ORIGINAL RESEARCH

Bagcilar Med Bull 2026;11(2):270-278
DOI: 10.4274/BMB.galenos.2026.88700

Uric Acid-to-albumin Ratio as an Independent Predictor of Short-term Mortality in Acute Pulmonary Embolism

Akut Pulmoner Embolide Kısa Dönem Mortalitenin Bağımsız Bir Belirteci Olarak Ürik Asit/Albümin Oranı

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Abstract

Objective: We aimed to evaluate whether the uric acid-to-albumin ratio (UAR) predicts 30-day mortality in patients with acute pulmonary embolism (PE).

Method: This retrospective study included adult patients diagnosed with acute PE between June 2011 and August 2024. After applying exclusion criteria, 261 patients were analyzed. PE diagnosis was confirmed by computed tomographic pulmonary angiography. The primary endpoint was 30-day all-cause mortality. ROC curve analysis was performed to determine the optimal UAR cut-off value. Independent predictors of mortality were identified using multivariable logistic regression analysis.

Results: The mean age of the study population was 63.6±15.9 years, and 60.2% were female. During the 30-day follow-up, 49 patients (18.8%) died. Non-survivors were significantly older. Furthermore, these patients exhibited significantly higher values for the UAR, systolic pulmonary artery pressure, and PE severity index scores. Also, a statistically significant increase in the frequency of a right ventricle/left ventricle ratio exceeding 1 and a history of malignancy, as well as significantly lower values albumin and the estimated glomerular filtration rate, were detected in non-survivors. ROC analysis specified a UAR cut-off value of 1.78 for predicting 30-day mortality [area under the curve: 0.788; 95% confidence interval (CI): 0.716-0.860], with 71.4% sensitivity and 67.9% specificity. In multivariable logistic

Öz

Amaç: Bu çalışmada, akut pulmoner emboli (PE) hastalarında ürik asit/albümin oranının (ÜAO) 30 günlük mortaliteyi öngörmedeki değerini araştırmayı amaçladık.

Yöntem: Haziran 2011-Ağustos 2024 tarihleri arasında akut PE tanısı alan 261 hasta retrospektif olarak değerlendirildi. PE tanısı bilgisayarlı tomografi pulmoner anjiyografi ile doğrulandı. Birincil sonlanım noktası 30 günlük tüm nedenlere bağlı mortalite olarak belirlendi. ÜAO için optimal kesim değeri ROC eğrisi analizi ile belirlendi. Mortalite için bağımsız prediktörler çok değişkenli lojistik regresyon analizi ile değerlendirildi.

Bulgular: Çalışma popülasyonunun yaş ortalaması 63,6±15,9 yıl olup, hastaların %60,2'si kadındı. Otuz günlük mortalite 49 hastada (%18,8) saptandı. Mortalite izlenen hastalar anlamlı olarak daha ileri yaşta olup ÜAO, sistolik pulmoner arter basıncı, PE şiddet indeks skorları, sağ ventrikül/sol ventrikül oranı 1'den büyük olma ve malignite öyküsü sıklığı daha fazla saptandı. Bunun yanı sıra tahmini glomerüler filtrasyon hızı mortalite izlenen grupta belirgin olarak daha düşük bulundu. ROC analizinde ÜAO için 1,78 kesim değeri 30 günlük mortaliteyi %71,4 duyarlılık ve %67,9 özgüllük ile öngördü [eğri altında kalan alan: 0,788; %95 güven aralığı (GA): 0,716-0,860]. Çok değişkenli lojistik regresyon analizinde yüksek ÜAO [olasılık oranı (OO): 4,798; %95 GA: 2,485-9,262; p<0,001] ve malignite öyküsü (OO: 3,343; %95 GA: 1,218-9,170; p=0,019) 30 günlük mortalitenin bağımsız prediktörleri olarak saptandı.

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Received: 18.02.2026 **Accepted:** 06.06.2026 **Epub:** 10.06.2026 **Publication Date:** 24.06.2026

Cite this article as: İnce O, Dönmez E, Deniz MF, Terzioğlu C, Bulut S, Özcan S. Uric acid-to-albumin ratio as an independent predictor of short-term mortality in acute pulmonary embolism. Bagcilar Med Bull. 2026;11(2):270-278

Abstract

regression analysis, high UAR [odds ratio (OR) 4.798; 95% CI 2.485-9.262; p<0.001] and history of malignancy (OR 3.343; 95% CI 1.218-9.170; p=0.019) were independent predictors of 30-day mortality.

