mean troponin level was 0.52 ng/mL in patients who died and 0.26 ng/mL in survivors (p=0.024). In addition, the mortality rate was 31.0% among patients with positive troponin levels, compared with 14.9% in those with normal troponin levels. Troponin positivity significantly predicted mortality (p=0.024). A ROC analysis was performed to evaluate the association between elevated troponin levels and mortality. Using a troponin-I cut-off value of 0.014 ng/mL, the analysis showed that troponin-I demonstrated moderate discriminative ability for predicting mortality (AUC =0.67) (95% CI: 0.33-0.80; p=0.023). The sensitivity was 87.5% and the specificity was 43.3% (Figure 2b).

Table 2. Distribution of demographic characteristics, vital signs, comorbidities, and mortality among patients with low, intermediate, and high Wells scores

Parameters	Low (≤2) (n=14)	Intermediate (3-6) (n=69)	High (≥7) (n=47)	p-value
Age	63.93±15.22	64.80±15.45	63.77±17.08	0.9392
Height	165.50±6.91	165.79±7.65	168.85±9.19	0.1561
Weight	79.00±13.54	77.13±13.23	79.95±18.08	0.6479
BMI	27.99±2.33	26.84±4.90	26.64±4.27	0.7712
BP systolic	120.71±31.68	117.35±19.15	112.00±19.49	0.2724
BP diastolic	77.79±25.98	72.24±11.83	69.16±12.65	0.1343
Pulce	99.14±24.87	88.84±16.21	95.51±22.23	0.0837
Temperature	36.60±0.32	36.70±0.54	36.68±0.44	0.7747
Respiratory rate	20.86±2.18	22.06±4.24	22.51±4.61	0.4405
Troponin-I	0.13±0.22	0.38±1.03	0.14±0.18	0.2378
RV/LV	0.89±0.16	0.93±0.22	0.92±0.20	0.7620
DM %	35.7	21.7	36.2	0.1955
HT %	57.1	40.6	51.1	0.3681
CAD %	28.6	13.0	6.4	0.0823
CKD %	14.	5.8	4.3	0.3845
CVD %	0.	5.8	14.9	0.1088
CHF %	85.7	4.3	2.1	<0.0001
Malignancy %	0.	8.7	10.6	0.4524
COPD %	35.7	8.7	8.5	0.0112
Mortality %	0.0 (0/14)	7.2 (5/69)	23. (11/47)	0.0111

BMI: Body mass index, BP: Blood pressure, RV/LV: Right ventricle /left ventricle diameter ratio, DM: Diabetes mellitus, HT: Hypertension, CAD: Coronary artery disease, CKD, Chronic kidney disease, CVA: Cerebrovascular accident, CHF: Congestive heart failure, COPD: Chronic obstructive pulmonary disease.

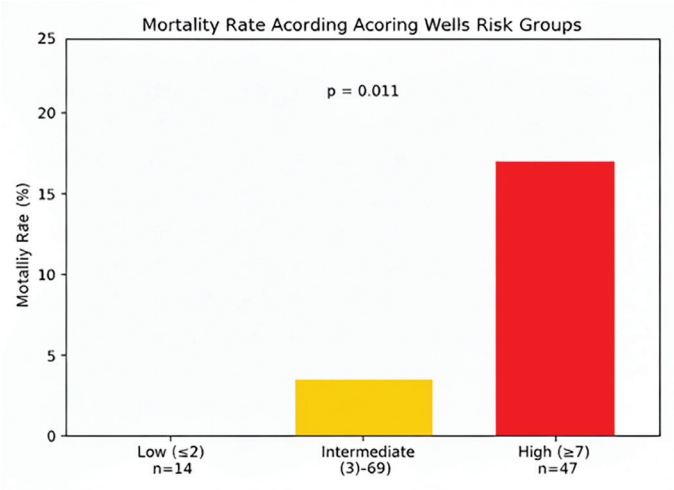

Figure 1. Mortality rate according to Wells risk groups

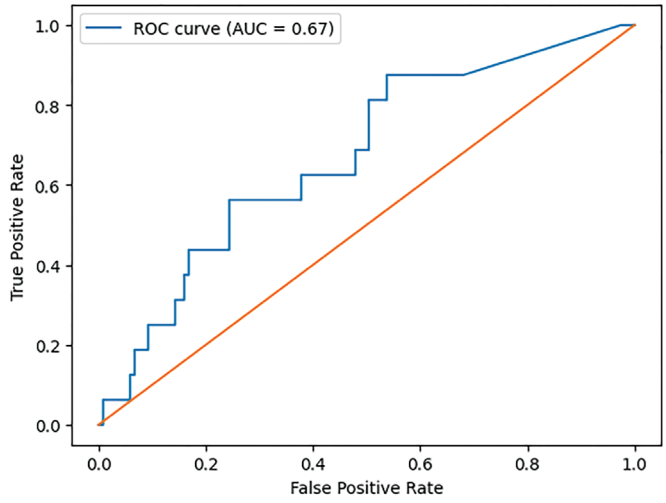

Figure 2b. ROC analysis demonstrating the association between troponin-I and mortality

