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Original Research

Predictors of Mortality in Pulmonary Embolism: A Real-Life Study

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ABSTRACT

Objective: The primary aim of this study was to investigate the mortality and associated factors in patients with pulmonary embolism.

Methods: A retrospective analysis was performed on adult patients with pulmonary embolism who applied to Gaziantep University Hospital between January 1, 2017, and January 1, 2023. All-cause mortality and related factors in pulmonary embolism patients were determined.

Results: This study included 152 patients with a median age of 59 years and 81 (53.3%) women. The all-cause mortality rate was 25.7%, and pulmonary embolism-related deaths were 1.3%. Age ($p<0.001$), chronic obstructive pulmonary disease (COPD) ($p=0.013$), heart failure ($p=0.018$), atrial fibrillation ($p=0.015$), massive pulmonary embolism ($p=0.029$), hemoglobin level ($p<0.001$) and NT-Pro BNP level ($p<0.001$) were significantly associated with increased all-cause mortality. In binary logistic regression analysis, for each unit of increasing pulmonary embolism severity index (PESI) score, mortality increased 2.2-fold (95% CI:1.03-5.09), massive PTE 1.6-fold (95% CI:0.14-17.86), anticoagulant duration (daily) 0.98-fold (95% CI:0.98-0.99) and Hb level (per unit Hb reduction) 0.67-fold (95% CI:0.45-1.02) mortality was increasing. There was no statistical difference between the number of hospitalization days for patients with low and high PESI and simplified PE severity index (sPESI) scores.

Conclusions: All-cause mortality in patients with pulmonary embolism increased with age, cardiac diseases, and COPD comorbidities. The PESI and sPESI scores used in the acute phase of PTE were found to be highly reliable in predicting all-cause mortality in PE patients. The diagnosis of massive PE and elevated NT-proBNP levels, a marker of right ventricular dysfunction, were factors that increased mortality.

Keywords: Pulmonary Embolism; Mortality; Risk Factors; Prognosis.

INTRODUCTION

Pulmonary thromboembolism (PTE) is still one of the most common pulmonary vascular diseases with high mortality. The average annual incidence of venous thromboembolism (VTE) is between 23-269/100.000 [1]. The most reported risk factors that increase susceptibility to VTE are a previous history of VTE, active cancer, major trauma, surgery, hospitalization, long flights, immobility, obesity, contraceptives containing estrogen, and concomitant heart diseases [1,2]. Clinical manifestations of PTE; it is divided into three massive, submassive, and nonmassive. Differentiation of the patient diagnosed with acute pulmonary thromboembolism as high-risk, intermediate-risk, or low-risk in terms of early mortality affects treatment options and prognosis [2,3]. Vital signs with electrocardiogram

(ECG), hemogram, lower extremity venous ultrasonography, echocardiography, d-dimer, cardiac troponin, and N-terminus pro-B-type natriuretic peptide (NT-proBNP) tests are used to determine the diagnosis and severity of pulmonary embolism (PE) [2]. The pulmonary embolism severity index (PESI) is used in the prognostic evaluation of PE which is especially effective in determining the first 30-day mortality [4,5]. Respectively, in the study conducted using PESI scoring, early mortality was reported as 0.7% and 1.2% in the low-risk group (Class I and II), while it was reported as 4.8%, 13.6%, and 25% in the high-risk group (Class III-V) [5]. The simplified pulmonary embolism severity index (sPESI), which includes fewer parameters, also had the same efficacy as the PESI index. A score of 0 indicates a low risk for a 30-day poor prognosis and a score of 1 ≥ indicates a high risk for a poor 30-day prognosis [6].

Although outpatient treatment is recommended for patients with low-risk PE with stable hemodynamics, they are still treated as inpatients for an average of 4 days in early discharge and 9 days in long hospitalizations [7]. It can be fatal at varying rates according to co-morbidities, risk status, and the clinical severity of PE. In a large recording study, the 30-day mortality due to PE was 1.8%, and the seven-day mortality was 1.1%. In the same cohort, all-cause mortality was 4.8% at 30 days and 1.9% at seven days [8]. Randomized controlled trials, prospective cohort treatment studies, and meta-analyses reported no difference in treatment efficacy and safety (recurrence, bleeding, and mortality) in nonmassive PTE patients with low risk of complications when compared to outpatient and inpatient treatment [2,9]. Clinically stable, with good cardiopulmonary reserve, low PESI or SPESI scores, easy access to treatment centers when necessary, and treatment compliance, 13-51% of patients were eligible for early discharge and outpatient treatment [9,10].