Conclusion: In acute PE, UAR was identified as an independent risk factor for 30-day mortality. This inexpensive biomarker, obtained from routine tests, can contribute to early risk stratification and aid in determining treatment strategies when integrated into existing prognostic models. However, large-scale, multicenter prospective studies are needed to confirm these results and demonstrate the additional clinical benefit of UAR in assessing the prognosis of acute PE patients.

Keywords: Acute pulmonary embolism, mortality, prognosis, risk stratification, uric acid-to-albumin ratio

Öz

Sonuç: Akut PE hastalarında ÜAO'ı 30 günlük mortalitenin bağımsız bir risk belirteci olarak saptanmıştır. Rutin laboratuvar testlerinden elde edilen bu düşük maliyetli biyobelirteç, mevcut prognostik modellere entegre edildiğinde erken risk sınıflamasına katkı sağlayabilir ve daha yoğun tedavi stratejilerinden fayda görebilecek hastaların belirlenmesine katkı sağlayabilir. Ancak bu bulguların doğrulanması ve ÜAO'ın ek klinik katkısının ortaya konulması için geniş ölçekli, çok merkezli ve prospektif çalışmalara ihtiyaç vardır.

Anahtar kelimeler: Akut pulmoner emboli, mortalite, prognoz, risk sınıflaması, ürik asit/albümin oranı

Introduction

The acute pulmonary embolism (PE) in-hospital mortality rate ranges between 5% and 10%. Among cardiovascular diseases, it constitutes the third most common cause of mortality, following myocardial infarction and stroke (1,2). Approximately 5% of patients with PE present with hemodynamic instability or cardiopulmonary arrest secondary to acute right ventricular failure, with a mortality rate of about 30% (3). Therefore, risk stratification and prognosis assessment are crucial for determining the most appropriate treatment strategy.

Patients with hemodynamic instability are classified as high-risk. Most patients do not have hemodynamic instability. These patients require further risk assessment by using clinical conditions, comorbidities, and laboratory and imaging indicators of PE severity, such as cardiac troponins, heart-type fatty acid-binding protein (H-FABP), N-terminal pro-brain natriuretic peptide (NT-proBNP) levels, and signs of right ventricular dysfunction detected by echocardiography or computed tomographic pulmonary angiography (CTPA). Hence, different combinations of these variables have been employed to develop 30-day mortality prediction scores. Among clinical scores that combine PE severity with underlying disease, the pulmonary embolism severity index (PESI) currently possesses the most validation evidence (4). These scoring systems provide valuable prognostic information regarding short-term mortality risk but cannot precisely predict mortality for every individual patient. So, additional factors are needed to increase the predictive value of mortality in acute PE patients (5).

Uric acid (UA) promotes hypercoagulability through oxidative stress, inflammation, and endothelial dysfunction. During its production via the xanthine oxidase pathway, reactive oxygen species are formed, which reduces the protective nitric oxide level and causes endothelial damage. Elevated serum UA level is associated with endothelial activation and increased production of inflammatory cytokines, which enhance the activity of thrombotic factors and inhibit fibrinolysis (6). Data from the literature suggests that hyperuricemia may predispose individuals to a higher incidence of thromboembolic events (7,8).

Albumin is an acute phase reactive protein, and it has several essential functions, such as osmotic pressure maintenance, transport, and regulation of pH. Serum albumin has various protective mechanisms in the vascular bed. It has antioxidant properties, improves endothelial function, and inhibits platelet aggregation via neutralizing reactive oxygen and nitrogen species. Furthermore, albumin binds and activates antithrombin, inhibits factor Xa, and decreases the hepatic synthesis of factors V and VII. Meta-analyses have demonstrated a significant association between hypoalbuminemia and venous thromboembolism (VTE) (9,10).

Increased UA and decreased serum albumin are associated with prothrombotic and inflammatory states and could contribute to VTE pathogenesis. Given the established roles of UA and albumin in thrombosis, we explored whether their ratio could predict 30-day all-cause mortality in acute PE.

