

The Impact of Thrombolytic Therapy on in-Hospital Survival in Patients with Low-Intermediate and High-Intermediate Risk Pulmonary Embolism

Edibe Betül Börklü,¹ Tolga Sinan Güvenç,² Ömer Kozan³

¹Department of Cardiology, Dr. Siyami Ersek Thoracic and Cardiovascular Surgery Training and Research Hospital, İstanbul, Türkiye

²Department of Cardiology, Memorial Healthcare Group Göztepe Hospital, İstanbul, Türkiye

³Department of Cardiology, Başkent University İstanbul Hospital, İstanbul, Türkiye

This study was derived from the medical specialization thesis of the lead author, titled "Orta düşük ve orta yüksek riskli pulmoner embolide trombolitik tedavinin hastane içi sağkalıma etkisinin retrospektif olarak araştırılması" [Retrospective investigation of the effect of thrombolytic therapy on in-hospital survival in low-intermediate and high-intermediate risk pulmonary embolism], completed in 2017 at Dr. Siyami Ersek Thoracic and Cardiovascular Surgery Training and Research Hospital, İstanbul, Türkiye.

Cite This Article: Börklü EB, Güvenç TS, Kozan Ö. The Impact of Thrombolytic Therapy on in-Hospital Survival in Patients with Low-Intermediate and High-Intermediate Risk Pulmonary Embolism. Koşuyolu Heart J 2026;29(3):181–187

Address for Correspondence:

Edibe Betül Börklü

Department of Cardiology, Dr. Siyami Ersek Thoracic and Cardiovascular Surgery Training and Research Hospital, İstanbul, Türkiye

E-mail: edibe_betul@hotmail.com

Submitted: May 25, 2026

Revised: August 23, 2026

Accepted: August 26, 2026

Available Online: October 21, 2026



Copyright@Author(s) - Available online at kosuyoluheartjournal.com

OPEN ACCESS This work is licensed under a Creative Commons Attribution-ShareAlike 4.0 International License.



Abstract

Objective: The thrombolytic treatment is a subject of debate in acute pulmonary embolism (APE) with an intermediate risk for adverse outcomes. Our aim is to investigate retrospectively the effects of thrombolytics on mortality and other cardiovascular outcomes in intermediate-risk APE patients with further classification into low-intermediate or high-intermediate risk groups.

Methods: The study included 227 patients diagnosed as APE. Mortality risk stratification was done according to the 2014 European Society of Cardiology guidelines on APE. The primary outcome was in-hospital death related to APE. A composite of hemodynamic deterioration, cardiogenic shock, and total mortality was defined as the composite clinical endpoint.

Results: One hundred and sixty-nine (74.4%) of patients were classified as low-intermediate risk, and 58 (25.6%) of patients were classified as high-intermediate risk. The total number of patients who received alteplase (tissue plasminogen activator) was 157 (69.2%), with no difference between both groups ($p=0.97$). In the low-intermediate risk group, there were no differences in primary and composite endpoints regarding to thrombolytic therapy. In the high-intermediate risk group, both total mortality and APE-related mortality were lower in patients treated with thrombolytics ($p=0.025$ for both).

Conclusion: Classifying patients into low-intermediate and high-intermediate risk subsets is helpful for determining patients who may benefit from thrombolytic treatment.

Keywords: Intermediate-risk pulmonary embolism; mortality risk classification; pulmonary embolism; thrombolytic treatment.

Orta düşük ve Orta Yüksek Riskli Pulmoner Embolide Trombolitik Tedavinin Hastane İçi Sağkalıma Etkisi

Özet

Amaç: Olumsuz sonuçlar açısından orta riskli akut pulmoner emboli (APE) hastalarında trombolitik tedavi halen bir tartışma konusudur. Amacımız, trombolitiklerin orta riskli APE hastaları üzerindeki mortalite ve diğer kardiyovasküler sonuçlara etkilerini; hastaları düşük-orta risk veya yüksek-orta risk gruplarına ayırarak retrospektif olarak incelemektir.

Yöntem: Çalışmaya APE tanısı alan 227 hasta dahil edildi. Mortalite risk sınıflandırması, 2014 Avrupa Kardiyo-loji Derneği (ESC) APE kılavuzuna göre yapıldı. Birincil sonlanım noktası, APE ile ilişkili hastane içi ölüm olarak belirlendi. Hemodinamik bozulma, kardiyojenik şok ve toplam mortalitenin birleşimi ise birleşik klinik sonlanım noktası olarak tanımlandı.

Bulgular: Hastaların 169'u (%74,4) düşük-orta riskli, 58'i (%25,6) ise yüksek-orta riskli olarak sınıflandırıldı. Alteplaz (doku plazminojen aktivatörü; tPA) alan toplam hasta sayısı 157 (%69,2) idi ve her iki grup arasında bu açıdan bir fark yoktu ($p=0,97$). Düşük-orta risk grubunda, trombolitik tedavi açısından birincil ve birleşik sonlanım noktalarında bir fark saptanmadı. Yüksek-orta risk grubunda ise, trombolitiklerle tedavi edilen hastalarda hem toplam mortalite hem de APE ile ilişkili mortalite daha düşüktü (her ikisi için $p=0,025$).

Sonuç: Hastaların düşük-orta ve yüksek-orta risk alt gruplarına göre sınıflandırılması, trombolitik tedaviden fayda görebilecek hastaları belirlemek için faydalıdır.

Anahtar sözcükler: Orta riskli pulmoner emboli; mortalite risk sınıflandırması; pulmoner emboli; trombolitik tedavi.