Main Points;

- The pulmonary embolism severity index (PESI) and simplified PE severity index (sPESI) used in the acute phase of PTE were quite reliable in predicting all-cause mortality in patients with pulmonary embolism.
- PESI score, massive PTE, Hb level, and NT-Pro BNP level were independent factors in pulmonary embolism-related mortality.
- No statistically significant difference was found in the mean hospitalization days of patients with sPESI scores of 0 and 1.

Although there are studies on in-hospital and first-trimester mortality rates of patients with pulmonary embolism, real-life data on long-term mortality rates are limited [11]. Determining pulmonary embolism severity and mortality determinants will facilitate the follow-up of patients. The primary aim of this study was to investigate the mortality and associated factors of patients with pulmonary embolism who were retrospectively diagnosed and treated in our hospital. The secondary purpose was to compare PESI or sPESI scores with outpatient and inpatient treatment rates and mortality rates for patients.

MATERIALS AND METHODS

Study Design and Participants

The design of this study is an observational, cross-sectional, retrospective study. This study was approved by the Gaziantep University Medical Ethics Committee (No. S-2022-268) and all steps were carried out in accordance with the principles of the Declaration of Helsinki.

This study included outpatient and/or inpatient patients older than 18 years of age who were diagnosed with pulmonary embolism and who were admitted to the department of pulmonary medicine and cardiology of the tertiary care hospital between January 1, 2017 and January 1, 2023. Cases with a definitive diagnosis of pulmonary embolism clinically and radiologically confirmed by spiral computed tomography (CT)-angiography or ventilation-perfusion scintigraphy were included in the study. The presence of deep vein thrombosis (DVT) was considered to be DVT in cases demonstrated by doppler ultrasound. Patients with clinical DVT findings but no findings on doppler ultrasound were considered to have no DVT. For massive PTE, hemodynamic instability parameters [need for cardiopulmonary resuscitation; systolic blood pressure < 90 mmHg; need for vasopressors to maintain systolic blood pressure ≥ 90 mmHg despite adequate fluid support; and presence of concomitant end-organ hypoperfusion (altered consciousness, oliguria/anuria, increased serum lactate level, systolic blood pressure drop of more than 40 mmHg) and/or echocardiographic findings] were required.

Patients with suspected PTE, cases with an unclear diagnosis that resulted in exitus at the time of diagnosis, no radiological evidence of thrombus by spiral CT-angiography or ventilation-perfusion scintigraphy, and chronic thromboembolic pulmonary hypertension (CTEPH) at presentation were all excluded.

Data Collection

Pulse rate, blood pressure, oxygen saturation (%SpO2), body mass index (BMI), comorbidities, previous history of VTE, hemogram, d-dimer, cardiac markers (NT-proBNP, troponin), echocardiography findings, sPESI, PESI score, diagnosis of massive PE, need for intensive care unit (ICU), length of hospital stay, anticoagulant treatments received, and deaths in hospital and after discharge were recorded. Since the study covered the pandemic period, those who were diagnosed with COVID-19 at the time of diagnosis and those whose cause of death was COVID-19 were recorded.

Anticoagulant therapies [low molecular weight heparin (LMWH), warfarin, new oral anti-coagulant (NOAC)], duration, and need for a vena cava filter were recorded at initial diagnosis and on the seventh day/after discharge. All-cause deaths while receiving PE treatment were recorded.

Statistical Analysis

IBM SPSS version 25.0 was used for statistical analysis. The compatibility of the numerical variables with normal distribution was tested by Shapiro-Wilk test. Mean and the standard deviation were used to represent the data fitting the normal distribution, and median values were used for those not fitting the normal distribution. Patients were divided into two subcategories as deceased and living groups according to their survival status The association of categorical variables with mortality calculated by chi-square analysis. The association of normally distributed numerical variables with mortality was measured by Student-T test, and the association of non-normally distributed variables with mortality were measured by Mann-Whitney U test. Binary logistic regression analysis was used to determine the independent factors determining mortality. In statistical analysis, $P<0.05$ was considered statistically significant at 95% confidence interval.

