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Predictive value of clinical and laboratory parameters in differentiating hypertensive pulmonary edema and acute pulmonary embolism

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Abstract. – OBJECTIVE: Pulmonary embolism and acute pulmonary edema can often be confused. The aim of this study is to investigate the role of clinical and laboratory parameters in the differentiation of these two pathologies.

PATIENTS AND METHODS: Between March 2017 and December 2021, a total of 114 patients (51 patients with acute hypertensive pulmonary edema and 63 patients with pulmonary embolism) were included in the study. The medical history, hemodynamic findings, main echocardiographic data, and routine laboratory markers were recorded, retrospectively.

RESULTS: Coronary artery disease (CAD), chronic obstructive pulmonary disease (COPD), and recent operation histories were found as more common concomitant disorders in the pulmonary embolism group (p = 0.001, p = 0.011, p = 0.001, respectively). In addition, patients with pulmonary embolism had a higher heart rate (p = 0.001) and systolic pulmonary artery pressure (SPAP) (p = 0.001) compared to those with hypertensive pulmonary edema, while patients with hypertensive pulmonary edema had higher blood pressure (p = 0.001). While significantly low albumin levels (p = 0.001) were found among blood parameters in the pulmonary embolism group, D-Dimer, fibrinogen, troponin, alanine aminotransferase (ALT), aspartate aminotransferase (AST), lactate dehydrogenase (LDH), creatine kinase myocardial band (CK-MB), red blood cell distribution width (RDW), and creatinine values were found to be higher (p < 0.001). The most sensitive (95%) and specific (92%) clinical parameter was the SPAP with a 19.00 mmHg cut-off level. Additionally, the most sensitive (98%) and specific (97%) laboratory parameter was the D-Dimer, with a 260.5 ng/mL cut-off level.

CONCLUSIONS: Especially simple blood parameters such as D-dimer and echocardiographic evaluation of SPAP seem to be quite effective in distinguishing pulmonary embolism from hypertensive pulmonary edema.

Key Words:
Pulmonary embolism, Hypertensive pulmonary edema, Clinical findings, Laboratory parameters, SPAP, D-Dimer.

Introduction

Pulmonary pathologies are common reasons for applying to the emergency department for chest pain and shortness of breath[1]. In particular, hypertensive pulmonary edema resulting from end-organ damage caused by hypertensive crises and pulmonary embolism due to deep vein thrombosis are emergencies that may present with similar symptoms[1-3].

Pulmonary edema, which constitutes an important part of hypertensive emergencies (39%), is mostly managed with a medication based on blood pressure regulation and diuretics, in which venodilators such as nitroglycerin are included in the treatment[4]. Pulmonary embolism is an emergency pathology that may require more aggressive thrombosis management and more advanced treatments such as mechanical ventilation, which may require the use of invasive methods[5]. In hypertensive crisis, the pathophysiology may trigger myocardial infarction, which can lead to diastolic dysfunction, as well as symptoms that occur as a result of increased adrenergic system activation and related increased blood pressure and afterload[6]. However, considering that it may have not only pulmonary but also other systemic effects, multisystemic action is necessary[6,7]. Indeed, pulmonary embolism is also a source of pulmonary edema in its own right, and depending on the severity of the embolism, it is quite possible to be confused with other conditions that cause pulmonary edema[7]. In emer-

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gency clinics, direct chest radiography is insufficient for diagnosis, and advanced techniques such as echocardiography, contrast-enhanced tomography or magnetic resonance imaging are very useful in differential diagnosis. However, the importance of standard laboratory tests is great. They are fast and inexpensive solutions, especially in the differentiation of cardiogenic and non-cardiogenic causes[8]. However, there is no comprehensive data on the role of standard laboratory tests in the differentiation of hypertensive pulmonary edema and pulmonary embolism.

This study aimed to investigate the predictive value of standard blood tests in patients with hypertensive pulmonary edema and pulmonary embolism. The sensitivity and specificity of blood tests to the disease were evaluated by recording the standard laboratory parameters and demographic data of the patients diagnosed with these diseases after presenting to the emergency department.

Patients and Methods

Study Participants

The study was conducted among patients who were diagnosed with hypertensive pulmonary edema and pulmonary embolism, who between March 2017 and December 2021 applied to the emergency department with the complaint of shortness of breath. The data of all patients were scanned retrospectively, and the study data were recorded. All steps of the study were planned in accordance with the Declaration of Helsinki Ethics Principles and ethics approval was obtained from the Local Ethics Committee.

