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Article

The Role of Pulmonary Artery Obstruction Index Ratio in Predicting Clinical Course in Pulmonary Embolism

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Abstract: ***Background/Objective*:** This study aimed to investigate the relationship between pulmonary arterial computed tomography obstruction index ratio (CTOI) and simplified pulmonary embolism severity index (sPESI), one of the clinical probability scoring modalities, in determining the severity of PE and to determine whether CTOI is a mortality marker. ***Methods:*** The study included 117 patients diagnosed with PE by Computed Tomography Pulmonary Angiography (CTPA). CTOI was determined according to the localization of the embolus and the obstruction caused by the embolus in the vessel. Patients were divided into groups according to sPESI. Patient deaths up to six months after PE diagnosis were recorded. Logistic regression analysis was performed to identify predictors of mortality. ***Results:*** According to sPESI classification, there was no difference in CTOI between high-risk and low-risk groups. After six months of follow-up, there was no difference in CTOI rate between the patients who died and those who survived. ***Conclusion*** Although CTPA is the gold standard for diagnosing PE, it would be more appropriate to use it together with clinical findings to determine the severity of the disease. Further evaluation is needed to investigate the usefulness of the obstruction index and CT findings of right ventricular dysfunction for classifying patient risk and determining therapeutic options.

Keywords: acute pulmonary embolism (APE); sPESI; CTOI; mortality; IVSD; PAD/AoD; IVCR

1. Introduction

Venous thromboembolism (VTE) is the third most common cardiovascular disease after cerebrovascular disease and acute myocardial infarction, and its mortality can be up to 25% [1,2]. Pulmonary embolism (PE) patients may have different outcomes and require different intensities of clinical care. Risk assessment is, therefore, necessary to decide on the appropriate management strategy [2]. This risk assessment can be based on clinical, biochemical, and imaging parameters [3]. Simplified pulmonary embolism severity index (sPESI), one of the clinical parameters used to determine prognosis in PE patients, is important in determining the clinical course [4]. D-dimer, brain natriuretic peptide (BNP), cardiac troponin, Heart-type fatty acid-binding protein (H-FABP), and lactate values are among the laboratory tests used to determine prognosis [3]. Right ventricular (RV) failure due to a massive embolism is a critical determinant of clinical severity and outcome [5]. Computed Tomography Pulmonary Angiography (CTPA), and transthoracic echocardiography (TTE) can determine the degree of RV failure. Increased main pulmonary artery diameter (PAD),

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pulmonary artery to aortic diameter ratio (PAD/AoD), interventricular septal deviation (IVSD), and inferior vena cava reflux (IVCR) are among the parameters that can be evaluated with CTPA [6]. Qanadli et al. defined the pulmonary arterial computed tomography obstruction index ratio (CTOI) to quantify the degree of pulmonary arterial obstruction and to determine whether it correlated with pulmonary artery pressure and right ventricle function [7]. The primary aim of our study was to determine whether CTOI is a mortality predictor by revealing the relationship between pulmonary arterial computed tomography obstruction index ratio (CTOI) and simplified pulmonary embolism severity index (sPESI), which is one of the clinical probability scores, in determining the severity of PE. The secondary aim is to determine other factors affecting mortality in PE patients.