RESULTS

Study Population

The retrospective study included 152 patients with a median age of 59 years and 81 (53.3%) women. BMI (mean±SD) was 32.7±7.8. Concurrent with pulmonary embolism, 38.7% of the patients had DVT. A previous VTE was seen in 9.9% of the patients. The most common comorbidity was hypertension (27%), followed by diabetes mellitus (25%). COVID-19 was found in 13.5% of the patients at the time of PE diagnosis. Systolic blood pressure (SBP) (mean±SD) at admission was 119±19 mmHg, heart rate/min (mean±SD) was 94±16, and median respiratory rate/min was 22. In the echocardiography of the patients, the mean (mean±SD) sPAP was 38±20 mmHg, and 34% had right ventricular dysfunction. The sPESI score of 43.9% of the patients was 1 point. Of the study population, 17.3% were diagnosed with massive pulmonary embolism, and 28.9% needed an intensive care unit (ICU). In the study population, all patients were started on anticoagulant therapy, and one patient had a vena cava filter inserted due to bleeding. The duration of hospitalization (mean±SD) was 11±10 days. The duration of anticoagulant therapy for pulmonary embolism in the cohort was (mean±SD) 269±322 days (Table 1).

Table 1. Descriptive Statistics

Characteristics of the Patients (n=152)	n (%)
Age, years, median (min-max)	59 (17-92)
Female	81 (53.3)
BMI, mean ± SD	32.7±7.8
Family history of PTE	3 (2)
Hypertension	41 (27)
Diabetes mellitus	38 (25)
Chronic kidney disease	5 (3.3)
History of cancer	17 (11.2)
Active cancer	13 (8.6)
COPD	16 (10.5)
Heart failure	16 (10.5)
Atrial fibrillation	2 (1.3)
Connective tissue disease	3 (2)
History of MI	16 (10.6)
History of stroke	10 (6.6)
History of VTE	15 (9.9)
AFS	2 (5.1)
COVID-19 at diagnosis	20 (13.5)
Varicose vein	7 (6.2)
DVT at diagnosis	43 (38.7)
SBP (mmHg), mean±SD	119±19
HR/min, mean±SD	94±16
RR/ min, median (min-max)	22 (15-30)
spO2%, mean±SD	92±7
D-dimer (µg/L), median (min-max)	3.6 (0.23-35.2)
Troponin (ng/mL), median (min-max)	9.1 (0.01-4560.3)
NT-Pro BNP (pg/mL), median (min-max)	840.4 (10-23950)
Hb, median (min-max)	12.9±2
Platelets (10^9/L), mean±SD	280±115
sPAP(mmHg), mean±SD	38±20
Right ventricular dysfunction	35 (34)

PESI	2±1	
sPESI	0	83 (56.1)
	1	65 (43.9)
ICU	44 (28.9)	
Massive PTE	26 (17.3)	
Warfarin	91 (60.3)	
LMWH	147 (97.4)	
NOAC	79 (52.3)	
Unfractionated heparin	10 (6.6)	
Vena cava filter	1 (0.7)	
Hospitalization (days), mean±SD	11±10	
Anticoagulation therapy (days), mean±SD	269±322	
All-cause of deaths	39 (25.7)	
Cardiovascular deaths	10 (6.9)	
PE-related deaths	2 (1.3)	
Bleeding-related deaths	1 (0.7)	
Cancer-related deaths	8 (5.7)	
Non-cardiovascular non-cancer deaths	11 (7.7)	
Unknown deaths	9 (6.1)	
COVID-19-related deaths	2 (1.4)	

SD; Standard deviation, **BMI;** Body mass index, **PTE;** Pulmonary thromboembolism, **COPD;** Chronic obstructive pulmonary disease, **MI;** Myocardial infarction, **VTE;** Venous thromboembolism, **AFS;** Antiphospholipid syndrome, **DVT;** Deep vein thrombosis, **SBP;** Systolic blood pressure, **HR;** Heart rate, **RR;** Respiratory rate, **LMWH;** Low molecular weight heparin, **NOAC;** New oral anti-coagulant.

Mortality and Affecting Factors Analysis

The all-cause mortality rate of the study population was 25.7%. When the causes of mortality were analyzed; cancer and non-cardiovascular deaths were 7.7%, cardiovascular deaths were 6.9%, deaths of unknown cause were 6.1%, cancer-related deaths were 5.7%, COVID-19-related deaths were 1.4%, VTE-related deaths were 1.3%, and bleeding-related deaths were 0.7%. When the factors associated with mortality in pulmonary embolism patients in the survival and mortality groups were univariate analyzed, age ($p<0.001$), chronic obstructive pulmonary disease (COPD) ($p=0.013$), heart failure ($p=0.018$), and atrial fibrillation ($p=0.015$) were significantly associated with increased mortality. The diagnosis of massive PTE was a factor that significantly increased mortality ($p=0.029$). Among the scalar variables, decreased hemoglobin (Hb) level ($p<0.001$) and increased NT-Pro BNP level ($p<0.001$) were associated with increased mortality.