A total of 114 patients over the age of 18 years old who presented to the emergency department and were diagnosed with hypertensive pulmonary edema or pulmonary embolism were included in the study. Patients with known genetic pathology, patients with pulmonary embolism under the age of 18, COVID-19 (+) patients, patients with myocardial infarction, patients with a history of recurrent thrombus and taking anticoagulant therapy, cancer patients, patients with a history of immunosuppressive use, and patients with a diagnosis of congestive heart failure were excluded from the study as they may affect the results.

Patients

While 51 of the patients included in the study developed pulmonary edema after hypertensive crisis, 63 patients were diagnosed with pulmonary embolism by tomography. After the demographic data of the patients were recorded, heart rhythm rates, blood pressure values, left ventricular ejection fraction and systolic pulmonary artery pressure (SPAP) were evaluated at the time of first admission to the emergency department. In addition, the standard blood parameters that were studied at the time of the patients' first admission to the emergency department were also evaluated. All data were statistically compared between hypertensive pulmonary edema and pulmonary embolism groups and their significance was examined.

Blood Parameters

Complete blood count parameters, including hemoglobin (Hgb, ref. 11.9-14.6 g/dL), main corpuscular volume (MCV, ref. 82.9-98.0 fL), main platelet volume (MPV, ref. 9.1-11.9 fL), red cell distribution width (RDW, ref. 39.2-49.2 fL), and platelet distribution width (PDW, ref. 9.9-15.4 fL) were recorded. Biochemically, creatinine (ref. 05-1.4 mg/dL), alanine aminotransferase (ALT, ref. 0-55 U/L), aspartate aminotransferase (AST, ref. 5-34 IU/L), lactate dehydrogenase (LDH, ref. 100-250 U/L), creatine kinase myocardial band (CK-MB, ref. 0-4.9 ng/mL), Troponin T (ref. 0-0.04 ng/mL), albumin (ref. 3.4-5.4 g/dL), fibrinogen (ref. 167-399 mg/dL), D-Dimer (ref. 0-500 ng/mL) were evaluated.

Statistical Analysis

In this study, the Chi-Square Test was used to compare the discontinuous demographic data according to the groups. Normal distribution analysis of continuous data was performed using 5 parameters (skewness-kurtosis, Histogram, MEAN/STD, Q-Q plots, Shapiro-Wilk Test). Normally distributed continuous data were shown as Mean±SEM (standard error mean), and Independent Samples *t*-test was used for comparisons between pairs. Continuous data that were not normally distributed were shown as Median (Minimum-Maximum), and comparisons between pairs were made with Mann-Whitney U Test. Cut-off values were created for specificity, sensitivity calculation and regression analysis using the Receiver Operating Characteristic (ROC) curve method. The data categorized by the created cut-off values were used for the estimation of pulmonary embolism by performing Multiple Binary Logistic Analysis. Statistical analysis of this study was performed by using the SPSS v. 23.0, (IBM Corp., Armonk, NY, USA) software program. $p < 0.05$ was considered statistically significant.

Results

When the demographic data of the patients were compared, a significant correlation was found between the frequency of pulmonary embolism patients and a history of previous coronary artery stenosis, a history of chronic obstructive pulmonary disease, a history of immobilization, a recent operation, and, as expected, and a history of deep vein thrombosis (DVT) ($p < 0.05$). Comparison of demographic data between two groups is summarized in Table I.

While no significant difference was observed between the groups in terms of gender, it was determined that patients with pulmonary embolism were in the older age group (Table II, $p = 0.000$). When evaluated in terms of clinical parameters, the difference between heart rate, systolic blood pressure, and diastolic blood pressure was significant ($p < 0.01$). In addition, echocardiographic ejection fraction and systolic pulmonary artery pressure values were significantly different ($p < 0.01$). Blood values were compared between the groups. There was a significant difference between albumin, hemoglobin, MCV, RDW, PDW, D-Dimer, creatinine, ALT, AST, LDH, CK-MB, Troponin, Fibrinogen values ($p < 0.05$). Comparison of parameters between groups is shown in Table II.