Introduction

Risk stratification is a crucial part of evaluation for patients diagnosed with acute pulmonary embolism (APE). Patients with hypotension and organ hypoperfusion at presentation are candidates for immediate thrombus removal by thrombolysis or other means.^[1–3] On the other hand, patients with no clinical risk factors for adverse outcomes and no indication for right ventricular (RV) dysfunction or myocardial injury have a favorable short- and long-term outcome that can be safely managed as outpatients.^[4] Remaining patients, who were labeled as having an “intermediate” risk, are not under an imminent risk for death, but the 30-day mortality rate may reach up to 25%.^[5] Selecting an appropriate treatment strategy for this group is more complex, given that some but not all studies have shown an overall benefit of thrombolytic therapy over a more conservative strategy with heparin.^[6–9]

To clarify the treatment strategy in patients with an intermediate risk for adverse outcomes, the 2014 European Society of Cardiology (ESC) guidelines on the diagnosis and management of APE introduced, for the 1st time, the division of this group into “low-intermediate” and “high-intermediate” risk subgroups.^[10] This approach was based on observational studies suggesting an overall worse outcome for patients with a higher pulmonary embolism severity score (PESI), together with RV dysfunction and positive biomarkers for myocardial injury.^[11,12] Although early thrombolytic treatment is supported by at least one randomized controlled trial^[7] to reduce short-term (7-day) adverse outcomes in normotensive patients with RV dysfunction and elevated troponin levels at admission, its long-term efficacy has not been demonstrated.^[7,13,14] In addition, entry criteria for the latter study did not align with the 2014 ESC guideline recommendations, given that PESI or simplified PESI (sPESI) scores were omitted in identifying intermediate-risk patients. Therefore, to the best of our knowledge, data on the benefit of thrombolytic treatment for these “high-intermediate” risk patients, as defined by the 2014 ESC guidelines, are still lacking. In the present study, we retrospectively reviewed the medical records of patients diagnosed with APE and stratified their short-term mortality risk in accordance with the 2014 ESC guidelines. Consequently, we aimed to evaluate the comparative impact of thrombolytic therapy on mortality and other major adverse cardiovascular outcomes among patients categorized into “low-intermediate” and “high-intermediate” risk subgroups.

Materials and Methods

Patient Selection

For the present analysis, electronic medical records between January 2009 and December 2013 were retrospectively screened using the search term “pulmonary embolism.” Subsequently, patients aged 18 years or older with a diagnosis confirmed by computed tomography (CT) angiography were assessed for eligibility. Patients with incomplete records were excluded from the analysis. The remaining study population was retrospectively stratified into low, low-intermediate, high-intermediate, and high-risk groups as detailed below. After excluding the low- and high-risk categories, 227 out of 423 patients categorized into either the low-intermediate or high-intermediate risk subgroups were included in the final analysis. The demographic, clinical, laboratory, echocardiographic, and outcome data of the patients were retrieved from the electronic medical database.

The analysis was performed according to the 1975 Declaration of Helsinki, and this study was approved by the Haydarpaşa Numune Training and Research Hospital Ethic Committee (Date: 26.02.2018, Decision no: HNEAH-KAEK 2018/KK/28).

Definitions

The diagnosis of APE was confirmed with CT angiography in all patients. PESI and sPESI scores were computed according to previous definitions.^[15–17] A troponin level above the 99th percentile was accepted as a positive troponin value for myocardial injury. For all patients, evaluation of RV function was performed with echocardiography, and RV dysfunction was defined as a tricuspid regurgitation velocity higher than 2.8 m/s and one of the following: (a) absolute RV end-diastolic diameter equal to or higher than 42 mm on apical 4-chamber view or (b) A right-to-left ventricular end-diastolic diameter ratio higher than 0.9. Patients with PESI risk class III–V or a sPESI score ≥ 1 were classified as having an intermediate risk. Patients with an intermediate risk, RV dysfunction, and myocardial injury as defined above were classified as high-intermediate risk. Remaining patients, as well as those with a PESI risk class I–II or sPESI < 1 but who had either RV dysfunction or myocardial injury, were also classified as low-intermediate risk.^[10] Mortality was attributed to APE if no other causes were found and the death was preceded by hemodynamic deterioration. Fatal bleeding was defined as any bleeding event that resulted in in-hospital mortality. Total mortality was defined as all-cause mortality during the hospital stay.

Hemodynamic deterioration was defined as need for inotropic agents to maintain vital organ perfusion and blood pressure in the presence of an adequate RV preload. Cardiogenic shock was defined as hemodynamic deterioration together with signs of vital organ malperfusion or cardiopulmonary arrest. A composite of hemodynamic deterioration, cardiogenic shock, and total mortality was defined as the composite clinical endpoint.

Conventional Treatment and Thrombolytic Regimen

All patients received unfractionated heparin (UFH) or weight-adjusted low molecular weight heparin (enoxaparin) as soon as the diagnosis was established. The dose of UFH was adjusted to reach a target value of 1.5–2.5 times above the normal cut-off value for activated partial thromboplastin time. All patients who underwent thrombolytic treatment received a standard dose of 100 mg alteplase (tissue plasminogen activator [tPA]) that was infused within 2 h. Anticoagulation was achieved with UFH in all patients who received tPA.

Statistical Analysis

For continuous variables, normal distribution and quality of variances were checked with Shapiro–Wilk and Levene's tests, respectively. Continuous data were presented as median and interquartile range. Categorical variables were presented as percentages. The sample was divided into low-intermediate-risk and high-intermediate-risk groups. Further subgroups were formed according to administration of tPA at admission. Comparisons between individual groups and subgroups were performed using Student's T-test and Mann–Whitney U tests. Since the t-test is relatively robust for deviations from a normal distribution when the sample size for each group is large (≥ 30), the t-test was preferred over Mann–Whitney U test for the majority of analyses. For subgroup analyses of the high-intermediate risk group, Mann–Whitney test was used. Kaplan–Meier curves were drawn to determine in-hospital survival for patients who did or did not received tPA at admission. Differences between tPA (+) and tPA (–) subgroups were assessed using log-rank analysis. To determine univariate and multivariate determinants of in-hospital total mortality in tPA subgroups, a Cox proportional hazards model was used. Variables that had a $p < 0.05$ on univariate analyses were introduced the final multivariate models. No model was constructed for the low-intermediate risk group due to an exceedingly low number of events ($n=3$).

Results

The median age of the patients was 66 (range, 18–92) years, and 121 (53.3%) of the patients were females. 77 patients (33.9%) had RV dysfunction, and 215 (94.7%) had myocardial injury on initial evaluation. The median PESI and sPESI scores were 89 (range, 28–221) and 1 (range, 0–3), respectively. According to these findings, 169 (74.4%) of patients were classified as low-intermediate risk, while the remaining 58 (25.6%) patients were classified as high-intermediate risk. tPA was given to 157 (69.2%) of patients, with no difference between low-interme-

diate and high-intermediate risk groups ($p=0.97$). 14 (6.2%) of patients needed inotropic agents during hospitalization, while 12 (5.3%) patients had hemodynamic deterioration during follow-up. Total in-hospital mortality and composite end-point were observed in 14 (6.2%) and 17 (7.5%) patients, respectively. Median in-hospital follow-up was 8 (range, 1–34) days and 138 (60.8%) patients hospitalized for 7 days or longer.