In the retrospective data analysis, all patients with pulmonary embolism received either LMWH or unfractionated heparin at first admission. Taking warfarin ($p=0.05$) or NOAC ($p=0.044$) as anticoagulant therapy was associated with significantly reduced mortality. Prolonged anticoagulant treatment duration (days) was associated with reduced mortality ($p<0.001$) (Table 2).

For factors influencing mortality, a multivariate analysis was undertaken. A binary logistic regression analysis was performed to determine the factors independently affecting mortality in pulmonary embolism; for each unit of increasing PESI score, 2.2-fold (95% CI: 1.03-5.09), massive PTE 1.6-fold (95% CI: 0.14-17.86), anticoagulant duration (per day) 0.98-fold (95% CI: 0.98-0.99), and Hb level (Table 3).

Table 2. Factors related to mortality

		Life status		p value
		Alive	Death	
		n (%)	n (%)	
Gender	Female	55 (67.9)	26 (32.1)	0.052
	Male	58 (81.7)	13 (18.3)	
Family history of PTE	no	110 (74.3)	38 (25.7)	
	yes	2 (66.7)	1 (33.3)	0.764
Hypertension	no	85 (76.6)	26 (23.4)	
	yes	28 (68.3)	13 (31.7)	0.299
Diabetes mellitus	no	87 (76.3)	27 (23.7)	
	yes	26 (68.4)	12 (31.6)	0.335
Chronic kidney disease	no	108 (73.5)	39 (26.5)	
	yes	5 (100)	0 (0)	0.182

History of cancer	no	103 (76.3)	32 (23.7)	
	yes	10 (58.8)	7 (41.2)	0.120
Active cancer at diagnosis	no	105 (76.1)	33 (23.9)	
	yes	7 (53.8)	6 (46.2)	0.080
COPD	no	97 (71.3)	39 (28.7)	
	yes	16 (100)	0 (0)	**0.013***
Heart failure	no	105 (77.2)	31 (22.8)	
	yes	8 (50)	8 (50)	**0.018***
Atrial fibrillation	no	113 (75.3)	37 (24.7)	
	yes	0 (0)	2 (100)	**0.015***
Connective tissue disease	no	110 (73.8)	39 (26.2)	
	yes	3 (100)	0 (0)	0.304
History of MI	no	103 (76.3)	32 (23.7)	
	yes	10 (62.5)	6 (37.5)	0.229
History of stroke	no	106 (74.6)	36 (25.4)	
	yes	7 (70)	3 (30)	0.745
History of VTE	no	101 (74.3)	35 (25.7)	
	yes	12 (80)	3 (20)	0.627
AFS	no	31 (83.8)	6 (16.2)	
	yes	2 (100)	0 (0)	0.536
COVID-19 at diagnosis	no	96 (75)	32 (25)	
	yes	15 (75)	5 (25)	1.000
Varicose Vein	no	85 (80.2)	21 (19.8)	
	yes	7 (100)	0 (0)	0.192
DVT at diagnosis	no	55 (80.9)	13 (19.1)	
	yes	35 (81.4)	8 (18.6)	0.946
Right ventricular dysfunction	no	54 (79.4)	14 (20.6)	
	yes	25 (71.4)	10 (28.6)	0.364
ICU	no	83 (76.9)	25 (23.1)	
	yes	30 (68.2)	14 (31.8)	0.267
Massive PTE	no	97 (78.2)	27 (21.8)	
	yes	15 (57.7)	11 (42.3)	**0.029***
Warfarin	no	37 (61.7)	23 (38.3)	
	yes	75 (82.4)	16 (17.6)	**0.004***
LMWH	no	3 (75)	1 (25)	
	yes	109 (74.1)	38 (25.9)	0.969
NOAC	no	48 (66.7)	24 (33.3)	
	yes	64 (81)	15 (19)	**0.044***
Unfractionated heparin	no	105 (74.5)	36 (25.5)	
	yes	7 (70)	3 (30)	0.755
Vena cava filter	no	112 (74.7)	38 (25.3)	
	yes	0 (0)	1 (100)	0.089

Chi-square Test. *Significant at the 0.05 level. **PTE:** Pulmonary thromboembolism**, COPD:** Chronic obstructive pulmonary disease. **MI:** Myocardial infarction. **VTE:** Venous thromboembolism. **AFS:** Antiphospholipid syndrome. **DVT:** Deep vein thrombosis, **ICU:** intensive care unit **LMWH:** Low molecular weight heparin. **NOAC:** New oral anti-coagulant.

