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Evaluation of patients who underwent computed tomography angiography with the pre-diagnosis of pulmonary embolism

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Abstract

Aim: Pulmonary embolism (PE) is a disease that develops as a result of occlusion of the pulmonary artery or its divisions by thrombus deriving from the systemic deep venous system. PE ranks third among deaths caused by cardiovascular disease. PE is a preventable illness with elevated mortality and morbidity, repetitive and hard to diagnose. In this study, computed tomography angiography (CTA) images and blood parameters of patients who underwent CTA with a prediagnosis of PE in Adıyaman Training and Research Hospital between 01/03/2020-01/09/2022 were examined.

Materials and Methods: Patients who underwent CTA with suspected PE were analyzed, and a comparison was made between the sociodemographic and laboratory data of those diagnosed with PE and those who were not.

Results: The mean age of PE patients was importantly higher than non-PE patients, and there was no statistical variance in the gender distribution of the groups. Monocyte count was markedly higher in the PE group, and the CAR value remarkably higher in the non-PE group than in the PE group. According to the ROC analysis, the AUC value was the highest when the D-dimer and troponin I value were considered together. Two different cut-offs were found for D-dimer. When D-dimer was 655 mg/dL, the specificity was 96.5%, while when D-dimer was 438.5 mg/dL, the sensitivity was 92.2%. The highest specificity was found with troponin I (97.4%). The likelihood ratio (LR) was the highest, at 26.08, when D-dimer was 655 mg/dL and above.

Conclusion: As a result, the main message of our study is that when making differential diagnosis in patients who apply to the emergency department with dyspnea and chest pain, better diagnostic accuracy can be achieved with the combined evaluation of tomographic findings and laboratory findings. Therefore, evaluation of laboratory values such as D-dimer, hemogram parameters and troponin I is important in diagnosis and risk classification.

Introduction

Pulmonary embolism (PE) is a notable and possibly mortal medical ailment caused by a blood clot forming in the pulmonary arteries. This can obstruct blood flow and damage lung tissue, leading to a range of symptoms and complications. The mean yearly occurrence of PE is around 75-269/100.000, and the disease's occurrence rises with increasing age [1].

Deep vein thrombosis (DVT) typically occurs when a blood clot emerges in one of the deep veins in the body, most often in the lower extremities. From there, the blood clot can travel through the bloodstream to the lungs, where it can lodge in the pulmonary arteries and cause a blockage. This situation may lead to PE [2].

Several hazardous conditions can elevate the likelihood of developing a blood clot and, therefore, increase the risk of PE. These include a record of DVT or PE, prolonged periods of inactivity, such as bed rest or long flights, cancer or chemotherapy treatment, surgery, particularly orthopedic surgery or abdominal surgery, pregnancy or recent childbirth, hormone replacement therapy or oral contraceptives, obesity, smoking, inherited blood clotting disorders [3].

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The symptoms of PE can vary depending on the largeness of the blood clot and the size of the blockage in the lungs. Some of the most frequent features include shortness of breath, especially during exertion, chest pain or discomfort, which may be sharp or stabbing and worsen with deep breathing or coughing, tachycardia, cough, which may produce blood-streaked sputum, dizziness or lightheadedness, fainting, swelling in the legs [4].

If PE is suspected, a doctor will typically execute a couple of tests to confirm the diagnosis. These may comprise laboratory tests to measure levels of a substance called D-dimer, which can indicate the presence of a blood clot, imaging tests including a chest X-ray, computed tomography angiography (CTA), or ultrasound, and pulmonary function tests to assess lung function [5].

The specificity of symptoms and clinical findings in PE is low. PE should be contemplated for differential diagnosis in patients aged 65 years and older who are admitted with dyspnea and chest pain. Diagnosis of PE is difficult because patients present to the emergency department with non-specific complaints such as shortness of breath, chest pain, etc. Although the diagnosis of PE can be successfully excluded in a certain group of patients with clinical evaluation and laboratory findings, pulmonary CTA remains the gold standard in this regard [6].

In this study, patients with a prediagnosis of PE, who underwent CTA, and whose hemogram and biochemistry analyses were requested, were retrospectively examined. Accordingly, the predictive properties of hemogram and biochemistry results in diagnosing PE in people with and without PE were examined. The data obtained from our study will be a source for future studies and can be used in the analysis of possible risk factors that may cause suspected PE.