Materials and Methods

This retrospective study initially assessed 320 adult patients diagnosed with acute PE and admitted to our center between June 2011 and August 2024. We obtained data on demographics, clinical status, and laboratory

results by examining both written medical files and the hospital database. Acute PE diagnosis was confirmed by CTPA. In patients with hemodynamic instability and no absolute contraindications for thrombolysis, systemic fibrinolysis by infusing 100 mg of recombinant tissue-type plasminogen activator over two hours was administered. In hemodynamically unstable patients for whom thrombolytic therapy was an absolute contraindication, percutaneous catheter-directed interventions were performed as reperfusion therapy. Initial anticoagulation for all patients was provided with either low-molecular-weight heparin or a direct oral anticoagulant, specifically apixaban or rivaroxaban. For a proportion of the patients, long-term oral anticoagulation was achieved with a vitamin K antagonist, following a bridging period with low-molecular-weight heparin.

Comorbidities were defined according to established diagnostic criteria: Cerebrovascular accident (11), coronary artery disease (CAD) (12), hypertension (13), diabetes mellitus (14), chronic kidney disease (CKD) (15), chronic obstructive pulmonary disease (16), and heart failure (HF) (17). The estimated glomerular filtration rate (eGFR) was derived from the chronic kidney disease epidemiology collaboration (CKD-EPI) equation. Patients who died from any cause in the first month of follow-up were labeled as non-survivors, and the remaining as survivors.

After excluding patients with active infection, severe CKD (eGFR <30 mL/min/1.73 m²) (15), inflammatory or hematological disorders, a history of gout or use of urate-lowering therapy, significant hepatic dysfunction (irreversible hepatic dysfunction, manifested by ascites, hepatic encephalopathy, variceal bleeding, or jaundice) (18), or incomplete datasets, the final analysis had 261 patients (Figure 1).

Transthoracic echocardiography (Vivid S70; GE Medical System, Horten, Norway) was carried out to assess the cardiac function, valve morphology, systolic pulmonary artery pressure (sPAP), tricuspid annular plane systolic excursion (TAPSE), and cardiac chamber dimensions. Left ventricular ejection fraction (LV-EF) was measured using Simpson's method, and sPAP was estimated from the Doppler-derived peak tricuspid regurgitation gradient, to which an estimated right atrial pressure was added. Right atrial pressure was inferred from inferior vena cava diameter and its respiratory variation during a forced inspiratory maneuver. The right ventricle-to-left ventricle diameter (RV/LV) ratio was determined using CTPA. Right and left ventricular short-axis dimensions were measured in optimal four-chamber views, just below the respective atrioventricular valve.

We obtained venous blood specimens from each patient at baseline, prior to starting treatment. Measurements were conducted using a BS-2000M chemistry analyzer (Mindray, Shenzhen, China). The uric acid-to-albumin ratio (UAR) was calculated as the ratio of baseline serum UA (mg/dL) to baseline serum albumin (g/dL). We calculated original PESI scores using the established criteria summarized in Table 1 (4). Patients were divided into low-risk, moderate/high-risk, and very high-risk groups based on their PESI scores.

Table 1. Original pulmonary embolism severity index

Variables		1-month mortality risk
Age	Years	≤65 points: Very low (0-1.6%)
Male sex	+10 points	66-85 points: Low (1.7-3.5%)
Cancer	+30 points	86-105 points: Moderate (3.2-7.1%)
Chronic heart failure	+10 points	106-125 points: High (4-11.4%)
Chronic pulmonary disease	+10 points	>125 points: Very high (10-24.5%)
Heart rate ≥110 b.p.m.	+20 points	
SBP <100 mmHg	+30 points	
Respiratory rate >30 per min	+20 points	
Temperature <36 °C	+20 points	
Altered level of consciousness	+60 points	
Arterial oxygen saturation <90%	+20 points	

b.p.m.: Beats per minute, SBP: Systolic blood pressure

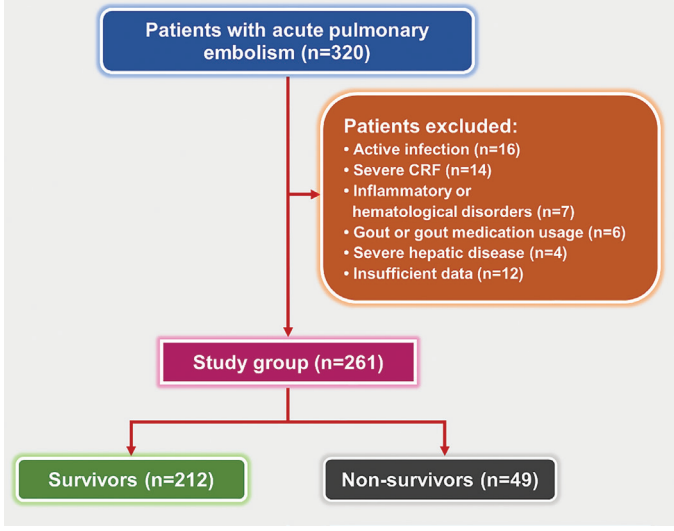

Figure 1. Flow diagram for the study
CRF: Chronic renal failure

The study received approval from the Local Ethics Committee of University of Health Sciences Turkey, İstanbul Bağcılar Training and Research Hospital (decision number: 2025/10/15/099, date: 24.10.2025) and was executed in line with the Declaration of Helsinki. In our study, data from patients between June 2011 and August 2024 were analyzed retrospectively.