Table 3. Binary Regression Analysis for Determining Factors Predicting Mortality in Pulmonary Embolism

Variables	B	S.E.	Wald	df	Sig.	Exp(B) 95% C.I.
NT-Pro BNP	0.00	0.00	2.28	1	0.131	1.000 (1.0-1.0)
Hb	-0.38	0.20	3.47	1	0.062	0.678 (0.45-1.02)
PESI score	0.83	0.40	4.18	1	0.041	2.298 (1.03-5.09)
Anticoagulation therapy (days)	-0.01	0.005	5.74	1	0.016	0.989 (0.98-0.99)
Atrial fibrillation	-19.89	40192.99	0.00	1	1.000	0.000 (0-0)
Massive PE	0.47	1.23	0.14	1	0.702	1.601 (0.14-17.86)
Constant	22.25	40192.99	0.00	1	1.000	4614585140.462

a.Variables: NT-Pro BNP, Hb (hemoglobin), PESI, Anticoagulation therapy (days), Atrial fibrillation. Massive PE (Pulmonary embolism), NOAC (new oral anti-coagulant). Confidence interval (CI).

Nagelkerke R Square: 0.647

Pulmonary Embolism Risk Scoring Analysis

The mean hospitalization day of patients with sPESI score 0 was 10.6±10.6 and 12.1±8.6 for patients with sPESI score 1, and no statistically significant difference was found between the two groups in terms of hospitalization days (p=0.332). In the analysis of all-cause mortality in the study registry, there was a statistically significant increase in mortality with increasing PESI score (p<0.001) and sPESI ≥ 1 (p<0.001) scores.

DISCUSSION

In this study, real-life mortality rates and associated factors for patients diagnosed with PTE were shown. In the study, the mortality rate due to pulmonary embolism was 1.3% and the all-cause mortality rate was 25.7%. In a retrospective pulmonary embolism study of 1023 patients with a mean follow-up of 4 years, the all-cause mortality rate was 35.5%, which is higher than the present study. In the same study, the most common causes of mortality after discharge were malignancy, cardiovascular and sepsis [12]. In this study, the non-cardiovascular non-cancer death rate, which includes infection and other causes, was the highest, with cardiovascular death ranking second and cancer-related death ranking third. In a long-term mortality study of pulmonary embolism in Turkey, mortality rates were 13.3% (95% CI: 10.1-16.7) at 30 days, 21.8% (95% CI: 17.8-25.9) at 90 days, 32.6% (95% CI: 28.1-37.0) at one year and 51% (95% CI: 46.0-55.8) at five years [13]. Previously, in a large multicenter study involving 123 countries for the investigation of deaths due to pulmonary embolism, it was shown that all deaths due to pulmonary embolism increased with age, although the rates differed according to countries, as in our study [14].

When the factors affecting all-cause mortality are analyzed, comorbidities of lung and heart diseases appear to be the factors that increase mortality. The comorbidities (heart and lung diseases) in the PESI scoring, which are more useful in predicting 30-day mortality, may similarly be useful in predicting long-term mortality [4,5]. In previous retrospective and prospective studies, the presence of cancer was found to be a factor associated with mortality in patients with pulmonary embolism [4,8,12,13,15]. In our study, a history of cancer or active cancer did not make a statistically significant difference in all-cause mortality. Variables included in the PESI scoring were not included in the regression analysis. In regression analysis of this cohort, each unit increase in PESI scoring increased all-cause mortality by 2.2-fold (95% CI: 1.03-5.09). PESI scoring at the PTE diagnosis stage has been useful in predicting mortality in this study. The statistical significance of PESI and sPESI scoring for mortality after 30 days in PTE was also shown in another study [13]. In our study cohort, the association of SBP value, heart rate, respiratory rate, respiratory rate, oxygen saturation percentage, and paO2 vital scalar values at diagnosis with all-cause mortality was not statistically significant. However, tachycardia, low SBP, respiratory failure (tachypnea and/or low SaO2), and syncope, alone or in combination, were associated with an increased risk of short-term pulmonary embolism mortality in acute PTE [2,4]. Although vital signs at diagnosis are important for acute pulmonary embolism mortality, their long-term effect is not clear. Our study found no statistically significant association between right ventricular (RV) dysfunction and sPAP with mortality. A meta-analysis suggested that RV dysfunction on echocardiography is associated with an increased risk of short-term mortality in patients who appear hemodynamically stable on admission [16]. In our study, massive PTE was found to be an independent risk factor for mortality. In previous studies, massive PTE short- and long-term mortality rates were higher than nonmassive PTE [3,17,18]. A large case series study showed a 17.5-fold increased risk of fatal PTE in patients presenting with symptomatic massive PTE [3]. In the regression analysis