Increased D-Dimer, SPAP, Troponin, and Fibrinogen values have been observed in patients with pulmonary embolism, and this increase is both statistically and clinically significant ($p < 0.01$). Albumin was found to be statistically significantly low. In particular, serum albumin, fibrinogen and D-Dimer levels were more than 90% sensitive to pulmonary embolism, while D-Dimer was the most specific biomarker (96%). As an echocardiographic finding, SPAP was found to be a very effective parameter with 95% sensitivity and 92% specificity. The sensitivity and specificity of markers on ROC curve analysis were presented in Figure 1 and Table III.

We studied abnormal Troponin, albumin and Fibrinogen parameters with a binary logistic regression model. Nagelkerke's R^2 determination coefficient was used to evaluate the predictive ability of the model. This model predicts of hypertensive pulmonary edema 87.5% and pulmonary embolism 94.1% correctly (Table IV). The Nagelkerke's

Table I. Analysis of demographic data by groups.

		Pulmonary Edema	Pulmonary Embolism	Chi-Square Test
Hypertension				
	-	30 (26.3%)	38 (33.3%)	Value: 0.000 (Yates)
	+	21 (18.4%)	25 (21.9%)	p = 01.000
Diabetes Mellitus				
	-	36 (31.6%)	44 (38.6%)	Value: 0.000 (Yates)
	+	15 (13.2%)	19 (16.7%)	p = 0.931
CAD				
	-	51 (45.1%)	51 (45.1%)	Fisher's Exact test
	+	0 (0.00%)	11 (9.7%)	p = 0.001
COPD				
	-	50 (43.9%)	51 (44.7%)	Value: 6.54 (Yates)
	+	1 (0.9%)	12 (10.5%)	p = 0.011
Immobilization				
	-	51 (44.7%)	49 (43.0%)	Value: 10.939 (Yates)
	+	0 (0.00%)	14 (12.3%)	p = 0.001
Previous PE				
	-	51 (44.7%)	60 (52.6%)	Fisher's Exact test
	+	0 (0.0%)	3 (2.6%)	p = 0.252
Previous DVT				
	-	51 (44.7%)	51 (54.4%)	Fisher's Exact test
	+	0 (0.00%)	11 (0.90%)	p = 0.001

CAD: Coronary artery disease, COPD: Chronic obstructive pulmonary disease, PE: Pulmonary embolism, DVT: Deep venous thrombosis. The frequency analysis was made by using Chi-square Test. Fisher's exact test: Minimum expected value < 5.00; Yates Chi-square Test: 5.00 ≤ minimum expected value < 25; Pearson's Chi-square test: 25 ≤ minimum expected value.

Table II. Comparison of blood and clinical parameters between groups.

	Pulmonary Edema	Pulmonary Embolism	Sig. (*p*)
Age***	51.03 ± 1.60	60.66 ± 2.04	0.000
Gender (m/%)	23/45%	27/42%	0.614
Heart Rate***	68.84 ± 11.32	114.27 ± 2.34	0.000
SBP**	180 (210-166)	110 (70-154)	0.000
DBP**	90 (80-110)	65 (30-113)	0.000
Albumin***	3.71 ± 0.06	2.85 ± 0.07	0.000
Hbg**	13.90 ± 0.20	13.03 ± 0.24	0.007
MCV*	84.10 (32.80-98.30)	87.70 (60.80-102.90)	0.027
RDW***	13.40 (11.70-18.60)	15.50 (11.90-23.10)	0.000
MPV	8.20 (6.40-10.20)	8.50 (6.70-14.50)	0.105
PDW***	16.30 (15.50-19.10)	16.60 (15.40-19.30)	0.000
EF***	61.25 ± 0.50	54.61 ± 1.29	0.000
SPAP***	9 (0-25)	45 (15-100)	0.000
D-Dimer***	100 (36-281)	2,391 (235-29,420)	0.000
Creatinine*	0.90 (0.60-1.90)	1.00 (0.50-7.70)	0.031
ALT***	19.50 (8.00-79.00)	37.00 (9.00-1,293.00)	0.000
AST***	24.00 (15.00-122.00)	33.00 (12.00-1,287.00)	0.000
LDH***	176.00 (11.00-300.00)	264.00 (155.00-4072)	0.000
CKMB**	11.00 (4.00-20.00)	16.00 (2.00-75.00)	0.001
Troponin***	0.010 (0.00-0.040)	0.050 (0.00-2.40)	0.000
Fibrinogen***	258 (70-445)	374 (225-844)	0.000