2. Materials and Methods

We retrospectively evaluated the data of 1035 patients hospitalized in Selçuk University Faculty of Medicine, Department of Chest Diseases and diagnosed with APE between 2019 and 2022. Patients with insufficient contrast passage on CTPA, suspicious filling defects, embolism diagnosed by ventilation-perfusion (V/Q) scintigraphy, presence of chronic thromboembolic pulmonary hypertension, pregnancy, age below 18 years, lung surgery, or incomplete clinical/laboratory findings were excluded. The study included 117 patients with CTPA-proven APE. Symptoms, physical examination findings, CTPA, doppler ultrasound and laboratory results were recorded on the evaluation form from hospital data processing records. All patients also underwent standard CTPA within 6 hours. A total of 60 mL of intravenous iohexol was administered by 4 mL/s flow rate. CTPA obtained with bolus tracking technique by putting region of interest to the pulmonary artery with a 256-MDCT (Multidetector Computed Tomography) scanner (Siemens Somatom Definition Flash, Erlangen, Germany). Scan parameters were 120 kV, 1-mm slice thickness and 1.5 pitch value. Coronal reformatted and axial/coronal 20-mm thick maximum intensity projection images were also created. İmage evalution done with mediastinum window by a senior pulmonary medicine resident. CTOI was calculated according to the localization of the embolus on Computed Tomography Pulmonary Angiography and the occlusion in the vessel [7]. IVSD, IVCR, and PAD/AoD were examined to determine right ventricular dysfunction.

Statistical Analyses

All statistical analyses were performed in R version 4.1.2 (www.r-project.org) with the help of a statistical programming language. Before the analyses, the normality of the data was checked using Shapiro-Wilk's normality test, Q-Q graphs, and Levene's test. Numerical variables are presented as mean ± standard deviation or median (quartiles), and categorical variables are presented as frequency (n) and percentage (%). Statistical differences in demographic, clinical and radiologic findings and blood parameters of patients according to sPESI groups and mortality status were analyzed by Independent sample t-test, Welch's test or Mann-Whitney U test for numerical variables and by Yates continuity-corrected chi-square, Fisher's exact test, or Fisher-Freeman-Halton test for categorical variables. In addition, CTOI values of radiologic parameters were compared using the Mann-Whitney U test. The level of significance was set at 5%

3. Results

The study included 117 patients (50 males, 67 females) with a mean age of 63.29 ± 16.29 (24-115) years. In all patients, clinical suspicion of PE was confirmed by the presence of at least one filling defect in the pulmonary arterial tree by CTPA. When etiologic factors were analyzed, the rate of hormone therapy was higher in the high-risk group (n=79/89; 88% vs. n=20/28; 71.4%, p=.037). Oral contraceptive (OCS) use, prolonged travel/immobility, trauma, previous surgery, genetics, obesity, or idiopathic causes were not significantly different between the groups. Comorbidities were present in 102 patients (87%). Arterial hypertension (HT) (70 patients), malignancy (28 patients), and chronic obstructive pulmonary disease (COPD) (25 patients) were the most common comorbidities (Table 1).

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Six patients (5.1%) received thrombolytic therapy. A total of 13 patients died. The most common accompanying symptoms were dyspnea, palpitations, and chest pain, respectively. These were followed by fatigue, leg swelling, syncope/presyncope, and hemoptysis. Dyspnea was present in 99 (84.6%) patients and was more common in high-risk patients (p=.037). There was no difference between the groups in other symptoms. The patients' mean body mass index (BMI) was 28.26 ± 4.82. Ten patients (8.5%) presented with recurrent PE. Deep vein thrombosis (DVT) was detected in 26 patients (22.6%). There was no difference between the groups regarding BMI, recurrent PE (p=.701), and DVT (p=.666). The mean CTOI on radiologic examination was 34.55 ± 25.97 (2.5-150). Parenchymal infarcts were detected in 32 patients (27.6%), IVSD in 16 (13.8%) and IVCR in 20 (17.2%). Mean AoD was 32.26 ±4.83 (21-46) mm; mean PAD was 28.29 ±4.78 (18-40) mm; right pulmonary artery (RPA) mean diameter was 18.45 ± 3.72 (10-29) mm; left pulmonary artery (LPA) diameter averaged 18.54 ± 3.67 (11-27) mm. PAD/AoD ratio was 1.17 ± 0.24 (0.70-1.89).

Table 1. Patient characteristics of the study cohort.