performed in this study, patients with a diagnosis of massive PTE had a 1.6-fold (95% CI: 0.14-17.86) increase in all-cause mortality. It was previously shown that thrombolytic therapy in acute massive pulmonary embolism made no difference in long-term mortality after 30 days [19]. Therefore, close follow-up of patients diagnosed with massive PTE who received thrombolytic therapy in the acute phase and who received anticoagulants may be useful after discharge. In this study, blood Hb values and NT-proBNP levels, which were not included in PESI scoring, were associated with all-cause mortality. Plasma levels of natriuretic peptides reflect the severity of right ventricular dysfunction and hemodynamic impairment in acute PTE [20]. Post-discharge cardiac and hemodynamic follow-up of patients with elevated NT-proBNP levels may be useful. The presence of echocardiographic RV dysfunction or elevated natriuretic peptides in pulmonary embolism patients has been associated with short-term mortality without hemodynamic deterioration [16]. In a 183-case study on long-term mortality due to PTE, there was no significant difference between the Hb parameter and pulmonary embolism clinical risk groups [11]. The Hb level was not included in PTE risk scoring in the PESI validation study [4,5]. However, in the 30-day mortality predictor model of the large acute pulmonary embolism study, each unit change in Hb level was statistically significant [8]. Control of blood Hb levels during anticoagulant treatment of pulmonary embolism patients may be critical in those at risk of bleeding. In the regression analysis of the study, a 0.98-fold (95% CI: 0.98-0.99) significant reduction in mortality was found as the duration (days) of anticoagulant therapy was prolonged. All-cause mortality among PTE patients receiving prolonged treatment was also reduced. Similarly, another previous study showed a statistically significant decrease in mortality (p=0.026) in patients receiving heparin therapy on PTE days 31-90 [13].

In this study, no statistically significant difference was found in the mean hospitalization days of patients with sPESI scores of 0 and 1. Early discharge and outpatient treatment are recommended for patients with clinically and cardiopulmonary stable PESI or low sPESI scores [9,10]. However, as seen in our study, pulmonary embolism patients had a similar number of hospitalization days even though the PESI score was low. Physicians preferred to give anticoagulant treatment during hospitalization. Transportation to the hospital, social support, and difficulty in follow-up may have been effective in this decision.

Limitations

This is a single-center, retrospective, observational cohort study with all of the limitations inherent in this type of study. Given the retrospective study design, data were missing for some variables.

CONCLUSIONS

In conclusion, all-cause mortality in patients with pulmonary embolism increased with age, cardiac diseases, and COPD comorbidities. The PESI and sPESI scores used in the acute phase of PTE were found to be highly reliable in predicting all-cause mortality in PE patients. The diagnosis of massive PE and elevated NT-proBNP levels, a marker of right ventricular dysfunction, were factors that increased mortality. Patients with low PESI and sPESI scores were hospitalized as much as patients with high scores, and outpatient follow-up was not preferred.

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Conflict of interest: None to declare.

Ethical Approval: This study was approved by the Gaziantep University Medical Ethics Committee (No: S-2022-368). All procedures performed in studies involving human participants were in accordance with the ethical standards of the institutional and/or national research committee and with the 1964 Helsinki Declaration and its later amendments or comparable ethical standards.

Contribution of the Authors: FF and IVD designed the study; FF and IZB collected the data; FF and MT analyzed the data; FF searched the literature and wrote the manuscript; FF edited and revised the manuscript according to the journal's instructions; FF and MT edited and controlled the final version of the manuscript. All the authors approved the final version of the manuscript.