Statistical Analysis

Descriptive statistics, group comparisons, and regression analyses were performed using SPSS version 26.0 (IBM Corp., Armonk, NY, USA). Receiver operating characteristic (ROC) curve analysis, including AUC estimation and comparison of correlated ROC curves using the DeLong method, were conducted in R statistical software (R Foundation for Statistical Computing, Vienna, Austria) with the pROC package. Nominal variables are represented as counts and percentages. Normality of continuous data was examined through the Kolmogorov-Smirnov test, and results were reported as the mean ± standard deviation or median with interquartile range. Differences in frequency distributions were tested with the Pearson chi-square test, the chi-square test with Yates' continuity correction, or Fisher's exact test, as indicated. Continuous variables were evaluated with the Mann-Whitney U test or Student's t-test, after assessing their distribution. Logistic regression analysis was employed to evaluate the impact of independent predictors on mortality. The multivariable model was built by incorporating variables that emerged as significant in the preceding univariable analysis. The two-tailed p-value <0.05 was considered statistically significant.

Results

After applying inclusion criteria, 261 patients with acute PE were retained for analysis. The patients' mean age was 63.6±15.9 years, with a female predominance of 60.2%. During the 30-day follow-up period, mortality occurred in 49 patients (18.8%). The average PESI score was 131.2±45.3.

Reperfusion therapy was administered to 77 of the 80 high-risk patients via intravenous rtPA treatment and to the three patients with thrombolytic contraindications via percutaneous catheter-directed intervention.

Survivors and non-survivors exhibited no statistically meaningful differences regarding sex, CAD, diabetes mellitus, cerebrovascular accident, hypertension, chronic obstructive pulmonary disease, HF, deep vein thrombosis, LV-EF, TAPSE, high-sensitivity cardiac troponin I (hs-cTnI), D-dimer, hemoglobin, and platelet count. Non-survivors had significantly higher age (69.5±16.4 vs. 62.3±15.6; p=0.004), PESI score (183.5±53.8 vs. 119.1±33.1; p<0.001), malignancy history (28.6% vs. 8.5%; p<0.001), sPAP (50.6±11.4 vs. 46.3±11.3; p=0.016), RV/LV ratio >1 (77.6% vs. 60.8%; p=0.042), UA (7.8±2.8 vs. 5.8±1.8; p<0.001), and UAR (2.5±1.1 vs. 1.5±0.5; p<0.001). However, eGFR (59.3±23 vs. 80±24.5; p<0.001) and albumin (3.3±0.6 vs. 3.9±0.5; p<0.001) were significantly lower among non-survivors (Table 2).

ROC curve analysis demonstrated that the UAR had a moderate discriminatory ability for predicting 30-day mortality in the overall cohort. The area under the curve (AUC) was 0.788 (95% CI: 0.716-0.860). The optimal cut-off value was 1.78, with a sensitivity of 71.4% and a specificity of 67.9% for predicting 30-day mortality. In the low-risk group (n=10), no 30-day mortality events were observed; therefore, ROC analysis could not be performed for this subgroup. In the moderate/high-risk group (n=171; 25 deaths), UAR demonstrated moderate discriminatory ability for predicting 30-day mortality, with an AUC of 0.753 (95% CI: 0.642-0.863). The optimal cut-off value was 1.7, yielding a sensitivity of 68% and a specificity of 67.1%. In the very high-risk group (n=80; 24 deaths), UAR also showed moderate predictive performance, with an AUC of 0.798 (95% CI: 0.691-0.906). The optimal cut-off value was 1.84, corresponding to a sensitivity of 66.7% and a specificity of 66.1% (Figure 2). Comparison of AUCs between the