Materials and Methods

Study design and Study group

For this research, permission was granted from the Adıyaman University Non-Interventional Clinical Research Ethics Committee with the decision dated 25/10/2022 and numbered 2022/7-54. A written informed consent form was taken from all participants to be included in the study, and they were informed that participation was voluntary and they could be free to withdraw from the research. In this study, CTA images and blood parameters of patients who underwent CTA with a prediagnosis of PE in Adıyaman Training and Research Hospital between 01/03/2020-01/09/2022 were examined. The sample size was calculated as 45 for each group using G*Power (3.1 Version, Dusseldorf, Germany) (The power of test: 0.8, alpha significance level: 0.05, Cohen's d effect size: 0.58). This study has a retrospective, observational, and cross-sectional structure. We examined the hemogram and biochemical values of patients who underwent CTA with the prediagnosis of PE and investigated the sensitivity and specificity of these parameters in the detection of PE.

Laboratory examination

White blood cell (WBC), neutrophil, lymphocyte, monocyte, basophil, eosinophil, and platelet counts in hemogram were evaluated in PE patients. In addition, hemoglobin, mean platelet volume (MPV), plateletcrit (PCT), and platelet distribution width (PDW) were evaluated in the hemogram. C-reactive protein (CRP), albumin, and uric acid were examined in biochemistry tests. Troponin I level, one of the cardiac enzymes, was evaluated. D-dimer, which is a valuable parameter in the differential diagnosis of PE, was examined.

Radiological examination

The diagnosis was made by evaluating the CTA of all patients with suspected PE. CTA findings of PE may be as follows [7]:

1. Filling Defects: CT angiography may reveal intraluminal filling defects in the pulmonary arteries. These defects represent areas where blood flow is obstructed due to the presence of blood clots.
2. Vascular Cutoff Sign: The CT scan may show abrupt termination or cutoff of a pulmonary artery due to an embolus, indicating that a clot is blocking the blood flow downstream.
3. Mosaic Perfusion: In some cases, there may be areas of uneven lung perfusion, where certain lung segments receive less blood flow due to the obstruction caused by the embolus.
4. Enlarged Right Ventricle: The right ventricle of the heart may appear enlarged on the CT scan due to increased pressure caused by the pulmonary embolism.
5. Pulmonary Infarction: In severe cases, CT angiography might reveal areas of lung tissue that have undergone infarction due to the lack of blood supply caused by the embolism.

Figure 1 shows the image of PE in CTA.

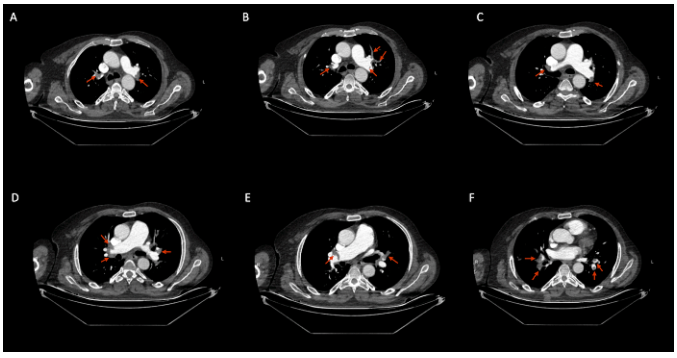

Figure 1. Massive pulmonary embolism in a 62-year-old man (In a 62-year-old male patient, filling defects consistent with pulmonary embolism in the right pulmonary artery, left pulmonary artery, lobar and segmental branches in axial sections of computed tomography angiography are shown with arrows).

Statistical analysis

Statistical analysis was carried out in IBM SPSS 26.0 Mac Version (IBM Corp., Armonk, NY, USA). Categorical values are shown as the number (percentage). Since the

gender data in the study is categorical, the Pearson chi-square test was used to compare the groups. Kolmogorov-Smirnov test was used to determine whether the age and laboratory values were normally distributed. The Mann–Whitney U test was used to compare the median values of the age and laboratory data that did not conform to the normal distribution between the PE and non-PE groups. Continuous data that did not conform to the normal distribution were shown with median, interquartile ranges, and confidence intervals. Receiver operating characteristics (ROC) analysis was utilized to evaluate the diagnostic sensitivity and specificity of the laboratory parameters in PE. In ROC analysis, area under the curve (AUC), 95% confidence interval (CI), cut-off, sensitivity, specificity, and likelihood ratio values were measured. The significance threshold was rated as $p<.05$ in statistical analyses.