	PESI groups		
	Low risk (*n*=28)	**High risk (*n*=89)**	***p*-value**
Age (years), mean ± SD	52.43 ± 12.79	66.71 ± 15.83	**<.001**[1]
Gender (F/M), *n* (%)	14 (50) / 14 (50)	53 (59.6) / 36 (40.4)	.502[2]
BMI (kg/m^2), mean ± SD	29.46 ± 3.54	27.88 ± 5.12	.073[3]
Saturation, median (min-max)	92.5 (90 – 98)	90 (76 – 98)	**<.001**[4]
Comorbidity distributions, *n* (%)			
Presence of comorbidities	21 (75)	81 (91)	.047[5]
DM	6 (21.4)	13 (14.6)	.391[5]
HT	12 (42.9)	58 (65.2)	.060[2]
AF	1 (3.6)	12 (13.6)	.184[5]
CHF/CAD	0 (0)	15 (16.9)	.020[5]
BA	0 (0)	19 (21.3)	**.006**[5]
COPD	1 (3.6)	24 (27)	**.018**[2]
Malignity	0 (0)	28 (31.5)	**.002**[2]
CRF	0 (0)	2 (2.2)	>.999[2]
RA-Other	6 (21.4)	14 (15.7)	.566[5]
Symptom distribution, *n* (%)			
Dyspnea	20 (71.4)	79 (88.8)	.037[5]
Syncope/Presyncope	4 (14.3)	14 (15.7)	>.999[5]
Hemoptysis	3 (10.7)	13 (14.6)	.759[5]
Fatigue	3 (10.7)	19 (21.3)	.328[2]
Angina Pectoris	10 (35.7)	21 (23.6)	.307[2]
Palpitations	12 (42.9)	28 (31.5)	.379[2]
Leg Swelling	2 (7.1)	18 (20.2)	.152[5]
Etiologic Factor distributions, *n* (%)			
Hormone therapy	0 (0)	15 (16.9)	**.020**[5]
OKS kullanımı	0 (0)	2 (2.2)	>.999[5]
Prolonged travel/immobility	11 (39.3)	18 (20.2)	.074[2]
Trauma	0 (0)	2 (2.2)	>.999[5]
Past surgery	8 (28.6)	27 (30.3)	>.999[5]

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Genetics	5 (17.9)	11 (12.4)	.530[5]
Obesity	2 (7.1)	4 (4.5)	.628[5]
Idiopathic	6 (21.4)	18 (20.2)	>.999[5]
DVT , n (%)	5 (17.9)	21 (24.1)	.666[2]
Relapsing embolism, *n* (%)	3 (10.7)	7 (7.9)	.701[5]
Thrombolytic, *n* (%)	1 (3.6)	5 (5.6)	>.999[5]

[1] Independent sample t-test, [2] Yates continuity corrected chi-square test, [3] Welch's t-test, [4] Mann-whitney U test, [5] Fisher's exact test, [6] Fisher-Freeman-Halton test. Abbreviations: DM: Diabetes mellitus; HT: Hypertension; AF: Atrial Fibrillation; CHF/CAD: Congestive Heart Failure / Coronary Artery Disease; BA: Bronchial Asthma; COPD: Chronic Obstructive Pulmonary Disease; CRF: Chronic Renal Failure; RA: Rheumatoid Arthritis.

Demographic data of the patients are summarized in Table 1. There were 28 patients in the low-risk group (sPESI=0) and 89 patients in the high-risk group (sPESI ≥1). Gender distribution between groups (F/M: 53 (59.6) / 36 (40.4) vs 14 (50) / 14 (50), p=.502) and there was no difference in BMI (27.88 ± 5.12 vs 29.46 ± 3.54 p=.073). The mean age (66.71 ± 15.83 vs 52.43 ± 12.79, p<.001) and presence of comorbidity were higher in high-risk patients (81 (91%) vs 21 (75%), p.047). Congestive heart failure (CHF)/ coronary artery disease (CAD) (p=.020), bronchial asthma (p=.006), chronic obstructive pulmonary disease (COPD) (p=.018), and malignancy (p=.002) were higher in the high-risk group. Oxygen saturation was lower in this group (90% (76 - 98) vs 92.5% (90 - 98), p<.001). There was no difference between the groups regarding receiving thrombolytic therapy (1 in the low-risk group and 5 in the high-risk group (p=.999)). The mortality rate was higher in the high-risk group (p=.036).