SBP: Systolic blood pressure, DBP: Diastolic blood pressure, Hbg: Hemoglobin, MCV: Main corpuscular volume, RDW: Red cell distribution width, MPV: Main platelet volume, PDW: Platelet distribution volume, EF: Ejection fraction, SPAP: Systolic pulmonary artery pressure, ALT: Alanine aminotransferase, AST: Aspartate amino transferase, LDH: lactate dehydrogenase, CKMB: Creatine kinase myocardial band. Parametric data were presented as Mean±Standard error (SEM). Pairwise comparison tests were performed with the Independent Samples *t*-test. Nonparametric data were shown as Median (Minimum-Maximum) and pairwise comparisons were made with Mann-Whitney U Test. *The difference between groups is statistically significant at the 95% confidence interval ($p < 0.05$). **The difference between groups is statistically significant at 99% confidence interval ($p < 0.01$). ***The difference between groups was statistically significant at the 99.9% confidence interval ($p < 0.001$).

R^2 value indicates the existence of a relationship of 84.3% (Table IV). Additionally abnormal D-Dimer and SPAP parameters were evaluated with binary logistic regression model. Nagelkerke's R^2 determination coefficient was used to evaluate the predictive ability of the model. This model can correctly predict hypertensive pulmonary edema (96.8%) and pulmonary embolism (98.1%) (Table V). The Nagelkerke's R^2 value indicates the existence of a relationship (95.6%) (Table V).

Discussion

As far as we know, there are not enough studies that comprehensively examine the clinical and blood parameters in the differentiation of patients presenting to the emergency department with hypertensive pulmonary edema and pulmonary embolism. Our findings showed that coronary artery disease (CAD), chronic obstructive pulmonary disease (COPD), and recent operation history were more common in the pulmonary embolism group. In addition, patients with pulmonary embolism had a higher heart rate compared to those with hypertensive pulmonary edema, while patients with hypertensive pulmonary edema had higher blood pressure. While significantly low albumin levels were found among blood parameters in the pulmonary embolism group, ALT, AST, LDH, CK-MB, RDW, and creatinine values were high. It was determined that increased SPAP and D-Dimer levels could accurately predict the diagnosis of pulmonary embolism at a rate of 98.1%. Troponin, fibrinogen and albumin values from routine blood tests were found to have a predictive value with an accuracy rate of 90.9% in the diagnosis of pulmonary embolism.