Results

The comparison of the mean age and gender distribution of patients with suspected PE is shown in Table 1. The mean age of PE patients was significantly higher than non-PE patients, and there was no significant difference in the gender distribution of the groups. Comparison of laboratory parameters of patients with suspected PE is shown in Table 2, comparison of inflammatory rates is shown in Table 3. Monocyte count was statistically higher in the PE group, and the CAR value was statistically higher in the non-PE group than in the PE group.

PE patients were examined by separating them according to unilateral and bilateral pulmonary vascular involvement. Accordingly, 29 PE patients had unilateral and 22 PE patients had bilateral pulmonary vascular involvement. The sociodemographic data of those with unilateral and bilateral pulmonary vascular involvement are compared in Table 4, laboratory values in Table 5, and inflammatory rates in Table 6. Accordingly, D-dimer and troponin I levels were found to be significantly higher in those with bilateral involvement ($p=0.022$, $p<0.001$).

ROC analysis of the parameters is shown in Table 7 and Figure 2. Following the ROC analysis, the AUC value was the highest when the D-dimer and troponin I values were considered together. Two different cut-offs were found for D-dimer. When D-dimer was 655 mg/dL, the specificity was 96.5%, while when D-dimer was 438.5 mg/dL, the sensitivity was 92.2%. The highest specificity was found with troponin I (97.4%). The likelihood ratio (LR) was the highest, at 26.08, when the D-dimer was 655 mg/dL and above.

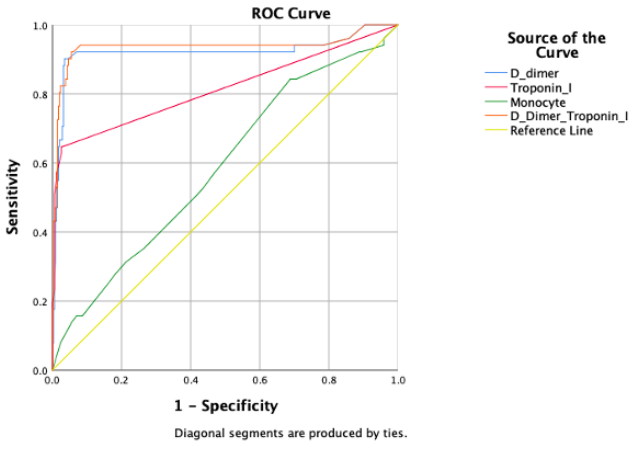

Figure 2. ROC curve of Lab values.

Discussion

PE is an illness with an elevated chance of mortality and confronts doctors in patient guidance in emergency units and inpatient services. There exist many researches on parameters predicting mortality in PE and research is up to this time in progress. Early diagnosis of this disease is key to predicting death. Laboratory parameters for example troponin I, brain natriuretic peptide (BNP), myoglobin, and WBC count are utilized as predictive measures in acute PE. The frequency of pulmonary embolism rises with growing age. Research has demonstrated that the mean age is 50 years or older, and the chance of VTE rises with growing age. In the present study, the mean age of the pulmonary embolism group was 73, which was consistent with the literature [8].

D-dimer is a laboratory parameter that quantifies the level of a protein particle made when blood clots are broken down. It is often used as a viewing marker for the detection of PE, as increased levels of D-dimer are commonly noted in patients with this condition [9]. However, the diagnostic sensitivity of D-dimer in PE is not 100%, meaning that some patients with PE may have regular D-dimer levels [10]. The sensitivity of D-dimer in PE can differ based on the patient population being studied and the cutoff value used to determine elevated levels. In general, studies have shown that D-dimer has a sensitivity of around 95% in patients with suspected PE when using a conventional cutoff value (usually 500 ng/mL or higher) [11]. However, several factors can affect the sensitivity of D-dimer in PE. For example, patients with a low pretest probability of PE (i.e., those who are less likely to have the condition based on their clinical history and physical examination) may have lower sensitivity to D-dimer [12]. Conversely, patients with a high pretest probability of PE (i.e., those who are more likely to have the condition regarding their clinical history and physical examination) may have higher sensitivity to D-dimer [13]. In summary, while D-dimer is a useful screening tool for the diagnosis of PE, it is not 100% sensitive and must be used in conjunction with other diagnostic tests and clinical judgment [14].