Low-Intermediate versus High-Intermediate Risk Groups

Demographic, clinical, echocardiographic, and outcome data for low-intermediate and high-intermediate risk groups are provided in Table 1. Patients within the high-intermediate group were older and more frequently had hypertension, diabetes, previous coronary artery disease, previous cerebrovascular disease, heart failure, and chronic obstructive pulmonary disease. RV dysfunction was more frequent in the high-intermediate risk group, along with a higher systolic pulmonary artery pressure (Table 1). There were no differences regarding to troponin value. Thrombolytic treatment was given to a similar number of patients in each risk group. Finally, APE-related mortality, cardiogenic shock, and inotrope requirement during follow-up were all significantly higher in the high-intermediate risk group (all $p<0.05$).

Patient Characteristics in Subgroups that Received Thrombolytic Therapy

Data regarding the demographic, clinical, echocardiographic, laboratory variables, and outcomes for subgroups of patients who did or did not receive thrombolytic treatment are presented in Appendix a and b. In the low-intermediate risk group, patients who received thrombolytic treatment had lower oxygen saturation and tricuspid annular plane systolic excursion (TAPSE) values ($p<0.005$ for both). In contrast, there were nearly no differences in patient characteristics regarding to tPA (+) and (–) subgroups in the high-intermediate risk patients (Appendix a, b). However, patients who did not receive tPA at admission had a higher PESI score, which did not reach statistical significance.

Outcomes for Patients that Received Thrombolytic Therapy

In the low-intermediate risk group, there were no differences regarding APE-related mortality and composite endpoint in patients who did or did not receive thrombolytic therapy. In the high-intermediate risk group, APE-related mortality was lower in patients treated with thrombolytics ($p=0.008$). In Kaplan–Meier analysis, there was a trend toward higher in-hospital survival in patients treated with thrombolytics (log-rank $p=0.098$) in the whole study group, though it was not statistically significant. Similarly, no significant difference in in-hospital survival was observed within the low-intermediate risk group (log-rank $p=0.26$). In contrast, among high-intermediate risk patients, survival was numerically higher in the tPA (+) group, demonstrating a notable clinical trend despite not reaching formal statistical significance (log-rank $p=0.10$).

Table 1. Demographic, clinical, echocardiographic and outcome data for low-intermediate and high-intermediate risk groups

	Mortality risk class		p		Mortality risk class		p
	Low-intermediate (n=120) n (%)	High-intermediate (n=107) n (%)			Low-intermediate (n=120) n (%)	High-intermediate (n=107) n (%)	
Age				Min–Max (Median)	12–32 (20)	14–32 (20)	
Mean±SD	62.51±16.36	66.66±15.23	0.664 ^a	Oxygen Saturation (%)			
Min–Max (Median)	18–89 (65)	24–92 (69)		Mean±SD	92.95±4.98	91.58±5.81	0.088 ^a
Gender				Min–Max (Median)	54.6–100 (94)	55–98.6 (93)	
Female	69 (57.5)	52 (48.6)	0.180 ^c	PESI score			
Male	51 (42.5)	55 (51.4)		Mean±SD	84.77±28.28	105.58±35.11	0.022 ^{b*}
Smoking				Min–Max (Median)	28–221 (81)	34–206 (99)	
Absent	70 (58.3)	66 (61.7)	0.607 ^c	Systolic blood pressure (mmHg)			
Present	50 (41.7)	41 (38.3)		Mean±SD	125.13±19.04	121.57±19.29	0.441 ^a
Hypertension				Min–Max (Median)	80–211 (121.5)	90–185 (120)	
Absent	65 (54.2)	41 (38.3)	0.017 ^{c*}	Echo RV diameter (mm)			
Present	55 (45.8)	66 (61.7)		Mean±SD	33.7±5	42.4±7	0.253 ^a
Diabetes				Min–Max (Median)	23–40 (35)	22–60 (42)	
Absent	94 (78.3)	67 (62.6)	0.009 ^{c**}	TAPSE (mm)			
Present	26 (21.7)	40 (37.4)		Mean±SD	17.95±3.43	14.75±3.16	0.838 ^a
Coronary artery disease				Min–Max (Median)	10–30 (18)	8–25 (15)	
Absent	103 (85.8)	80 (74.8)	0.035 ^{c*}	sPAP (mmHg)			
Present	17 (14.2)	27 (25.2)		Mean±SD	50.28±10.51	57.86±15.51	0.049 ^{a**}
Myocardial infarction				Min–Max (Median)	30–75 (50)	30–130 (55)	
Absent	112 (93.3)	96 (89.7)	0.326 ^c	RV dilatation (≥42 mm)			
Present	8 (6.7)	11 (10.3)		Absent	120 (100)	30 (28)	0.001 ^{c**}
Hypothyroidism				Present	0 (0)	77 (72)	
Absent	106 (88.3)	100 (93.5)	0.183 ^c	APE related mortality			
Present	14 (11.7)	7 (6.5)		Absent	119 (99.2)	96 (89.7)	0.004 ^{e**}
Hyperlipidemia				Present	1 (0.8)	11 (10.3)	
Absent	99 (82.5)	79 (73.8)	0.113 ^c	Composite endpoint			
Present	21 (17.5)	28 (26.2)		Absent	41 (34.5)	34 (32.1)	0.706 ^c
Cerebrovascular disease				Present	78 (65.5)	72 (67.9)	
Absent	117 (97.5)	98 (91.6)	0.047 ^{c*}	Hospital stay (days)			
Present	3 (2.5)	9 (8.4)		Mean±SD	9.35±4.31	9.11±4.9	0.537 ^b
Chronic kidney disease				Min–Max (Median)	1–34 (9)	1–31 (8)	
Absent	112 (93.3)	96 (89.7)	0.458 ^e	Prolonged hospitalization (≥7 days)			
Present	8 (6.7)	11 (10.3)		Absent	44 (36.7)	45 (42.1)	0.406 ^c
Heart failure				Present	76 (63.3)	62 (57.9)	
Absent	115 (95.8)	92 (86)	0.017 ^{e*}	Inotrope requirement (follow-up)			
Present	5 (4.2)	15 (14)		Absent	118 (98.3)	95 (88.8)	0.007 ^{c**}
COPD				Present	2 (1.7)	12 (11.2)	
Absent	112 (93.3)	90 (84.1)	0.045 ^{e*}	Cardiogenic shock (follow-up)			
Present	8 (6.7)	17 (15.9)		Absent	119 (99.2)	96 (89.7)	0.004 ^{e**}
Heart rate (bpm)				Present	1 (0.8)	11 (10.3)	
Mean±SD	103.98±18.73	106.69±17.41	0.333 ^a	In-hospital hemorrhage related mortality			
Min–Max (Median)	44–170 (108.5)	61–154 (110)		Absent	120 (100)	106 (99.1)	0.471 ^d
Respiratory rate (breaths/min)				Present	0 (0)	1 (0.9)	
Mean±SD	20.83±3.56	21.91±4.15	0.027 ^{a*}				