A comparison of laboratory findings between the groups is presented in Table 2. D-dimer (2083 (1165.75 - 4736.5) vs. 3583 (2055 - 7904.5), .046), Urea (33.5 (26.63 - 37.25 vs. 36 (30 - 49), .021), Creatine kinase MB isoenzyme (CK-MB) (1.08 (0.78 - 1.52) vs. 1.60 (1.02 - 2.90), (.017)) were high, while lymphocytes (2.31 ± 1.04 vs. 1.71 ± 0.84 (.003)), hemoglobin (12.96 ± 2.11 vs. 11.72 ± 2.09, .008)), hematocrit (38.97 ± 5.87 vs. 36.10 ± 6.01, (.031)) were low. No difference was found in other laboratory data.

Although CTOI, AoD, PAD, LPA, IVSD, IVCR, infarct and PAD/AoD ratio were higher in the high-risk group, statistical significance was found only in LPA (Table 3).

Table 2. Comparison of laboratory parameters.

	PESI groups		
	Low risk (*n*=28)	**High risk (*n*=89)**	***p*-value**
Laboratory Findings			
D-dimer	2083 (1165.75 – 4736.5)	3583 (2055 – 7904.5)	**.046[4]**
CRP	19.5 (4.5 – 46.75)	25 (5 – 67)	.410[4]
LDH	296 (209 – 376)	274 (225 – 329.5)	.957[4]
CKMB	1.08 (0.78 – 1.52)	1.60 (1.02 – 2.90)	**.017[4]**
Troponin	9.43 (3.77 – 25.75)	26.30 (7.92 – 43.70)	.054[4]
Lymphocytes	2.31 ± 1.04	1.71 ± 0.84	**.003[1]**
Lactate	2.5 (1.4 – 3.3)	1.8 (1.5 – 3)	.644[4]
ALT	21.5 (14.75 – 51)	22 (12 – 29)	.111[4]
AST	24 (16.75 – 36.25)	23 (16 – 34)	.643[4]
Creatine	0.87 (0.68 – 0.91)	0.81 (0.64 – 1.04)	.985[4]
Urea	33.5 (26.63 – 37.25)	36 (30 – 49)	**.021[4]**
Hb	12.96 ± 2.11	11.72 ± 2.09	**.008[1]**
MCHC	33.22 ± 1.56	32.43 ± 1.34	**.012[1]**

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RDW	13.60 (13.15 – 15.25)	14.90 (14 – 16.80)	**.002**[4]
WBC	9.40 (6.89 – 11.65)	8.81 (6.53 – 11.50)	.493[4]
Homocysteine	13.21 ± 6.12	14.53 ± 6.05	.374[1]
Protein-C	98.97 ± 33.41	102.60 ± 32.26	.644[1]
Protein-S	85.15 ± 22.39	81.25 ± 25.46	.511[1]
AT-3	105.32 ± 12.49	99.53 ± 20.09	.192[1]

[1] Independent sample t-test, [2] Yates continuity corrected chi-square test, [3] Welch's t-test, [4] Mann-Whitney U test, [5] Fisher's exact test, [6] Fisher-Freeman-Halton test. Abbreviations: CRP: C-reactive protein; LDH: Lactate Dehydrogenase; CK-MB: Creatine kinase MB isoenzyme; ALT: Alanine aminotransferase; AST: Aspartate transferase; MCHC: Mean Corpuscular Hemoglobin Concentration; RDW: Red cell distribution width; WBC: White Blood Count; AT-3: Antithrombin III.

Table 3. Comparison of radiologic parameters.