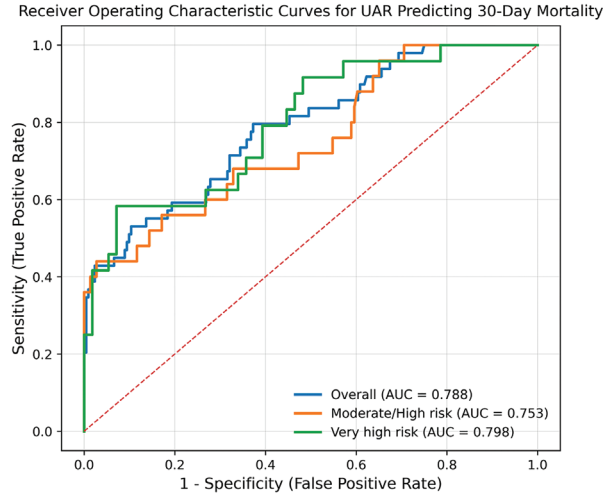

Figure 2. Receiver operating characteristic (ROC) curves of the uric acid-to-albumin ratio (UAR) for predicting 30-day mortality in the overall cohort and risk subgroups. UAR demonstrated moderate discriminative ability in the overall population (AUC =0.788), as well as in the moderate/high-risk (AUC =0.753) and very high-risk groups (AUC =0.798)

AUC: Area under the curve

moderate/high and very high-risk groups using DeLong's method did not show a statistically significant difference (ΔAUC =0.046; p=0.562).

Patients were categorized into low-UAR (<1.78, n=161) and high-UAR (≥1.78, n=100) groups. High-UAR patients exhibited significantly higher age (70.8±13.7 vs. 59.2±15.7; p<0.001), PESI score (147.8±46 vs. 120.8±41.8; p<0.001), hs-cTnI levels [86.6 (38.5-250) vs. 52.5 (18-182); p=0.022], and prevalence of CAD (21% vs. 7.5%; p=0.003), hypertension (59% vs. 40.4%; p=0.005), and HF (11% vs. 1.2%; p=0.001), along with lower LV-EF [55 (55-60) vs. 60 (55-60); p=0.004], TAPSE (14.2±3 vs. 15.3±3.1; p=0.006), and eGFR (61±23.5 vs. 85.5±22.3; p<0.001) (Table 3).

Univariable and multivariable logistic regression models were constructed to identify factors independently associated with 30-day mortality. Age, history of malignancy, right ventricle/left ventricle (RV/LV) ratio >1, sPAP, hs-cTnI, and UAR were each positively associated, whereas eGFR was negatively associated with mortality in the univariable analysis. A high degree of correlation was observed between RV/LV ratio and sPAP; therefore, to prevent collinearity in the regression model, only RV/LV ratio was entered into the multivariable analysis. Afterwards, these factors were evaluated with a multivariable model. The analysis indicated that malignancy history (OR 3.343; 95% CI 1.218-9.170; p=0.019) and high UAR (OR 4.798; 95% CI 2.485-9.262; p<0.001) independently predicted mortality within 30-day (Table 4).

Table 2. Baseline characteristics of patients in terms of 30-day mortality

Variables	Total population (n= 261)	Survivors (n= 212)	Non-survivors (n= 49)	p-value
Comorbidity and clinical properties				
Age, (years)	63.6±15.9	62.3±15.6	69.5±16.4	0.004
Female sex, n (%)	157 (60.2)	127 (59.9)	30 (61.2)	0.994
CAD, n (%)	33 (12.6)	26 (12.3)	7 (14.3)	0.701
DM, n (%)	54 (20.7)	46 (21.7)	8 (16.3)	0.522
Hypertension, n (%)	124 (47.5)	104 (49.1)	20 (40.8)	0.378
CVA, n (%)	14 (5.4)	9 (4.2)	5 (10.2)	0.149
COPD, n (%)	31 (11.9)	26 (12.3)	5 (10.2)	0.875
Heart failure, n (%)	13 (5)	9 (4.2)	4 (8.2)	0.274
Malignancy history, n (%)	32 (12.3)	18 (8.5)	14 (28.6)	**<0.001**
DVT, n (%)	75 (28.7)	60 (28.3)	15 (30.6)	0.747
PESI score	131.2±45.3	119.1±33.1	183.5±53.8	**<0.001**
Echocardiographic parameters				
• LV-EF (%)	58 [55-60]	60 [55-60]	57 [55-60]	0.239
• sPAP, (mmHg)	47.1±11.3	46.3±11.3	50.6±11.4	**0.016**
• TAPSE, (mm)	14.9±3.1	15.1±3.1	14.2±2.9	0.061
• RV/LV ratio >1, n (%)	167 (64)	129 (60.8)	38 (77.6)	**0.042**
Laboratory				
• hs-cTnI, (pg/mL)	64.5 [25-215]	58.7 [24.2-201]	89.9 [28.9-300]	0.146
• D-dimer, (ng/mL)	4.4 [1.8-7.9]	4.1 [1.8-7.8]	5.4 [1.1-8.2]	0.807
• eGFR, (mL/min/1.73 m^2)	76.1±25.7	80±24.5	59.3±23	**<0.001**
• Uric acid, (mg/dL)	6.1±2.2	5.8±1.8	7.8±2.8	**<0.001**
• Albumin, (g/dL)	3.7±0.6	3.9±0.5	3.3±0.6	**<0.001**
• Hemoglobin, (gr/dL)	12.4±1.9	12.5±1.9	12±2.2	0.160
• Platelet, (10^3 µL)	228.5±83.6	224.8±79.9	244.5±97.4	0.137
• UAR	1.7±0.8	1.5±0.5	2.5±1.1	**<0.001**