^a: Student t Test; ^b: Mann-Whitney U Test; ^c: Pearson Chi-square test; ^d: Fisher's exact test; ^e: Yates Continuity Correction test; *, p<0.05; **, p<0.01. PESI: Pulmonary embolism severity score; Echo: Echocardiography; RV: Right ventricle; TAPSE: Tricuspid annular plane systolic excursion; sPAP: Systolic pulmonary artery pressure; APE: Acute pulmonary embolism; SD: Standard deviation; COPD: Chronic obstructive pulmonary disease.

Table 2. Multivariable logistic regression analysis of risk factors associated with mortality

	p	ODDS	%95 CI	
			Lower	Upper
Step 3a				
PESI score	0.095	1.023	0.996	1.050
TAPSE at admission (mm)	0.014*	0.438	0.227	0.846
tPA(no)	0.061	10.137	0.902	113.94
Smoking (yes)	0.685	0.561	0.034	9.168
sPAP at admission (mmHg)	0.256	1.030	0.979	1.084

Values are shown from the final step of the regression model. *: $p < 0.05$. PESI: Pulmonary embolism severity score; TAPSE: Tricuspid annular plane systolic excursion; tPA: Tissue plasminogen activator; sPAP: Systolic pulmonary artery pressure; CI: Confidence interval.

However, the prognostic value of these variables became more evident in the multivariable logistic regression analysis. For the entire study population, lower TAPSE values were identified as the sole independent predictor of mortality (Table 2). Although not included in the main table, further subgroup analysis of patients with high-intermediate risk APE revealed that total PESI score and neutrophil count were significant independent predictors of mortality. Furthermore, while thrombolytic treatment was not associated with mortality in the overall group ($p=0.18$), it demonstrated a significant survival benefit specifically within this high-intermediate risk subgroup (hazard ratio: 0.182, 95% confidence interval: 0.05–0.73, $p=0.045$).

Discussion

Despite several limitations, the findings from the present study provide an argument favoring the risk stratification approach suggested by the 2014 ESC APE guideline. Although our study design was based on the 2014 guidelines, the risk stratification used herein remains fundamentally aligned with the 2019 ESC update, confirming the enduring clinical relevance of our results.^[18] Major findings of the present analysis can be summarized as follows: (i) Patients in high-intermediate risk group were at an increased risk for early mortality, (ii) Patients with high-intermediate risk APE are older and more frequently have serious comorbidities compared to patients with low-intermediate risk APE, (iii) Thrombolytic treatment at admission seems to provide an in-hospital survival benefit for patients with high-intermediate risk APE but not for patients with low-intermediate risk APE, (iv) Total PESI score and neutrophil count have incremental prognostic value in intermediate risk patients, even after adjusting for low- and high-intermediate risk classification. This latter finding may be useful for further risk classification in high-intermediate risk patients that may benefit from more advanced treatment for APE. Several studies have shown that PESI score, sPESI score, increased troponin I or T, and RV dysfunction as prognostic markers for normotensive APE patients.^[10,19–21] In a recent systematic review and meta-analysis, Elmewafy et al.^[22] demonstrated that troponin elevation was consistently linked not only to increased short-term mortality but also to clinical deterioration indicators such as intensive care unit admission, hypotension, and RV dilatation. 2014 ESC guidelines recommended using a combination

of clinical severity scores (PESI and sPESI) together with imaging markers of RV dysfunction and biomarkers of myocardial injury (troponins) to identify normotensive APE patients at highest risk for adverse outcomes.^[10] However, to the best of our knowledge, the prognostic capability of this risk classification strategy has not been evaluated in a prospective or retrospective study. The present analysis confirms that patients within high-intermediate risk APE have approximately eightfold risk for mortality following adjustment for other variables. In addition, we found that patients with high-intermediate risk APE were older and more frequently have comorbidities, although none of these factors were found as univariate or multivariate determinants for in-hospital mortality. However, the present analysis was limited to the in-hospital period, and these factors could be detrimental for the long-term outcomes of high-intermediate risk patients. Findings from the PEITHO trial suggest that mortality during long-term is much higher than mortality in the acute phase in a patient cohort with both RV dysfunction and positive troponin at baseline, where the cause of death was related with underlying conditions rather than long-term sequelae of APE.^[14] Since we have observed that comorbidities were more frequent in the high-intermediate risk patients than those with a low-intermediate risk, patients within the former group might be at a higher risk for death not only during the short term but also at the long-term compared with those within the latter group.

The effectivity of thrombolytic treatment in those without hemodynamic deterioration or cardiogenic shock at admission is a long-debated subject. While the present analysis has several limitations, it nonetheless suggests that thrombolytic treatment reduces the risk of APE-related mortality in the high-intermediate risk patients. This finding is a strength of the present analysis: The similarity in the baseline characteristics of patients within the high-intermediate risk group who did and did not receive thrombolysis (Appendix a, b). While the overall mortality rate for the whole study group was comparable to previous reports, in-hospital mortality rate was very high in the high-intermediate risk patients independent of thrombolytic treatment. In the PEITHO trial, mortality at 7 days was 1.2% in the tenecteplase group and 1.8% in the conventional treatment group.^[7] Becattini et al.^[11] have reported an overall 5.8% risk for patient with both RV dilatation and troponin elevation, while the composite endpoint was observed in 8.8% of patients. As both studies did not include PESI or sPESI scores as an inclusion criterion, the mortality rate between these studies and the present analysis was not directly comparable. In the high-intermediate risk group, the mean PESI score was 105.5 ± 35 , indicating a high mortality risk. This risk is likely further augmented by the presence of RV dysfunction and myocardial injury, since both parameters were omitted from the original PESI classification.^[10] Nonetheless, the present analysis suggests that integrating PESI/sPESI scores with RV dysfunction and myocardial injury markers can identify patients at the highest risk for mortality who may derive the most benefit from thrombolysis. This finding not only supports the recommendations of the 2014 ESC guidelines but also remains fully consistent with the 2019 ESC update, which further reinforces this integrated risk stratification as a cornerstone of clinical management.^[21]

In contrast to the high-intermediate risk group, patients with a low-intermediate risk had a significantly lower risk for in-hospital APE-related mortality independent of thrombolytic treatment. This finding is in line with previous trials and observations that found sole presence of RV dysfunction, “massive” APE, or symptoms is inadequate to identify patients who may benefit from systemic thrombolysis.^[8,23] In the present analysis, characteristics of patients who received thrombolytic treatment in the low-intermediate risk group were somewhat different from those who did not receive thrombolytic treatment. This last finding constitutes a serious compromise for the present analysis and limits conclusions that could be drawn from the outcome analysis in the low-intermediate risk group. Still, when considered together with the previous studies mentioned before, these findings nonetheless suggest that thrombolytic treatment is probably not useful in patients designated as having a low-intermediate risk.