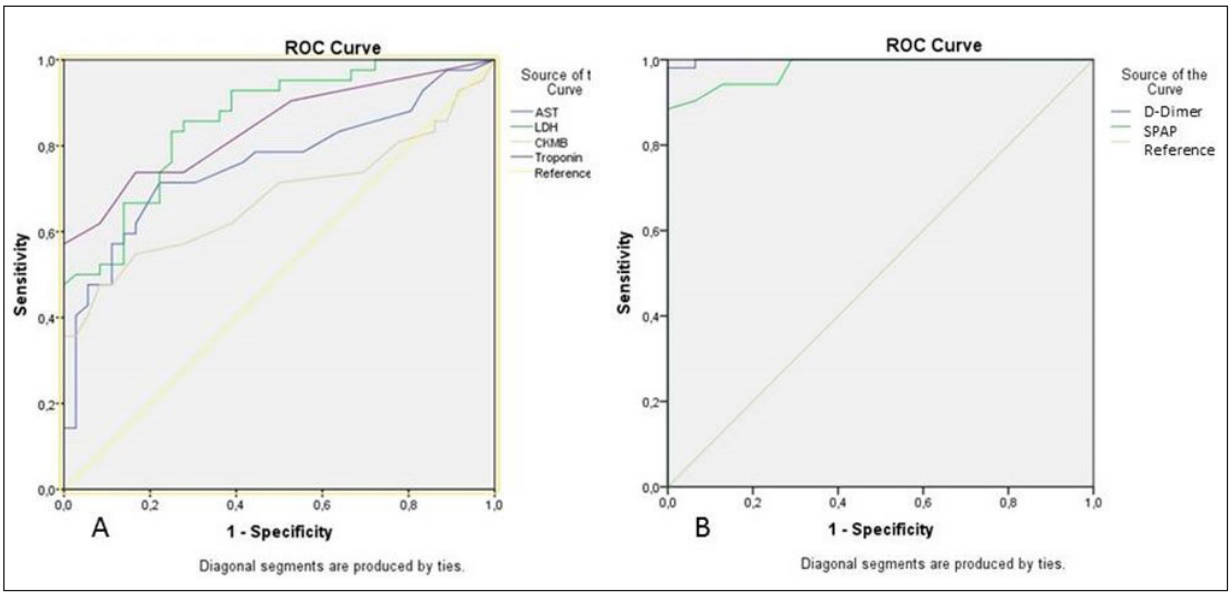

Figure 1. ROC Curve analyses of parameters. **A**, Sensitivity 71.4%, and specificity 77.8% for AST; Sensitivity 85.7%, and specificity 72.2% for LDH; Sensitivity 73.8%, and specificity 83.3% for Troponin. **B**, Sensitivity 98.2%, and specificity 96.8% for D-Dimer (p = 0.000; AUC: 0.999; 95% CI: 0.996-1.00); Sensitivity 94.9%, and specificity 92.2% for SPAP (p = 0.000; AUC: 0.988; 95% CI: 0.974-1.000).

Pulmonary embolism is usually a mortal pathology caused by underlying deep vein thrombosis. In particular, immobilization, major orthopedic surgery, malignancy and genetic predisposition have been shown to be among the main risk factors[9]. A previous history of venous thrombosis and embolism also increases the risk. When presenting to the emergency department, prompt diagnosis and treatment are the most important determinants to reduce mortality. Although computed tomographic angiography with contrast is important in the definitive diagnosis, blood parameters such as D-Dimer and fibrinogen are very valuable in determining the severity of the disease and the preliminary diagnosis[9,10]. The patients who have chest pain and shortness of breath when they apply to the emergency department should be quickly differentiated from cardiac pathologies and other causes of chest pain. Hypertensive pulmonary edema, especially affecting the pulmonary bed, is another pathology that should be differentiated, and it has a clinically similar appearance to pulmonary embolism[11]. Although the age range varies, hypertensive pulmonary edema has been reported more frequently in patients over 60 years of age in the literature, similar to the increased risk of pulmonary embolism with advanced age[10-12]. In our study, while CAD, COPD, immobilization, previous venous thromboembolism, and history of recent operation

Table III. ROC (Receiver Operating Characteristic) analysis of variables.

Risk factor	AUC (% 95)	Cut-off	p	Sensitivity	Specificity
AST	0.759 (0.651-0.868)	27.50	0.000	0.714	0.778
LDH	0.860 (0.780-0.939	210.50	0.000	0.857	0.722
Troponin	0.843 (0.757-0.930)	0.025	0.000	0.738	0.833
Albumin	0.921 (0.864-0.977)	3.25	0.000	0.731	0.939
Fibrinogen	0.818 (0.723-0.913)	366.00	0.000	0.923	0.667
D-Dimer	0.999 (0.996-1.00)	260.5	0.000	0.982	0.968
SPAP	0.988 (0.974-1.000)	19.00	0.000	0.949	0.922

AST: Aspartate amino transferase, LDH: lactate dehydrogenase, SPAP: Systolic pulmonary artery pressure, AUC: area under curve, LDH: Lactate dehydrogenase, SPAP: Systolic pulmonary artery pressure.

Table IV. Multiple Binary Logistic Regressions (Troponin, Albumin, Fibrinogen).

	B	S.E.	Wald	df	Sig.	Exp(B)	95% C.I.for EXP(B)	
							Lower	Upper
Troponin Normal	2.022	1.035	3.818	1	0.051	7.557	0.994	57.460
Albumin Normal	21.301	5815.797	0.000	1	0.997	1781579005	0.000	-
Fibrinogen Normal	20.864	5815.798	0.000	1	0.997	961663815.9	0.000	-
Constant	-0.251	0.77	0.188	1	0.664	0.778		

a. Variable(s) entered on step 1: Troponin, Albumin, Fibrinogen

Model Summary

Step	-2 log likelihood[a]	Cox & Snell R Square	Nagelkerge R Square	Step	
1	25.409	0.632	0.843	1	a.