Troponin I is a protein present in muscle cells of the heart that is released into the blood circulation when the heart is injured or stressed. Troponin is commonly used as a diagnostic marker for acute coronary syndrome (ACS) and other cardiac conditions, but its role in the diagnosis of PE is less clear [15]. While elevated troponin I levels have been reported in some patients with PE, the diagnostic sensitivity of troponin in PE is relatively low. A meta-analysis of 13 studies that included a total of 1,330 patients with PE found that the overall sensitivity of troponin I for the diagnosis of PE was 24.2%. This means that among 100 patients with confirmed PE, only about 24 will have elevated troponin I levels [11]. The low sensitivity of troponin I in PE is thought to be due to several factors. First, PE typically does not directly affect the heart muscle, so tro-

Table 1. Comparison of sociodemographic features of PTE Patients and Non-PE patients.

	PE (n=51)				Non-PE (n=347)				p
	Median	IQR	95% CI Lower	95% CI Upper	Median	IQR	95% CI Lower	95% CI Upper	
Age, years	73	24	65	74	59	29	56	60	**<0.001**[1]
Gender, n (%)									
Female	24 (47.1)				200 (57.6)				0.155[2]
Male	27 (52.9)				147 (42.4)				

PE: pulmonary embolism. [1]Mann-Whitney U test was used. [2]Chi-square test was used. p<0.05 was accepted as statistically significant.

Table 2. Comparison of laboratory parameters of PE patients and Non-PE patients

	PE (n=51)				Non-PE (n=347)				p
	Median	IQR	95% CI Lower	95% CI Upper	Median	IQR	95% CI Lower	95% CI Upper	
Hemoglobin, mg/dl	13.20	2.80	12.89	13.95	13.00	2.00	12.87	13.24	0.195
Albumin, mg/dl	3.50	1.3	3.39	3.96	3.60	1.1	3.60	3.80	0.487
Uric acid, 10^3/L	4.40	1.3	4.38	5.29	4.40	1.4	4.59	4.90	0.957
WBC, 10^3/L	8.20	4.40	8.08	10.43	8.20	2.90	8.11	9.36	0.468
Neutrophil, 10^6/L	6.00	4.10	5.66	7.26	5.50	2.80	5.76	6.39	0.190
Lymphocyte, 10^3/L	1.30	1.10	1.35	1.89	1.20	0.25	1.16	1.64	0.592
Platelet, 10^3/L	241	187	232	305	254	187	236	286	0.870
Monocyte, 10^3/L	0.60	0.20	0.53	0.62	0.50	0.25	0.49	0.54	**0.044**
Eosinophil, 10^3/L	0.03	0.02	0.03	0.06	0.02	0.02	0.02	0.05	0.747
Basophil, 10^3/L	0.05	0.03	0.04	0.09	0.05	0.07	0.05	0.08	0.735
MPV, mg/dL	8.20	1.2	7.60	8.20	8.20	1.8	7.80	8.22	0.578
PCT, mg/dL	0.35	0.34	0.30	0.43	0.35	0.46	0.35	0.42	0.882
PDW, mg/dL	12.70	2.6	12.20	14.82	12.60	2.5	12.50	13.80	0.916
D-dimer, mg/dL	1690	2665	1550	5084	214	184	210	340	**<0.001**
CRP, mg/dl	2.20	3.8	1.80	5.25	4.40	3.4	4.33	4.91	**0.001**
Troponin I, g/L	0.04	0.24	0.02	0.35	0.01	0.00	0.01	0.02	**<0.001**

PE: pulmonary thromboembolism; WBC: white blood cell; MPV: mean platelet volume; PCT: plateletcrit; PDW: platelet distribution width; CRP: C-reactive protein. Mann-Whitney U test was used. p <0.05 was accepted as statistically significant.