When considered as a dichotomous parameter, the sPESI score offers a similar predictive accuracy to the original PESI score for identifying higher-risk APE patients.^[10,24] Calculating sPESI is much simpler than the original PESI, and the presence of only one positive parameter is adequate to identify higher-risk patients.^[24] In the present study, we have found that the original PESI, but not sPESI, was significantly different between low-intermediate and high-intermediate risk patients, and the original PESI score remained as a useful tool for predicting mortality even after adjustment for other variables such as RV dysfunction or troponin. Since only patients with intermediate-risk APE were included in the present analysis, this finding did not contradict the findings of Jiménez et al.,^[16] but rather suggests a possible role of the PESI score for further risk classification of intermediate-risk APE. The clinical usefulness of the PESI score to determine intermediate-risk patients who may benefit from thrombolysis or other reperfusion strategies remains unclear and needs further research. A more practical approach might be the early identification of patients with high-intermediate risk APE in the emergency department by utilizing the PESI score before other test results (cardiac troponin and RV imaging) become available, given that this scoring system is entirely based on clinical variables.

Study Limitations

The present study was a retrospective analysis conducted using a patient database from a single institution; therefore, all limitations pertinent to retrospective analyses were present. Patients in whom thrombolytics were not given were treated with either UFH or low molecular weight heparin, while all patients who received tPA were anticoagulated using UFH. This difference in the baseline anticoagulation treatment might have affected the results. Data were only present for in-hospital events and mortality, so no long-term extrapolations should be made from these findings. While patient characteristics for tPA subgroups were similar in high-intermediate risk group, patients in the low-intermediate risk group that received thrombolytic treatment had different baseline characteristics compared to those that did not receive thrombolytics. An exceedingly low event rate in the low-intermediate risk group nonetheless suggests that thrombolytic treatment is probably unnecessary to reduce the risk of early mortality and might be even hazardous.

Conclusion

Patients with low-intermediate and high-intermediate risk APE have very different baseline clinical and echocardiographic characteristics that may affect short- and long-term outcomes. Thrombolytic treatment did not improve in-hospital outcomes when used indiscriminately in all intermediate-risk patients. Low-intermediate risk patients had a very low in-hospital event rate and excellent survival, regardless of thrombolytic therapy. In contrast, cardiogenic shock and in-hospital mortality due to APE were higher in the high-intermediate risk patients, who showed a better survival rate when thrombolytic treatment was administered. This real-world analysis validates the prognostic utility of sub-classifying intermediate-risk patients into low-intermediate and high-intermediate categories, as initially introduced by the 2014 ESC guidelines and maintained in subsequent updates. Our findings further emphasize the importance of this stratification in identifying patients who may derive the greatest survival benefit from thrombolytic therapy or other advanced therapies.

Disclosures

Ethics Committee Approval: The study was approved by the Haydarpaşa Numune Training and Research Hospital Ethics Committee (no: HNEAH-KAEK 2018/KK/28, date: 26/02/2018).

Informed Consent: Retrospective study.

Conflict of Interest Statement: The authors have no conflicts of interest to declare.

Funding: The authors declared that this study has received no financial support.

Use of AI for Writing Assistance: None declared.

Author Contributions: Concept – E.B.B., Ö.K.; Design – E.B.B., Ö.K.; Supervision – Ö.K.; Resource – E.B.B., Ö.K.; Materials – E.B.B., T.S.G., Ö.K.; Data collection and/or processing – E.B.B., T.S.G.; Data analysis and/or interpretation – T.S.G., E.B.B., Ö.K.; Literature search – E.B.B., T.S.G.; Writing – E.B.B.; Critical review – Ö.K., E.B.B., T.S.G.

Peer-review: Externally peer-reviewed.

References

- Butt S, Ashiq F, Kumar A, Senniappan R, Mashaal Z, Bhatnagar G, et al. Effective management of acute pulmonary embolism and deep vein thrombosis: insights from a case series on procedure benefits. *Cureus* 2024;16:e72694.
- Mohamad T, Kanaan E, Ogieuhi JJ, Mannaparambil AS, Ray R, Al-Nazer LWM, et al. Thrombolysis vs anticoagulation: unveiling the trade-offs in massive pulmonary embolism. *Cureus* 2024;16:e52675.
- Alkinj B, Pannu BS, Apala DR, Kotecha A, Kashyap R, Iyer VN. Saddle vs nonsaddle pulmonary embolism: clinical presentation, hemodynamics, management, and outcomes. *Mayo Clin Proc* 2017;92:1511–8.
- Bledsoe JR, Woller SC, Stevens SM, Aston V, Patten R, Allen T, et al. Management of low-risk pulmonary embolism patients without hospitalization: the low-risk pulmonary embolism prospective management study. *Chest* 2018;154:249–56.
- Alabi FO, Ghaneie A, Koury I, Alkhateeb HA, Liu J, Guo M, et al. Management of intermediate high-risk pulmonary embolism: a single-center retrospective study. *Front Cardiovasc Med* 2025;12:1483968.
- Mi YH, Liang Y, Lu YH, Li YM, Liu WX, Qian W. Recombinant tissue plasminogen activator plus heparin compared with heparin alone for patients with acute submassive pulmonary embolism: one-year outcome. *J Geriatr Cardiol* 2013;10:323–9.