REFERENCES

[1] Arseven O, Bingöl Z, Öngen HG, Uzun O, Okumuş NG, Çöplü L, Erol S, Oğuzülgen İK, Önen ZP, Özgür ES, Sevinç C, Topçu F, Uçar EY, Yıldızeli B (2021) Türk Toraks Derneği Pulmoner Tromboembolizm Tanı Ve Tedavi Uzlaşı Raporu 2021. Optimus Yayıncılık. pp.1-124. https://toraks.org.tr/site/community/library/dwkHt8qc8GMSPZfT

[2] Konstantinides SV, Meyer G, Becattini C, Bueno H, Geersing GJ, V.-P. Harjola VP, Huisman MV, Humbert M, Jennings CS, Jiménez D, Kucher N, Lang IM, Lankeit M, Lorusso R, Mazzolai L, Meneveau N, Ní Áinle F, Prandoni P, Pruszczyk P, Righini M, Torbicki A, Van Belle E, Zamorano JL (2020) ESC Scientific Document Group, 2019 ESC Guidelines for the diagnosis and management of acute pulmonary embolism developed in collaboration with the European Respiratory Society (ERS). Eur Heart J. 41:543–603. https://doi.org/10.1093/eurheartj/ehz405.

[3] Laporte S, Mismetti P, Décousus H, Uresandi F, Otero R, Lobo JL, Monreal M, the RIETE Investigators (2008) Clinical Predictors for Fatal Pulmonary Embolism in 15 520 Patients With Venous Thromboembolism. Circulation. 117: 1711–6. https://doi.org/10.1161/CIRCULATIONAHA.107.726232.

[4] Wicki J, Perrier A, Perneger TV, Bounameaux H, Junod AF (2000) Predicting adverse outcome in patients with acute pulmonary embolism: a risk score. Thromb Haemost. 84: 548–52.

[5] Aujesky D, Obrosky DS, Stone RA, Auble TE, Perrier A, Cornuz J, Roy PM, Fine MJ (2005) Derivation and validation of a prognostic model for pulmonary embolism. Am J Respir Crit Care Med. 172: 1041–6. https://doi.org/10.1164/rccm.200506-862OC.

[6] Lankeit M, Gómez V, Wagner C, Aujesky D, Recio M, Briongos S, Moores CLK, Yusen RD, Konstantinides S, Jiménez D, Instituto Ramón y Cajal de Investigación Sanitaria Pulmonary Embolism Study Group (2012) A strategy combining imaging and laboratory biomarkers in comparison with a simplified clinical score for risk stratification of patients with acute pulmonary embolism. Chest. 141:916–22. https://doi.org/10.1378/chest.11-1355.

[7] Mastroiacovo D, Dentali F, di Micco P, Maestre A, Jiménez D, Soler S, Sahuquillo JC, Verhamme P, Fidalgo A, López-Sáez JB, Skride A, Monreal M, RIETE Investigators (2019) Coordinator of the RIETE Registry: Manuel Monreal (Spain). RIETE Steering Committee Members:, Members of the RIETE Group are as follows. Spain:, Rate and duration of hospitalisation for acute pulmonary embolism in the real-world clinical practice of different countries: analysis from the RIETE registry, Eur Respir J. 53:1801677. https://doi.org/10.1183/13993003.01677-2018.

[8] Jiménez D, Miguel-Díez J, Guijarro R, Trujillo-Santos J, Otero R, Barba R, Muriel A, Meyer G, Yusen RD, Monreal M, RIETE Investigators (2016) Trends in the Management and Outcomes of Acute Pulmonary Embolism: Analysis From the RIETE Registry, J Am Coll Cardiol. 67:162–170. https://doi.org/10.1016/j.jacc.2015.10.060.

[9] Aujesky D, Roy PM, Verschuren F, Righini M, Osterwalder J, Egloff M, Renaud B, Verhamme P, Stone RA, Legall C, Sanchez O, Pugh NA, N'gako A, Cornuz J, Hugli O, Beer HJ, Perrier A, Fine MJ, Yealy DM (2011) Outpatient versus inpatient treatment for patients with acute pulmonary embolism: an international, open-label, randomised, non-inferiority trial. Lancet. 378:41–48. https://doi.org/10.1016/S0140-6736(11)60824-6.