Table 3. Comparison of inflammatory ratios of PE patients and Non-PE patients

	PE (n=51)				Non-PE (n=347)				p
	Median	IQR	95% CI Lower	95% CI Upper	Median	IQR	95% CI Lower	95% CI Upper	
NLR	4.66	4.93	4.52	8.80	4.18	4.03	4.12	5.89	0.604
MLR	0.38	0.39	0.38	0.76	0.36	0.36	0.34	0.48	0.581
PLR	162.72	227.78	152.33	391.75	175.23	189.16	172.22	271.36	0.853
CAR	0.60	1.00	0.51	1.76	1.20	0.98	1.18	1.44	**0.001**
NAR	1.62	1.12	1.61	2.22	1.48	0.92	1.42	1.87	0.311
UAR	1.31	0.83	1.26	1.57	1.23	0.63	1.20	1.42	0.422

PE: pulmonary thromboembolism; NLR: neutrophil lymphocyte ratio; MLR: monocyte to lymphocyte ratio; PLR: platelet lymphocyte ratio; CAR: CRP albumin ratio; NAR: neutrophil albumin ratio; UAR: uric acid albumin ratio. Mann-Whitney U test was used. p <0.05 was accepted as statistically significant.

ponin I release in PE may be due to indirect effects such as increased right ventricular strain or hypoxemia. Second, troponin I levels can be elevated in a variety of non-cardiac conditions, including renal failure, sepsis, and pulmonary disease, which can make it difficult to distinguish between the cause of elevated troponin I levels. Despite its low

Table 4. Comparison of sociodemographic characteristics of PE patients according to Unilateral and Bilateral lung involvement.

	Unilateral involvement (n=29)				Bilateral involvement (n=22)				p
	Median	IQR	95% CI Lower	95% CI Upper	Median	IQR	95% CI Lower	95% CI Upper	
Age, years	72	25	61	75	76	25	65	78	0.408[1]
Gender, n (%)									
Female	11 (37.9)				13 (59.1)				0.134[2]
Male	18 (62.1)				9 (40.9)				

PE: pulmonary embolism. [1]Mann-Whitney U test was used. [2]Chi-square test was used. p<0.05 was accepted as statistically significant.

Table 5. Comparison of laboratory parameters of PE patients according to Unilateral and Bilateral lung involvement.

	Unilateral involvement (n=29)				Bilateral involvement (n=22)				p
	Median	IQR	95% CI Lower	95% CI Upper	Median	IQR	95% CI Lower	95% CI Upper	
Hemoglobin, mg/dl	13.20	2.75	12.67	14.05	13.20	2.82	12.60	14.40	0.947
Albumin, mg/dl	3.50	1.2	3.19	3.91	3.65	2.2	3.32	4.31	0.272
Uric acid, 10^3/L	4.30	2.0	4.29	5.73	4.40	1.2	4.09	5.11	0.746
WBC, 10^3/L	7.40	4.10	7.38	9.30	9.10	5.05	8.00	12.88	0.177
Neutrophil, 10^6/L	6.00	3.21	5.31	6.85	6.50	4.27	5.32	8.57	0.753
Lymphocyte, 10^3/L	1.20	1.25	1.20	1.85	1.45	1.15	1.25	2.24	0.753
Platelet, 10^3/L	254	204	240	336	216	201	183	300	0.135
Monocyte, 10^3/L	0.60	0.25	0.48	0.63	0.57	0.19	0.54	0.66	0.609
Eosinophil, 10^3/L	0.02	0.02	0.02	0.04	0.02	0.03	0.02	0.09	0.424
Basophil, 10^3/L	0.05	0.07	0.05	0.11	0.06	0.10	0.05	0.12	0.654
MPV, mg/dL	8.20	1.50	7.49	8.32	7.95	1.20	7.42	8.37	0.731
PCT, mg/dL	0.36	0.43	0.31	0.49	0.32	0.38	0.22	0.41	0.318
PDW, mg/dL	12.30	2.10	12.06	14.37	13.10	5.10	12.84	16.41	0.185
D-dimer, mg/dL	1365	2272	974	4573	2604	2031	1756	7354	**0.022**
CRP, mg/dl	2.20	4.80	2.17	6.57	2.20	3.4	2.09	4.52	0.909
Troponin I, g/L	0.01	0.03	0.01	0.09	0.25	0 0.54	0.21	0.72	**<0.001**

PE: pulmonary thromboembolism; WBC: white blood cell; MPV: mean platelet volume; PCT: plateletcrit; PDW: platelet distribution width; CRP: C-reactive protein. Mann-Whitney U test was used. p <0.05 was accepted as statistically significant.