CAD: Coronary artery disease, COPD: Chronic obstructive pulmonary disease, CVA: Cerebrovascular accident, DM: Diabetes mellitus, DVT: Deep venous thrombosis, eGFR: Estimated glomerular filtration rate, EF: Ejection fraction, hs-cTnI: High-sensitivity cardiac troponin I, LV: Left ventricle, PESI: Pulmonary embolism severity index, RV/LV: Right ventricle-to-left ventricle diameter, sPAP: Systolic pulmonary artery pressure, TAPSE: Tricuspid annular plane systolic excursion, UAR: Uric acid-to-albumin ratio. P-value <0.05 was regarded as statistically significant

Table 3. Baseline characteristics of patients grouped by UAR

Variables	Total population (n=261)	Low-UAR (n=161)	High-UAR (n=100)	p-value
Comorbidity and clinical properties				
Age, (years)	63.6±15.9	59.2±15.7	70.8±13.7	**<0.001**
Female sex, n (%)	157 (60.2)	92 (57.1)	65 (65)	0.258
CAD, n (%)	33 (12.6)	12 (7.5)	21 (21)	**0.003**
DM, n (%)	54 (20.7)	38 (23.6)	16 (16)	0.188
Hypertension, n (%)	124 (47.5)	65 (40.4)	59 (59)	0.005
CVA, n (%)	14 (5.4)	7 (4.3)	7 (7)	0.355
COPD, n (%)	31 (11.9)	20 (12.4)	11 (11)	0.882
Heart failure, n (%)	13 (5)	2 (1.2)	11 (11)	**0.001**
Malignancy history, n (%)	32(12.3)	17 (10.6)	15 (15)	0.288
DVT, n (%)	75 (28.7)	49 (30.4)	26 (26)	0.529
PESI score	131.2±45.3	120.8±41.8	147.8±46	**<0.001**
Echocardiographic parameters				
• LV-EF (%)	58 [55-60]	60 [55-60]	55 [55-60]	**0.004**
• sPAP, (mmHg)	47.9±11.7	46.3±11.7	48.4±10.7	0.156
• TAPSE, (mm)	14.9±3.1	15.3±3.1	14.2±3	**0.006**
• RV/LV ratio >1, n (%)	167 (64)	96 (59.6)	71 (71)	0.084
Laboratory				
• hs-cTnI, (pg/mL)	65.5 [25-215]	52.5 [18-182]	86.6 [38.5-250]	**0.022**
• D-dimer, (ng/mL)	4.4 [1.8.-7.9]	4.4 [1.6-7.8]	4.4 [1.9-7.9]	0.812
• eGFR, (mL/min/1.73 m^2)	76.1±25.7	85.5±22.3	61±23.5	**<0.001**
• Hemoglobin, (gr/dL)	12.4±1.9	12.6±1.9	12.1±1.9	0.051
• Platelet, (10^3 µL)	228.5±83.6	225.9±81.5	232.7±87.1	0.519

CAD: Coronary artery disease, COPD: Chronic obstructive pulmonary disease, CVA: Cerebrovascular accident, DM: Diabetes mellitus, DVT: Deep venous thrombosis, eGFR: Estimated glomerular filtration rate, EF: Ejection fraction, hs-cTnI: High-sensitivity cardiac troponin I, LV: Left ventricle, PESI: Pulmonary embolism severity index, RV/LV: Right ventricle-to-left ventricle diameter, sPAP: Systolic pulmonary artery pressure, TAPSE: Tricuspid annular plane systolic excursion, UAR: Uric acid to albumin ratio. P-value <0.05 was regarded as statistically significant

Discussion

This retrospective study evaluated the prognostic value of the UAR in patients with acute PE. ROC curve analysis showed that UAR has a significant predictive performance for 30-day mortality in the acute PE population. Elevated UAR and the history of malignant disease were identified as independent predictors of mortality within 30 days. This is the first investigation, to the best of our knowledge, to assess the prognostic value of the UAR in this patient population.