7. Meyer G, Vicaut E, Danays T, Agnelli G, Becattini C, Beyer-Westendorf J, et al. Fibrinolysis for patients with intermediate-risk pulmonary embolism. *N Engl J Med* 2014;370:1402–11.
8. Hamel E, Pacouret G, Vincetelli D, Forissier JF, Peycher P, Pottier JM, et al. Thrombolysis or heparin therapy in massive pulmonary embolism with right ventricular dilation: results from a 128-patient monocenter registry. *Chest* 2001;120:120–5.
9. Rehman NU, Dar MI, Bansal M, Kasliwal RR. Clinical outcomes of submassive pulmonary embolism thrombolysis-an Indian experience. *Egypt Heart J* 2020;72:87.
10. Konstantinides SV, Torbicki A, Agnelli G, Danchin N, Fitzmaurice D, Galie N, et al. Task force for the diagnosis and management of acute pulmonary embolism of the European Society of Cardiology (ESC). 2014 ESC guidelines on the diagnosis and management of acute pulmonary embolism. *Eur Heart J* 2014;35:3033–69.
11. Becattini C, Casazza F, Forcione C, Porro F, Fadin BM, Stucchi A, et al. Acute pulmonary embolism: external validation of an integrated risk stratification model. *Chest* 2013;144:1539–45.
12. Konstantinides S, Goldhaber SZ. Pulmonary embolism: risk assessment and management. *Eur Heart J* 2012;33:3014–22.
13. Marín V A, Gazmuri B JT, Andresen V M, Andresen M. Thrombolytic therapy for submassive/ intermediate risk Pulmonary Embolism Evidence and suggestions after PEITHO trial. *RevMedChil* 2015;143:895–904.
14. Konstantinides SV, Vicaut E, Danays T, Becattini C, Bertolotti L, Beyer-Westendorf J, et al. Impact of thrombolytic therapy on the long-term outcome of intermediate-risk pulmonary embolism. *J Am Coll Cardiol* 2017;69:1536–44.
15. Donzé J, Le Gal G, Fine MJ, Roy PM, Sanchez O, Verschuren F, et al. Prospective validation of the Pulmonary Embolism Severity Index. A clinical prognostic model for pulmonary embolism. *Thromb Haemost* 2008;100:943–8.
16. Jiménez D, Yusen RD, Otero R, Uresandi F, Nauffal D, Laserna E, et al. Prognostic models for selecting patients with acute pulmonary embolism for initial outpatient therapy. *Chest* 2007;132:24–30.
17. Chan CM, Woods C, Shorr AF. The validation and reproducibility of the pulmonary embolism severity index. *J Thromb Haemost* 2010;8:1509–14.
18. Konstantinides SV, Meyer G, Becattini C, Bueno H, Geersing GJ, Harjola VP, et al. 2019 ESC Guidelines for the diagnosis and management of acute pulmonary embolism developed in collaboration with the European Respiratory Society (ERS): The Task Force for the diagnosis and management of acute pulmonary embolism of the European Society of Cardiology (ESC) *Eur Respir J* 2019;54:1901647.
19. Lankeit M, Jiménez D, Kostrubiec M, Dellas C, Hasenfuss G, Pruszczyk P, et al. Predictive value of the high-sensitivity troponin T assay and the simplified Pulmonary Embolism Severity Index in hemodynamically stable patients with acute pulmonary embolism: a prospective validation study. *Circulation* 2011;124:2716–24.
20. Kong WF, Wang YT, Yin LL, Pu H, Tao KY. Clinical risk stratification of acute pulmonary embolism: comparing the usefulness of CTA obstruction score and pulmonary perfusion defect score with dual-energy CT. *Int J Cardiovasc Imaging* 2017;33:2039–47.
21. Sanchez O, Trinquart L, Planquette B, Couturaud F, Verschuren F, Caille V, et al. Echocardiography and pulmonary embolism severity index have independent prognostic roles in pulmonary embolism. *Eur Respir J* 2013;42:681–8.
22. Elmewafy AT, Waller J, Alaguraja P, Desor K, Antoun I. The prognostic value of troponin in acute pulmonary embolism: a systematic review and meta-analysis. *Catheter Cardiovasc Interv* 2025;106:3899–16.
23. Prosperi-Porta G, Ronksley P, Kiamanesh O, Solverson K, Motazedian P, Weatherald J. Prognostic value of echocardiography-derived right ventricular dysfunction in haemodynamically stable pulmonary embolism: a systematic review and meta-analysis. *Eur Respir Rev* 2022;31:220120.
24. Jiménez D, Aujesky D, Moores L, Gómez V, Lobo JL, Uresandi F, et al. Simplification of the pulmonary embolism severity index for prognostication in patients with acute symptomatic pulmonary embolism. *Arch Intern Med* 2010;170:1383–9.

Appendix a. Demographic and laboratory data for low-intermediate and high-intermediate risk patients who did or did not receive thrombolytic treatment