The Classification Table

	Observed		Predicted Groups		Percentage Correct
			Pulmonary Edema	Pulmonary Embolism	
Step 1	**Groups**	**Pulmonary Edema**	87.5%	12.5%	87.5%
		Pulmonary Embolism	5.9%	94.1%	94.1%
Overall percentage					90.9%

a. The cut value is 0.500. **B** - This is the coefficient for the constant (also called the "intercept") in the model. **S.E.** - This is the standard error around the coefficient for the constant **Wald and Sig.** - This is the Wald chi-square test that tests the null hypothesis that the constant equals 0. This hypothesis is rejected because the *p*-value (listed in the column called "Sig.") is smaller than the critical *p*-value of .05 (or .01). Hence, we conclude that the constant is not 0. df - This is the degrees of freedom for the Wald chi-square test. There is only one degree of freedom because there is only one predictor in the model, namely the constant. **Exp(B)** - This is the exponentiation of the B coefficient, wh≤ch is an odds ratio. This value is given by default because odds ratios can be easier to interpret than the coefficient, which is in log-odds units.

were higher in the pulmonary embolism group, as expected, it was determined that the pulmonary embolism group was in the older age group. We thought that the predisposing factors might increase with age. However, the acute hypertensive pulmonary edema group also had a mean above middle age. Although the patient group was reported to be older in more hypertensive pulmonary edema studies in the literature, the younger age of our patients suggested that there may be regional differences.

Cardiac hemodynamics is affected in both diseases[10-12]. The increased SPAP values were reported in pulmonary embolism[10]. SPAP has even been shown as a parameter to measure the success of treatment in these patients[13]. Significant increases in systolic and diastolic blood pressure were found in hypertensive edema, and heart rate was reported in a variable range. In pulmonary embolism, increased heart rates have been detected even in normotensive condition, and it has been suggested that it may be associated with mortality[12,14]. There is no comprehensive study on the SPAP interval in acute hypertensive pulmonary edema. Most of the studies[12,15] on hypertensive edema focused on systolic and diastolic blood pressure and heart rate parameters, while SPAP was given within normal ranges. Another echocardiographic measurement parameter is left ventricular ejection fraction (EF). Left ventricular functions are less affected in both hypertensive edema and pulmonary embolism, and EF values are generally normal ranges if there is no underlying heart failure[11,12,16]. In our patient group, patients with acute hypertensive pulmonary edema had significantly higher systolic and diastolic

Table V. Multiple Binary Logistic Regressions (D-Dimer, SPAP).

		B	S.E.	Wald	df	Sig.	Exp(B)	95% C.I.for EXP(B)	
								Lower	Upper
D-Dimer	Abnormal	22.495	4667,765	0.000	1	0.996	5882767973	0.000	-
SPAP	Normal	20.010	4667,765	0.000	1	0.997	490230664	0.000	-
Constant		-0.144	0.804	0.032	1	0.858	0.778		

a. Variable(s) entered on step 1: D-Dimer, SPAP: Systolic pulmonary artery pressure

Model Summary

Step	-2 log likelihood[a]	Cox & Snell R Square	Nagelkerge R Square	Step	
1	9.503	0.701	0.956	1	a.

The Classification Table

			Predicted		
	Observed		Groups		Percentage Correct
			Pulmonary Edema	Pulmonary Embolism	
Step 1	Groups	Pulmonary Edema	96.8%	3.2%	96.8%
		Pulmonary Embolism	1.9%	98.1%	98.1%
Overall percentage					97.6%

a. The cut value is 0.500. **B** - This is the coefficient for the constant (also called the "intercept") in the model. **S.E.** - This is the standard error around the coefficient for the constant **Wald and Sig.** - This is the Wald chi-square test that tests the null hypothesis that the constant equals 0. This hypothesis is rejected because the *p*-value (listed in the column called "Sig.") is smaller than the critical *p*-value of .05 (or .01). Hence, we conclude that the constant is not 0. df - This is the degrees of freedom for the Wald chi-square test. There is only one degree of freedom because there is only one predictor in the model, namely the constant. **Exp(B)** - This is the exponentiation of the B coefficient, which is an odds ratio. This value is given by default because odds ratios can be easier to interpret than the coefficient, which is in log-odds units.

blood pressures, while heart rates were higher in the pulmonary embolism group. By the way, SPAP was found to be a basic parameter in differentiating pulmonary embolism from hypertensive edema (95% sensitivity and 92% specificity). Although EF values were different between the two groups, they were within the normal range.