ROC: Receiver operating characteristic, AUC: Area under the curve

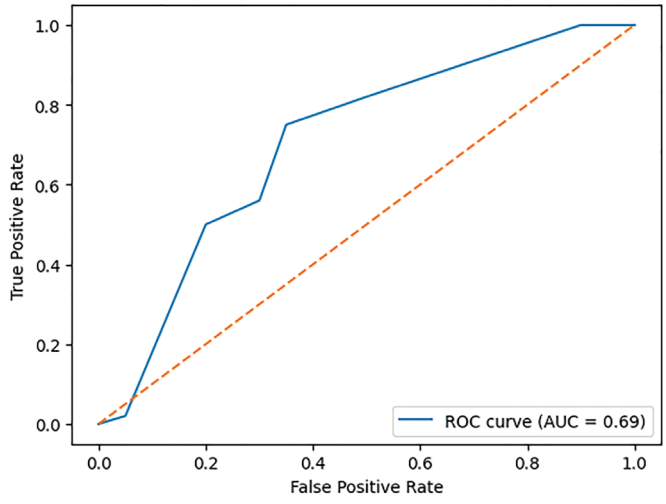

Figure 2a. ROC curve of the Wells score for mortality

ROC: Receiver operating characteristic, AUC: Area under the curve

The RV/LV ratio assessed by CTPA was greater than 0.9 in 48% of the patients. No assessed was found between the Wells score and the CTPA-derived RV/LV ratio (Spearman's ρ=-0.01; p=0.90). No significant difference in Wells scores between patients with and without RV dilatation was observed (p=0.33). An RV/LV ratio >1 was detected in 24% of the patients. The mortality rate in this group was 28.6%, whereas it was 16.4% in patients with an RV/LV ratio ≤1.

In univariable logistic regression analysis, age, Wells score, and troponin-I level were significantly associated with mortality, whereas the RV/LV ratio was not. Variables that were significant in the univariable analysis were subsequently included in the multivariable logistic regression model. In the multivariable analysis, age [odds ratio (OR) 1.13; 95% CI, 1.05-1.21; p<0.001], Wells score (OR 1.49; 95% CI, 1.16-1.92; p=0.002), and troponin-I level

Table 3. Univariable and multivariable logistic regression analyses

Variable	Univariable OR (95% CI)	p-value	Multivariable OR (95% CI)	p-value
Years	1.12 (1.06-1.18)	<0.001	1.13 (1.05-1.21)	<0.001
Wells score	1.42 (1.18-1.71)	<0.001	1.49 (1.16-1.92)	0.002
Troponin-I	1.85 (1.10-3.12)	0.021	2.01 (1.03-3.94)	0.041
Malignancy	2.84 (1.01-7.96)	0.048	30.9 (2.80-341.3)	0.005
cystolic BP ≤90 mmHg	3.92 (1.18-13.01)	0.026	2.51 (0.32-19.84)	0.384

Variables associated with mortality were initially evaluated using univariable logistic regression analysis and subsequently using multivariable logistic regression analysis. Results are presented as odds ratios (ORs), 95% confidence intervals (CIs), and p-values. BP: Blood pressure

(OR 2.01; 95% CI, 1.03-3.94; p=0.041) were independently associated with mortality (Table 3).

Discussion

In the present study, the Wells score, elevated troponin levels, and advanced age were significantly associated with mortality in patients with acute PTE. Notably, although higher troponin-I levels were associated with increased mortality and higher RV/LV ratios, there was no significant correlation between the Wells score and RV/LV ratios.

The RV/LV ratio has been widely investigated as an imaging marker of RVD and adverse outcomes in PTE. Previous studies have demonstrated that an increased RV/LV ratio is associated with reduced cardiac index, particularly in intermediate-risk patients, and may predict hemodynamic deterioration (19). An emergency-department-based cohort reported that the RV/LV ratio had high sensitivity for predicting 30-day mortality (20). D espite these findings, the clinical utility of the RV/LV ratio remains limited by substantial heterogeneity in measurement techniques and cut-off values. A comprehensive review, encompassing data from 35 studies, highlighted considerable variability in RV/LV ratio assessment methods and supported the use of an RV/LV ratio >1.0 measured on a single axial slice as a practical and reproducible approach. In our cohort, the absence of a correlation between the Wells score and the RV/LV ratio may be explained by this lack of standardization, as well as by the sensitivity of RV/LV ratio measurements to slice orientation, cardiac cycle phase, and operator-dependent variability (21). These factors may attenuate statistical associations, particularly in single-center studies with heterogeneous risk profiles.

In contrast, troponin-I emerged as a robust prognostic marker in our study. Elevated troponin levels were independently associated with early mortality among patients with normotensive pulmonary embolism. Mean troponin levels were substantially higher in non-survivors than in survivors. These findings are consistent with contemporary literature confirming the independent association between troponin elevation and short-term mortality in pulmonary embolism.