PE remains one of the contributing factors to high mortality rates from cardiovascular diseases. Although clinical prediction models such as PESI and simplified PESI are widely used, they rely primarily on demographic and clinical variables. Also, in patients with acute PE, various diagnostic tools—including cardiac troponins, CTPA, and transthoracic echocardiography—have been employed to assess right ventricular dysfunction and predict short-term outcomes. Additionally, emerging laboratory biomarkers such as H-FABP, NT-proBNP, and copeptin have been shown to have further prognostic information in observational cohort studies. While their role in guiding therapeutic strategies has not been validated through randomized controlled trials yet (4). These scores do not fully reflect some of the mechanisms, such as oxidative stress, inflammation, and endothelial injury, that may affect the prognosis in these patients.

UA contributes to vascular pathology through multiple mechanisms. It is not only an end-product of purine metabolism but also a pro-oxidant molecule that can stimulate inflammation and exacerbate endothelial dysfunction. Evidence suggests that high UA levels play a role in the stimulation of the renin-angiotensin-aldosterone system, increased oxidative stress, smooth muscle cell proliferation, and prothrombotic states (6,7,19-22). VTE is linked to the activation of both inflammatory

Table 4. Predictors of 30-day mortality in acute pulmonary embolism patients

Variables	Univariable OR (95% CI)	p-value	Multivariable OR (95% CI)	p-value
Age	1.03 (1.01-1.06)	0.005	0.997 (0.968-1.026)	0.822
Female sex	0.865 (0.501-1.789)	0.865		
CAD	1.192 (0.485-2.93)	0.701		
Diabetes mellitus	0.704 (0.309-1.607)	0.405		
Hypertension	0.761 (0.381-1.345)	0.299		
CVA	2.563 (0.819-8.02)	0.106		
COPD	0.813 (0.296-2.236)	0.688		
Heart failure	2.005 (0.591-6.8)	0.418		
Malignancy history	4.311 (1.965-9.459)	**<0.001**	3.343 (1.218-9.170)	**0.019**
DVT	1.118 (0.568-2.2)	0.747		
LV-EF	0.953 (0.895-1.014)	0.125		
sPAP	1.033 (1.006-1.062)	**0.018**	1.012 (0.978-1.047)	0.499
TAPSE	0.905 (0.814-1.005)	0.063		
RV/LV ratio >1	2.223 (1.076-4.592)	**0.031**		
eGFR	0.966 (0.952-0.984)	**<0.001**	0.989 (0.971-1.007)	0.236
Baseline hemoglobin	0.892 (0.76-1.046)	0.160		
Baseline platelet	1.003 (0.999-1.006)	0.139		
hs-cTnI	1.001 (1.00003-1.001)	**0.038**	1.000 (1.000-1.001)	0.319
D-dimer	1.017 (0.934-1.107)	0.700		
UAR	5.557 (3.21-9.688)	**<0.001**	4.798 (2.485-9.262)	**<0.001**

CAD: Coronary artery disease, COPD: Chronic obstructive pulmonary disease, CVA: Cerebrovascular accident, DVT: Deep venous thrombosis, EF: Ejection fraction, eGFR: Estimated glomerular filtration rate, hs-cTnI: High-sensitivity cardiac troponin I, LV: Left ventricle, RV/LVR: Right ventricle-to-left ventricle diameter, sPAP: systolic pulmonary artery pressure, TAPSE: Tricuspid annular plane systolic excursion, UAR: Uric acid to albumin ratio. P-value <0.05 was regarded as statistically significant

and coagulation pathways (23), and imbalances in these systems may increase the risk of acute PE. The Atherosclerosis Risk in Communities study, involving 14,126 participants, demonstrated a significant association between serum UA and VTE incidence (7). Moreover, 280 patients with previous VTE were evaluated in another study, and a significant association has also been identified between serum UA levels and the risk of recurrent VTE (24). Furthermore, among 337 patients with verified PE, those in the intermediate- and high-risk categories exhibited higher UA levels, which independently predicted mortality within 30 days (25).