Although there is no directly defined blood parameter in the diagnosis of hypertensive pulmonary embolism, tests such as brain natriuretic peptide (BNP), troponin, and creatinine have been investigated and no significant increase has been shown[17]. Despite blood tests are not included in the diagnosis of pulmonary edema, they have been evaluated in hypertensive crisis due to increased blood pressure, and it has been emphasized that these patients may have higher creatinine levels, that especially affect kidney functions[18]. In pulmonary embolism, blood tests were evaluated more intensively in diagnosis and treatment, and parameters such as D-Dimer, fibrinogen, and troponin were emphasized[19,20]. Although the specificity of D-Dimer varies, it is still shown[21] as one of the most valuable parameters in pulmonary embolism with its high sensitivity rates. Fibrinogen has been presented as another important parameter like D-Dimer, but it has been reported[21] that it is not specific and will vary in other thrombotic events. Various ratios have been studied to increase the specificity of these two parameters[22]. Troponin, which is basically a marker of myocardial ischemia, has also been investigated[23] in pulmonary embolism and it has been suggested that it will rise due to cardiac effects of the pulmonary bed. However, recently, it has been emphasized that inflammatory parameters will increase the specificity and sensitivity of these previous parameters and should be evaluated together[23]. In recent publications[24-26], it has been shown that serum albumin levels are both an acute phase reactant and a determinant of oncotic pressure, especially in vascular events. It has

been shown[24] that especially the albumin levels decrease in venous events and the predictive value of fibrinogen increases. In addition, the role of albumin in thrombotic events has been emphasized and it has been claimed to have predictive effectiveness[25]. It has even been demonstrated[26] that lower albumin levels may be predictive in asymptomatic pulmonary embolism, even if D-Dimer and fibrinogen levels remain normal. Our results supported that D-Dimer, fibrinogen, and troponin levels are higher in patients with pulmonary embolism compared with acute hypertensive pulmonary edema. In particular, D-Dimer was found to be the most valuable parameter in the differentiation of two clinical conditions (cut-off value: 260.5 ng/mL, 98% sensitivity, and 96% specificity). In addition, in our study, albumin levels were found to be lower in embolism patients, which is consistent with previous studies[26].

The main limitation of the study is its retrospective nature. Retrospective recording of data can cause data deficiency and errors. Furthermore, comprehensive data that present information for right ventricle function, past medications, and additional conditions are lacking for each group comparison. Another limitation is related to the interpretation of data based on single-center and single-site analysis. It will be possible to obtain more comprehensive and consistent results with the data of different regions and different centers.

Conclusions

In conclusion, clinical and laboratory parameters seem to be effective in the differential diagnosis of patients with hypertensive pulmonary edema and pulmonary embolism who applied to the emergency department with similar complaints. In particular, our results support that D-Dimer, fibrinogen, albumin, and troponin levels can be used as a laboratory test to strengthen the preliminary diagnosis and echocardiographic SPAP can be useful in cases where tomographic evaluation cannot be performed or until the process of tomography.

Conflict of Interest
There is no conflict of interest.

Funding
None.

Ethics Approval
Ethics approval was obtained from Mustafa Kemal University Local Ethics Committee (Date: 30/06/2022, Approval No.: 08/19).

Informed Consent
The authors declare that the patients included in the study signed informed consent forms to use their medical information in the studies.

Authors' Contributions
Concept: Barış Kavvasoglu; Design: Barış Kavvasoglu; Supervision: Barış Kavvasoglu, Gamze Hande Kavvasoglu; Funding: Barış Kavvasoglu; Materials: Barış Kavvasoglu, Gamze Hande Kavvasoglu; Data: Barış Kavvasoglu, Gamze Hande Kavvasoglu; Analysis: Barış Kavvasoglu, Gamze Hande Kavvasoglu; Literature search: Barış Kavvasoglu, Gamze Hande Kavvasoglu; Writing: Barış Kavvasoglu, Gamze Hande Kavvasoglu; Critical revision: Barış Kavvasoglu, Gamze Hande Kavvasoglu.

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