[10] Piran S, Le Gal G, Wells PS, Gandara E, Righini M, Rodger MA, Carrier M (2013) Outpatient treatment of symptomatic pulmonary embolism: a systematic review and meta-analysis. Thromb Res. 132: 515–9. https://doi.org/10.1016/j.thromres.2013.08.012.

[11] Gupta R, Ammari Z, Dasa O, Ruzieh M, Burlen JJ, Shunnar KM, Nguyen HT, Xie Y, Brewster P, Chen T, Aronow HD, Cooper CJ (2020) Long-term mortality after massive, submassive, and low-risk pulmonary embolism. Vasc Med. 25:141–149. https://doi.org/10.1177/1358863X19886374.

[12] Ng ACC, Chung T, A. Sze Chiang Yong A, Siu Ping Wong H, Chow V, Celermajer DS, Kritharides L (2011) Long-Term Cardiovascular and Noncardiovascular Mortality of 1023 Patients With Confirmed Acute Pulmonary Embolism, Circulation: Cardiovascular Quality and Outcomes. 4:122–8. https://doi.org/10.1161/CIRCOUTCOMES.110.958397.

[13] Sandal A, Korkmaz ET, Aksu F, Köksal D, Selçuk ZT, Demir AU, Emri S, Çöplü L (2021) Performance of pulmonary embolism severity index in predicting long-term mortality after acute pulmonary embolism. Anatol J Cardiol. 25:544–554. https://doi.org/10.5152/AnatolJCardiol.2021.99345.

[14] S. Barco B, Valerio L, Gallo A, Turatti G, Mahmoudpour SH, Ageno W, Castellucci LA, Cesarman-Maus G, Ddungu H, De Paula EV, Dumantepe M, Goldhaber SZ, Guillermo Esposito MC, Klok FA, Kucher N, McLintock C, Ní Áinle F, Simioni P, Spirk D, Spyropoulos AC, Urano T, Zhai ZG, Hunt BJ, Konstantinides SV (2021) Global reporting of pulmonary embolism-related deaths in the World Health

Organization mortality database: Vital registration data from 123 countries. Res Pract Thromb Haemost.5: e12520. https://doi.org/10.1002/rth2.12520.

[15] Gupta R, Fortman DD, Morgenstern DR, Cooper CJ (2018) Short- and Long-term Mortality Risk After Acute Pulmonary Embolism. Curr Cardiol Rep. 20:135. https://doi.org/10.1007/s11886-018-1084-6.

[16] Coutance G, Cauderlier E, Ehtisham J, Hamon M, Hamon M (2011) The prognostic value of markers of right ventricular dysfunction in pulmonary embolism: a meta-analysis. Crit Care. 15: R103. https://doi.org/10.1186/cc10119.

[17] Kasper W, Konstantinides S, Geibel A, Olschewski M, Heinrich F, Grosser KD, Rauber K, Iversen S, Redecker M, Kienast J (1997) Management strategies and determinants of outcome in acute major pulmonary embolism: results of a multicenter registry. J Am Coll Cardiol. 30: 1165–71. https://doi.org/10.1016/s0735-1097(97)00319-7.

[18] Kucher N, Rossi E, De Rosa M, Goldhaber SZ (2006) Massive pulmonary embolism. Circulation. 113 (2006) 577–82. https://doi.org/10.1161/CIRCULATIONAHA.105.592592.

[19] Konstantinides SV, Vicaut E, Danays T, Becattini C, Bertoletti L, Beyer-Westendorf J, Bouvaist H, Couturaud F, Dellas C, Duerschmied D, Empen K, Ferrari E, Galiè N, Jiménez D, Kostrubiec M, Kozak M, Kupatt C, Lang IM, Lankeit M, Meneveau N, Palazzini M, Pruszczyk P, Rugolotto M, Salvi A, Sanchez O, Schellong S, Sobkowicz B, Meyer G (2017) Impact of Thrombolytic Therapy on the Long-Term Outcome of Intermediate-Risk Pulmonary Embolism, J Am Coll Cardiol. 69:1536-44. https://doi.org/10.1016/j.jacc.2016.12.039.

[20] Henzler T, Roeger S, Meyer M, Schoepf UJ, Nance JW, Haghi D, Kaminski WE, Neumaier M, Schoenberg SO, Fink C (2012)Pulmonary embolism: CT signs and cardiac biomarkers for predicting right ventricular dysfunction, Eur Respir J. 39 919–926. https://doi.org/10.1183/09031936.00088711.

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