Table 6. Comparison of inflammatory ratios of PE patients according to Unilateral and Bilateral lung involvement.

	Unilateral involvement (n=29)				Bilateral involvement (n=22)				p
	Median	IQR	95% CI Lower	95% CI Upper	Median	IQR	95% CI Lower	95% CI Upper	
NLR	4.66	5.17	3.93	11.16	4.66	4.66	3.76	7.22	0.984
MLR	0.37	0.39	0.32	0.97	0.38	0.43	0.33	0.60	0.970
PLR	198.57	294.08	180.43	531.81	154.76	134.52	118.81	273.14	0.180
CAR	0.63	1.37	0.61	2.15	0.56	0.86	0.45	1.70	0.648
NAR	1.57	1.04	1.51	2.13	1.68	1.78	1.42	2.64	0.866
UAR	1.37	0.85	1.26	1.71	1.20	0.82	1.09	1.53	0.250

PE: pulmonary thromboembolism; NLR: neutrophil lymphocyte ratio; MLR: monocyte to lymphocyte ratio; PLR: platelet lymphocyte ratio; CAR: CRP albumin ratio; NAR: neutrophil albumin ratio; UAR: uric acid albumin ratio. Mann-Whitney U test was used. p <0.05 was accepted as statistically significant.

sensitivity, troponin I can still be useful in the diagnosis of PE in certain situations. For example, in patients with suspected PE who also have chest pain or other signs of cardiac involvement, troponin I can help identify those

Table 7. ROC curve analyses of laboratory parameters.

Variables	AUC	%95 CI		p	Cut-off	Sen (%)	Spe (%)	LR
		Lower	Upper					
D-dimer	0.921	0.861	0.982	**<0.001**	655	90.2	96.5	26.08
					438.5	92.2	93.1	13.32
Troponin I	0.815	0.733	0.896	**<0.001**	0.0125	64.7	97.4	24.94
Monocyte	0.586	0.503	0.670	**0.046**	0.475	4.38	68.9	1.22
D-dimer*Troponin I	0.937	0.881	0.993	**<0.001**	5.57	92.2	94.5	16.83

ROC: receiver operating characteristics; AUC: area under curve; CI: confidence interval; LR: likelihood ratio.

who may be at increased risk of adverse outcomes and require more aggressive treatment. In addition, troponin I may be used as a prognostic marker in patients with confirmed PE, as elevated troponin I levels have been related to increased mortality and other adverse outcomes [16]. While troponin I is not a sensitive diagnostic marker for PE, it can still provide valuable information in certain clinical contexts. Clinicians must interpret troponin I levels in the condition of the patient's clinical history and other diagnostic symptoms to make an accurate diagnosis and determine appropriate treatment [17].

White blood cells known as monocytes are crucial in the immune response to infections and other forms of tissue damage. While monocytes have not been widely studied as a diagnostic marker for pulmonary embolism (PE), there is some proof to propose that they may be useful in identifying patients with this condition. According to a 2015 research in the Journal of Thrombosis and Hemostasis, people with acute PE had considerably more monocytes in their peripheral blood than healthy individuals did. In addition, the study found that the monocyte-to-lymphocyte ratio (MLR) was significantly greater in patients with PE than in those without the condition. The MLR is a ratio of the number of monocytes to the number of lymphocytes in the blood and has been proposed as an index of systemic inflammation [18]. Another study found that the proportion of CD16+ monocytes (a subtype of monocytes that has been associated with inflammation) was statistically greater in patients with acute PE compared to those without the condition. The study also discovered a favorable correlation between PE severity and the percentage of CD16+ monocytes [19]. While these studies proposed that monocytes may be a useful diagnostic marker for PE, more research is needed to confirm their diagnostic sensitivity and specificity. In addition, the use of monocytes as a diagnostic marker for PE may be limited by the fact that monocyte levels can be impacted by a variety of other factors, including infection, inflammation, and certain medications. While there is some evidence to suggest that monocytes may be a useful diagnostic marker for PE, more research is needed to confirm their diagnostic sensitivity and specificity. To correctly diagnose PE, clinicians must evaluate monocyte levels in the situation of the patient's clinical history and other diagnostic findings.