	Mortality risk class				p	
	Low-intermediate (n=120)		p	High-intermediate (n=107)		
	tPA (–) (n=41)	tPA (+) (n=79)		tPA (–) (n=29)		tPA (+) (n=78)
Age						
Mean±SD	65.71±17.4	60.85±15.65	0.123 ^a	70.14±15.16	65.37±15.14	0.151 ^a
Min–Max (Median)	18–87 (70)	19–89 (64)		32–90 (72)	24–92 (69)	
Gender; n (%)						
Female	25 (61)	44 (55.7)	0.579 ^c	14 (48.3)	38 (48.7)	0.968 ^c
Male	16 (39)	35 (44.3)		15 (51.7)	40 (51.3)	
Smoking; n (%)						
Absent	21 (51.2)	49 (62)	0.255 ^c	20 (69)	46 (59)	0.345 ^c
Present	20 (48.8)	30 (38)		9 (31)	32 (41)	
Hypertension; n (%)						
Absent	20 (48.8)	45 (57)	0.394 ^c	11 (37.9)	30 (38.5)	0.960 ^c
Present	21 (51.2)	34 (43)		18 (62.1)	48 (61.5)	
Diabetes; n (%)						
Absent	35 (85.4)	59 (74.7)	0.178 ^c	16 (55.2)	51 (65.4)	0.332 ^c
Present	6 (14.6)	20 (25.3)		13 (44.8)	27 (34.6)	
Coronary artery disease; n (%)						
Absent	37 (90.2)	66 (83.5)	0.318 ^c	24 (82.8)	56 (71.8)	0.246 ^c
Present	4 (9.8)	13 (16.5)		5 (17.2)	22 (28.2)	
Myocardial infarction; n (%)						
Absent	40 (97.6)	72 (91.1)	0.262 ^d	25 (86.2)	71 (91)	0.485 ^d
Present	1 (2.4)	7 (8.9)		4 (13.8)	7 (9)	
Hypothyroidism; n (%)						
Absent	39 (95.1)	67 (84.8)	0.135 ^d	26 (89.7)	74 (94.9)	0.386 ^d
Present	2 (4.9)	12 (15.2)		3 (10.3)	4 (5.1)	
Hyperlipidemia; n (%)						
Absent	32 (78)	67 (84.8)	0.355 ^c	24 (82.8)	55 (70.5)	0.200 ^c
Present	9 (22)	12 (15.2)		5 (17.2)	23 (29.5)	
Cerebrovascular disease; n (%)						
Absent	39 (95.1)	78 (98.7)	0.269 ^d	23 (79.3)	75 (96.2)	0.011 ^{d*}
Present	2 (4.9)	1 (1.3)		6 (20.7)	3 (3.8)	
Chronic kidney disease; n (%)						
Absent	38 (92.7)	74 (93.7)	1.000 ^d	26 (89.7)	70 (89.7)	1.000 ^d
Present	3 (7.3)	5 (6.3)		3 (10.3)	8 (10.3)	
Heart failure; n (%)						
Absent	39 (95.1)	76 (96.2)	1.000 ^d	24 (82.8)	68 (87.2)	0.545 ^d
Present	2 (4.9)	3 (3.8)		5 (17.2)	10 (12.8)	
Chronic obstructive pulmonary disease; n (%)						
Absent	38 (92.7)	74 (93.7)	1.000 ^d	26 (89.7)	64 (82.1)	0.552 ^d
Present	3 (7.3)	5 (6.3)		3 (10.3)	14 (17.9)	
GFR epi						
Mean±SD	72.6±26.78	72.73±25.25	0.868^b	67.61±24.55	66.81±44.15	0.453^b
Min–Max (Median)	6.1–135.8 (71.4)	16.9–125.3 (71.9)		23.7–116.9 (68.6)	11.5–372 (59.7)	
GFR mdrd						
Mean±SD	74.97±28.21	74.81±32.68	0.947^b	71.11±25.65	64.29±28.75	0.202^b
Min–Max (Median)	7.6–153 (69.7)	18.8–265.5 (72.3)		26.3–120.4 (70.9)	13.7–190 (62)	
D dimer						
Mean±SD	5610.5±6740.26	4334.51±4432.51	0.321^b	4753±3689.19	4652.99±4828	0.574^b
Min–Max (Median)	235–37676 (3327)	36–19639 (2600)		50–11720 (3080)	269–20000 (3014)	
CKMB						

Appendix a. Cont.

		Mortality risk class				p	
		Low-intermediate (n=120)		p	High-intermediate (n=107)		
		tPA (–) (n=41)	tPA (+) (n=79)		tPA (–) (n=29)		tPA (+) (n=78)
	Mean±SD	28.68±21.15	37.29±113.34	0.351^b	24.94±13.57	29.18±17.65	0.329^b
	Min–Max (Median)	10–101 (22)	1.5–960 (21)		1.4–67 (22)	8–106 (24)	
Glucose							
	Mean±SD	140.83±59.17	159.71±88.93	0.167^b	159.52±67.51	168.97±79.15	0.705^b
	Min–Max (Median)	94–394 (121)	70–695 (136)		69–335 (148)	87–428 (140)	
Alt							
	Mean±SD	26.56±17.72	58.1±95.3	0.019^{b*}	56.17±75.25	81.41±223.39	0.570^b
	Min–Max (Median)	6–72 (20)	8–574 (26.5)		8–266 (26)	9–1713 (29)	
Ast							
	Mean±SD	32.29±18.83	57.32±100.02	0.187^b	64.79±75.32	59.05±92.36	0.601^b
	Min–Max (Median)	15–100 (26)	10–694 (28)		16–369 (39)	10–713 (37)	
LDH							
	Mean±SD	328.63±146.6	356.19±218.04	0.386^b	346.93±144.31	460.57±531.94	0.096^b
	Min–Max (Median)	205–856 (287)	162–1708 (299)		184–833(301.5)	94–4518 (351.5)	
Potassium							
	Mean±SD	4.54±0.71	4.29±0.61	0.053	4.41±0.59	4.61±0.7	0.163
	Min–Max (Median)	3.5–6.6 (4.4)	2.8–6.4 (4.3)		3.1–5.3 (4.5)	3.5–6.6 (4.6)	
Sodium							
	Mean±SD	138.37±3.12	138.56±3.55	0.764	138.83±2.77	138.53±3.7	0.692
	Min–Max (Median)	130–145 (139)	128–147 (139)		133–143 (139)	128–150 (139)	
WBC							
	Mean±SD	10.25±2.39	11.49±3.57	0.047[*]	11.52±3.02	12.4±4.56	0.338
	Min–Max (Median)	5.2–15.5 (10.4)	4–23.7 (10.9)		5.2–19 (10.8)	5.6–32.5 (11.8)	
Hemoglobin							
	Mean±SD	12.22±1.96	13.1±1.64	0.010^{**}	12.42±1.78	13.37±4.29	0.255
	Min–Max (Median)	8.5–16.3 (12.5)	8.7–16.9 (13.3)		8.3–15.1 (12.5)	4.6–45.6 (13)	
Hematocrit (HTC)							
	Mean±SD	36.9±5.49	38.94±4.9	0.040[*]	37.39±5.71	38.88±5.69	0.231
	Min–Max (Median)	27–48.6 (37.2)	26.7–51.5 (39.3)		23.7–47.5 (37.5)	27.2–55.1 (38.4)	
Platelet							
	Mean±SD	226.02±65.4	221.71±68.94	0.741	190.07±56.35	229.24±98.33	0.046[*]
	Min–Max (Median)	127–420 (215)	101–447 (218)		78–308 (185)	58.7–670 (210)	
Neutrophil							
	Mean±SD	7.79±2.4	8.93±5.52	0.505^b	10.63±8.56	9.75±4.44	0.952^b
	Min–Max (Median)	3.3–13.1 (7.9)	3.6–50 (7.9)		3.5–52.2 (8.7)	3.1–27.4 (8.9)	
Lymphocyte							
	Mean±SD	1.64±0.62	1.95±0.79	0.020^{b*}	1.67±1	1.82±1.02	0.280^b
	Min–Max (Median)	0.4–3.4 (1.6)	0.2–3.8 (1.9)		0.3–3.7 (1.3)	0.4–5.9 (1.6)	
Troponin							
	Mean±SD	0.61±0.6	0.74±1.9	0.110^b	0.74±0.96	1.1±1.7	0.324^b
	Min–Max (Median)	0.1–2.1 (0.3)	0–15.3 (0.2)		0.1–4.2 (0.4)	0–10 (0.5)	
Bun							
	Mean±SD	20.37±13.19	21.52±10.94	0.532^b	27.83±18.14	25.93±14.88	0.935^b
	Min–Max (Median)	8–94 (20)	4–62 (20)		11–83 (20)	8–105 (22)	
Creatine							
	Mean±SD	1.12±1.01	1.07±0.48	0.692^b	1.09±0.41	1.27±0.63	0.144^b
	Min–Max (Median)	0.5–7.3 (0.9)	0.3–3.4 (1)		0.5–2.3 (1)	0.4–4.4 (1.1)	
Troponin (+) (above reference value); n (%)							
	Absent	0 (0)	5 (6.5)	0.162^d	0 (0)	1 (1.4)	1.000^d
	Present	41 (100)	72 (93.5)		29 (100)	73 (98.6)	