Albumin is an indicator of both nutritional status and systemic inflammation. Owing to its negative acute-phase characteristics, systemic inflammation and oxidative stress cause a decrease in its concentration. Albumin exhibits anti-inflammatory, antioxidant, and anticoagulant effects by neutralizing reactive oxygen species, enhancing endothelial stability, and reducing platelet aggregation (26-31). Hypoalbuminemia has been associated with hypercoagulability, increased blood viscosity, and impaired vascular integrity, which all predispose to thrombotic complications (9). Evidence from meta-analyses reveals that hypoalbuminemia is a significant risk factor for VTE (9,10).

The UAR combines two biomarkers with opposite biological effects: UA has oxidative, inflammatory, and thrombogenic properties, whereas albumin has anti-inflammatory, antioxidant, and anti-thrombogenic capacity. Hence, UAR may more accurately reflect the overall pathophysiological processes than either marker alone. Recent studies have demonstrated that UAR independently predicts mortality in several cardiovascular diseases, such as acute myocardial infarction (32), unstable angina (33), and acute aortic dissection (34). We found UAR as an independent predictor of 30-day mortality. Our findings support the fact that systemic antithrombotic, antioxidant, and anti-inflammatory dysregulation plays a key role in physiopathology and the prognosis of PE.

UAR can be calculated easily using routine laboratory parameters that are inexpensive and widely available, without requiring specialized assays or additional costs. Incorporating UAR into existing risk assessment models

could enhance their early mortality predictive accuracy, particularly in patients with intermediate to high risk.

Study Limitations

This study has various limitations. First, it is a retrospective and single-center investigation that limits the ability to infer definitive causal conclusions and carries the potential for selection and information biases. Although consecutive patients were evaluated, the generalizability of the results may be affected due to the restriction of the final analysis to patients with complete data. Furthermore, statistical power may be reduced despite multivariable adjustment because of modest sample size and limited number of events that increase the risk of residual confounding. Second, the findings were confined to short-term mortality in acute PE patients, and long-term outcomes were not evaluated. Third, although patients with active infection, severe CKD, significant hepatic dysfunction, inflammatory or hematological disorders, gout, or urate-lowering therapy were excluded, several other conditions that may influence serum UA and albumin levels were not systematically assessed. Serum albumin levels may be affected by nutritional status, nephrotic proteinuria, protein-losing enteropathy, volume overload, or acute stress states. Besides, serum UA levels can be influenced by dietary factors, obesity, thyroid dysfunction, diseases that cause increased cell turnover, subclinical renal diseases, dehydration, genetic factors, and medications that are not included in our analysis, such as levodopa, theophylline, and anabolic steroids. The lack of detailed investigation of these potential confounding factors may have affected the observed results. Fourth, only baseline values of serum UA and albumin were considered, but serial measurements were not performed. Therefore, the prognostic value of the UAR dynamic change was not established. Despite these limitations, our findings highlight UAR as a readily accessible biomarker predicting short-term mortality risk in patients with acute PE.

Conclusion

In this study, the UAR was found to be an independent prognostic factor for 30-day mortality in acute PE. This simple, inexpensive biomarker, which is derived from routine tests, may enhance early risk stratification and guide more individualized management strategies when integrated into existing prognostic models. However, large-scale, multicenter prospective studies are needed to confirm these findings and clarify the clinical utility of UAR in the prognostic assessment of acute PE patients.

Ethics

Ethics Committee Approval: The study received approval from the Local Ethics Committee of University of Health Sciences Turkey, İstanbul Bağcılar Training and Research Hospital (decision number: 2025/10/15/099, date: 24.10.2025).

Informed Consent: In our study, data from patients between June 2011 and August 2024 were analyzed retrospectively.

Footnotes

Authorship Contributions

Surgical and Medical Practices: O.İ., E.D., M.F.D., C.T., S.B., S.Ö., Concept: O.İ., S.Ö., Design: O.İ., S.Ö., Data Collection or Processing: O.İ., E.D., M.F.D., C.T., S.B., S.Ö., Analysis or Interpretation: O.İ., Literature Search: O.İ., M.F.D., C.T., S.B., Writing: O.İ., E.D., S.Ö.

Conflict of Interest: No conflict of interest was declared by the authors.

Financial Disclosure: The authors declared that this study received no financial support.

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