Troponin elevation reflects RV pressure overload, ischemia, and myocardial injury, and therefore serves as a direct marker of the severity of RVD in pulmonary embolism. In our study, elevated troponin levels were associated with a substantially higher mortality risk compared with normal levels (22). This finding supports the concept that cardiac biomarkers provide incremental prognostic information beyond clinical risk scores alone and, in certain clinical settings, may offer greater prognostic utility than imaging-based parameters such as the RV/LV ratio. The concomitant presence of elevated troponin levels and an increased RV/LV ratio appears to define a higher-risk clinical profile, potentially necessitating closer clinical monitoring and more intensive management strategies. In contrast, the combination of normal troponin levels and an RV/LV ratio ≤1 may identify a relatively low-risk subgroup of patients, in whom outpatient management could be considered in carefully selected cases (23).

Accumulating evidence supports the use of multimodal risk stratification strategies that integrate clinical risk scores, imaging findings, and cardiac biomarkers. In particular, the pulmonary embolism severity index (PESI) and its simplified version, which incorporate variables such as age, comorbidities, and hemodynamic parameters, have been extensively validated as independent predictors of short- and long-term mortality in patients with pulmonary embolism. Several recent studies have shown that combining clinical scores (such as Wells, Geneva, and PESI) with imaging markers like the RV/LV ratio and biomarkers provides superior prognostic discrimination compared with reliance on any single parameter alone (24). Consistent with this framework, our findings suggest that patients with both low clinical risk and limited imaging evidence of RVinvolvement may represent a subgroup suitable for less-intensive management, with potential implications for safe outpatient care and more efficient use of healthcare resources.

In the context of current clinical practice, our findings can be interpreted alongside the ESC/ERS risk stratification framework for acute PTE. According to these guidelines, patients are classified into low, intermediate-low, intermediate-high, and high-risk categories based on hemodynamic status RVD, and cardiac biomarker elevation. Our results—demonstrating that elevated troponin levels and increased RV/LV ratios are associated with higher mortality, while low Wells scores and normal imaging parameters may indicate lower risk— align with this stratification system. These observations support the clinical relevance of combining easily obtainable clinical scores, imaging findings, and biomarkers to refine risk assessment and guide management decisions, including the selection of patients suitable for outpatient care or closer monitoring.

To our knowledge, this study is among the first to directly examine the relationship between the Wells score, RV/LV ratio, and mortality in patients with PTE. While previous studies have largely addressed the diagnostic performance of the Wells score or the prognostic value of imaging markers in isolation, our findings suggest that the Wells score may also have prognostic relevance (25). These results highlight that a simple and widely accessible clinical score can provide meaningful prognostic information, potentially complementing imaging- and biomarker-based risk stratification without requiring advanced diagnostic modalities.

Advanced age was strongly associated with mortality in our cohort, consistent with current European Society of Cardiology/European Respiratory Society guidelines, which recognize age as an independent predictor of adverse outcomes in PTE. Taken together, our findings emphasize the value of an integrated risk stratification approach that combines clinical assessment, cardiac biomarkers, and imaging parameters. In this context, the coexistence of troponin elevation and an increased RV/LV ratio appears to identify a higher-risk patient profile that may warrant closer clinical surveillance (26).

Study Limitations

This study has several limitations. Its retrospective and single-center design may limit the generalizability of the findings. The relatively small sample size may have reduced the statistical power of the analyses. Potential heterogeneity in RV/LV ratio measurement protocols on CTPA and the lack of standardized measurement techniques may have introduced variability and bias. A further limitation of this study is that cardiac troponin levels were measured at a single time point; the absence of serial measurements may have limited our ability to evaluate dynamic changes and temporal trends in troponin levels. Another limitation of this study is that interobserver variability in RV and LV measurements was not formally assessed, which may have affected measurement reliability. Therefore, the results should be interpreted with caution and validated in prospective studies with larger sample sizes and standardized imaging protocols.

Conclusion

Although no significant association was observed between the Wells score and the RV/LV ratio, concordance was observed between the Wells score and troponin levels. These findings may suggest that clinical risk scores are more closely aligned with biomarker-defined myocardial injury than with imaging-based measures alone. Overall, our results support the potential value of an integrated, multilayered risk stratification approach combining clinical assessment, biomarkers, and imaging for short-term prognostic evaluation in patients with pulmonary embolism. Larger prospective studies are warranted to further clarify and confirm these observations.

Ethics

Ethics Committee Approval: The study protocol was approved by the Ethics Committee of Bezmialem Vakıf University (approval number: E-540224451-050.04-216335, date: 31.10.2025).

Informed Consent: Retrospective study.

Footnotes

Authorship Contributions

Surgical and Medical Practices: M.A., M.H.D., İ.Ş., S.S.Y., Concept: M.A., M.G.A., M.H.D., İ.Ş., S.S.Y., Design: M.A., M.G.A., M.H.D., S.S.Y., Data Collection or Processing: M.A., M.G.A., M.H.D., İ.Ş., S.S.Y., Analysis or Interpretation: M.A., M.G.A., M.H.D., İ.Ş., S.S.Y., Literature Search: M.A., M.G.A., İ.Ş., S.S.Y., Writing: M.A., M.G.A., İ.Ş., S.S.Y.

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