NLR and PLR have been established in earlier research to have an elevated inflammatory response and prognostic value in PE and cardiovascular illnesses. Increases NLR, and PLR have also been linked to 30-day mortality in PE, according to research. NLR and PLR were discovered to be elevated in research that was done in a group with severe PE [20]. Another study found that because of the enhanced inflammatory response, NLR might be utilized as a predictor of 30-day death [21]. The NLR and PLR rates between the group with and without PE did not differ significantly in our investigation, contrary to what has been reported in the literature. However, we found a significantly higher monocyte count in the PE group. In a study investigating in-hospital mortality in patients followed up for PE in the literature, MHR was found to be higher in the group with mortality compared to the group without mortality [22]. Although we did not have information on mortality rates in this study, comparisons could be made with literature data after follow-up of these patients.

The CAR value in our study's non-PE group was discovered to be substantially greater than that in the PE group. The high CAR value in the group without PE in our study may be caused by other illnesses where inflammation is obvious, such as pneumonia, upper respiratory tract infection, and pericarditis, even though inflammatory markers were shown to be high in PE. High CAR was shown to have a predictive value for 6-month mortality in a study on the CAR value in PE in the literature [23]. Previous research has demonstrated that the pulmonary vascular occlusion and contraction seen in PE can cause a sharp elevation in right ventricular and pulmonary artery pressure. This rise in troponin I levels can result in right ventricular dilatation, right ventricular myocardial ischemia, or possibly myocardial infarction [24]. PE can also result in a sudden rise in right ventricular and pericardial tension, which can constrict the coronary artery, produce partial myocardial ischemia, damage to and necrosis of myocardial cells, and release troponin I [25]. Patients with PE who have elevated blood troponin I levels have right ventricular dysfunction, which is linked to a much higher risk of right heart failure and cardiogenic shock. Serum troponin I levels were discovered to be elevated in PE patients by Walter et al. [26]. The probability of mortality was strongly linked with the size of this rise in troponin I level in PE patients with right ventricular failure. In conclusion, even if their hemodynamics are stable, individuals with PE and elevated troponin I levels are at an increased risk of passing away [27].

Although laboratory findings and imaging provide valuable information in patients with suspected PE, clinicians should pay attention to the medical history, initial rhythm, and auscultation findings of these patients. Although PE patients often present hemodynamically stable, some patients may present with cardiac arrest or pulseless electrical activity. Massive PE is a condition that should be con-

sidered in unexplained out-of-hospital cardiac arrests [28]. This study has some limitations. The previous hemogram parameters, CRP and D-dimer, and troponin I level of the cases with PE were unknown, so the previous and subsequent hemogram, CRP, and D-dimer levels could be compared. In our study, we think that factors that can change blood parameters should be used in future studies since there is no data on whether severe PE cases received emergency thrombolytic therapy or whether they were followed in the intensive care unit. Since mortality records were not obtained, the mortality rates of high-risk PE patients in this study and the inability to evaluate the causes that may affect it are among the limitations of our study. In addition, the lack of transthoracic echocardiographic findings of PE patients and the lack of information on whether right heart failure develops in these patients is a limitation.

In the present study, one of the most important indicators in the diagnosis of PE, similar to those in the literature, was the D-dimer and troponin I test. The highest specificity in excluding the diagnosis of PE was found with troponin I (97.4%). According to the ROC analysis in the present study, when the D-dimer and troponin I values were considered together, the AUC value was found to be the highest (AUC: 0.937). Two different cut-offs were found for D-dimer. When D-dimer was 655 mg/dL, the specificity was 96.5%, while when D-dimer was 438.5 mg/dL, the sensitivity was 92.2%.

As a result, the main message of the present study is that when making differential diagnoses in patients who apply to the emergency department with dyspnea and chest pain, better diagnostic accuracy can be achieved with the combined evaluation of tomographic findings and laboratory findings. In addition, higher D-dimer and troponin I blood levels were detected in PE patients with bilateral lung involvement, indicating the importance of these two parameters in PE patients.

Ethical approval

The study was accomplished after the consent of the Adıyaman University Non-Interventional Clinical Research Ethics Committee (Decision number: 2022/7-54). A signed informed consent form was taken from all participants to be included in the study, and they were enlightened that participation was non-compulsory and that they could be free to quit from the research. The study went on in compliance with the Helsinki Declaration.

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