∗: Student t-test; ^b: Mann–Whitney U Test; ∴: Pearson Chi-square test; ^d: Fisher's exact test; ∴: Yates Continuity Correction test; ∗: p<0.05; ∗∗: p<0.01. tPA: Tissue plasminogen activator, GFR: Glomerular filtration rate.

Appendix b. Clinical, echocardiographic and outcome data for low-intermediate and high-intermediate risk patients that did or did not receive thrombolytic treatment

	Mortality risk class				p	
	Low-intermediate (n=120)		p	High-intermediate (n=107)		
	tPA (–) (n=41)	tPA (+) (n=79)		tPA (–) (n=29)	tPA (+) (n=78)	
Heart rate (bpm)						
Mean±SD	98.05±20.48	107.06±17.08	0.012*	101.72±16.08	108.54±17.63	0.072
Min–Max (Median)	44–160 (96)	69–170 (110)		70–145 (105)	61–154 (110)	
Respiratory rate (breaths/min)						
Mean±SD	20.24±3.74	21.13±3.45	0.199	21.17±4.18	22.18±4.14	0.267
Min–Max (Median)	12–28 (20)	12–32 (20)		16–32 (20)	14–32 (20)	
Oxygen saturation (%)						
Mean±SD	94.17±2.59	92.32±5.77	0.049*	92.23±5.57	91.34±5.91	0.483
Min–Max (Median)	85–99 (94)	54.6–100 (93)		72–98 (94)	55–98.6 (92)	
PESI score						
Mean±SD	84.76±29.05	84.77±28.05	0.998b	117.38±41.75	101.19±31.49	0.065 ^b
Min–Max (Median)	28–157 (81)	36–221 (81)		45–206 (108)	34–205 (97)	
Systolic blood pressure (mmHg)						
Mean±SD	129.24±17.86	123±19.39	0.088	119.66±16.29	122.28±20.35	0.534
Min–Max (Median)	90–180 (130)	80–211 (120)		90–160 (120)	90–185 (120)	
Echo RV diameter (mm)						
Mean±SD	3.47±0.38	3.33±0.54	0.194	4.35±0.54	4.19±0.76	0.356
Min–Max (Median)	2.6–4 (3.6)	2.3–4 (3.5)		3.2–5.5 (4.2)	2.2–6 (4.2)	
TAPSE (mm)						
Mean±SD	18.88±2.9	17.26±3.66	0.036*	15.3±3.6	14.53±2.97	0.325
Min–Max (Median)	15–25 (19)	10–30 (17)		11–25 (15)	8–22 (15)	
sPAP (mmHg)						
Mean±SD	48.47±8.52	51.38±11.49	0.174	55.88±19.1	58.56±14.12	0.524
Min–Max (Median)	35–70 (50)	30–75 (50)		35–120 (50)	30–130 (55)	
RV dilatation (≥42 mm); n (%)						
Absent	41 (100)	79 (100)	–	8 (27.6)	22 (28.2)	1.000 ^d
Present	0 (0)	0 (0)		21 (72.4)	56 (71.8)	
APE related mortality; n (%)						
Absent	41 (100)	78 (98.7)	1.000 ^d	22 (75.9)	74 (94.9)	0.008 ^{d***}
Present	0 (0)	1 (1.3)		7 (24.1)	4 (5.1)	
Composite endpoint; n (%)						
Absent	17 (41.5)	24 (30.8)	0.243 ^c	9 (31)	25 (32.5)	0.888 ^c
Present	24 (58.5)	54 (69.2)		20 (69)	52 (67.5)	
Hospital stay (days)						
Mean±SD	9.05±5.18	9.51±3.82	0.303 ^b	8.1±4.68	9.49±4.96	0.144 ^b
Min–Max (Median)	1–34 (9)	2–21 (9)		2–22 (7)	1–31 (8.5)	
Prolonged hospitalization (≥7 days); n (%)						
Absent	18 (43.9)	26 (32.9)	0.236 ^c	15 (51.7)	30 (38.5)	0.217 ^c
Present	23 (56.1)	53 (67.1)		14 (48.3)	48 (61.5)	
Inotrope requirement (follow-up); n (%)						
Absent	40 (97.6)	78 (98.7)	1.000 ^d	24 (82.8)	71 (91)	0.300 ^d
Present	1 (2.4)	1 (1.3)		5 (17.2)	7 (9)	
Cardiogenic shock (follow-up); n (%)						
Absent	41 (100)	78 (98.7)	1.000 ^d	24 (82.8)	72 (92.3)	0.164 ^d
Present	0 (0)	1 (1.3)		5 (17.2)	6 (7.7)	
In-hospital hemorrhage related mortality; n (%)						
Absent	41 (100)	79 (100)	–	29 (100)	77 (98.7)	1.000 ^c
Present	0 (0)	0 (0)		0 (0)	1 (1.3)	

^a: Student t-test; ^b: Mann-Whitney U Test; ^c: Pearson Chi-square test; ^d: Fisher's exact test; ^e: Yates Continuity Correction test; *, p<0.05; **, p<0.01. tPA: Tissue plasminogen activator; PESI: Pulmonary embolism severity score; Echo: Echocardiography; RV: Right ventricle; TAPSE: Tricuspid annular plane systolic excursion; sPAP: Systolic pulmonary artery pressure; APE: Acute pulmonary embolism; SD: Standard deviation.