	PESI groups		
	Low risk (n=28)	**High risk (n=89)**	**p-value**
Radiological parameters			
CTOI	27.5 (14.38 – 53.13)	30 (12.5 – 50)	.674[4]
AoD	31.07 ± 5.07	32.63 ± 4.73	.138[1]
PAD	27.93 ± 3.87	28.40 ± 5.05	.648[1]
RPA	18.25 ± 3.53	18.52 ± 3.79	.742[1]
LPA	17.21 ± 2.64	18.96 ± 3.86	**.009**[3]
IVSD	5 (17.9)	11 (12.5)	.532[5]
IVCR	5 (17.9)	15 (17)	>.999[5]
Infarct	8 (28.6)	24 (27.3)	>.999[2]
PAD/AoD (>1)	20 (71.4)	65 (73)	>.999[2]
Mortality	0 (0)	13 (14.6)	**.036**[5]

[1] Independent sample t-test, [2] Yates continuity corrected chi-square test, [3] Welch's t-test, [4] Mann-Whitney U test, [5] Fisher's exact test, [6] Fisher-Freeman-Halton test. Abbreviations: CTOI: Computed tomography obstruction index ratio; AoD: Aortic diameter; PAD: Pulmonary artery diameter; RPA: Right pulmonary artery; LPA: Left pulmonary artery; IVSD: Interventricular septal deviation; IVCR: Inferior vena cava reflux; PAD/AoD: Ratio of pulmonary artery diameter to aortic diameter.

Table 4. Comparison of radiologic findings in patients with and without mortality.

	Mortality		
	Alive (n=104)	**Died (n=13)**	**p-value**
Radiological parameters			
CTOI	31.25 (12.50 – 50)	20 (12.5 – 27.5)	.242[4]
AoD	31.94 ± 4.80	34.77 ± 4.49	**.046**[1]
PAD	28.33 ± 4.72	28.00 ± 5.42	.817[1]
RPA	18.38 ± 3.71	19.08 ± 3.90	.523[1]
LPA	18.30 ± 3.49	20.46 ± 4.61	**.044**[1]
IVSD	16 (15.4)	0 (0)	.214[5]

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IVCR	19 (18.3)	1 (8.3)	.688[5]
Infarct	29 (27.9)	3 (25)	>.999[5]
PAD/AoD (>1)	73 (70.2)	12 (92.3)	.110[5]

[1] Independent sample t-test, [2] Yates continuity corrected chi-square test, [3] Welch's t-test, [4] Mann-Whitney U test, [5] Fisher's exact test, [6] Fisher-Freeman-Halton test. Abbreviations: CTOI: computed tomography obstruction index ratio; AoD: Aortic diameter; PAD: Pulmonary artery diameter; RPA: Right pulmonary artery; LPA: Left pulmonary artery; IVSD: Interventricular septal deviation; IVCR: Inferior vena cava reflux; PAD/AoD: Ratio of pulmonary artery diameter to aortic diameter.

The patients' laboratory, clinical and CTPA findings according to six-month survival are compared in Table 4. After six months of follow-up, 13 patients died (11.11%), and 104 (88.89%) were alive. The deceased group had a higher mean age (62.16 ± 16.44 vs. 72.31 ± 12.18, .034) and lower BMI (28.60 ± 4.91 vs. 25.53 ± 2.91, .030). The history of syncope/presyncope and palpitations was higher in the group that died compared to the group that survived (p=.029 and p=.034, respectively). There was no difference between the groups in terms of laboratory data. In radiologic findings, AoD (31.94 ± 4.80, 34.77 ± 4.49, .046) and LPA (18.30 ± 3.49, 20.46 ± 4.61, .044) were more increased in the deceased group. There was no statistically significant difference in the mean CTOI (31.25 (12.50 - 50), 20 (12.5 - 27.5), p=.242), MPA (28.33 ± 4.72, 28.00 ± 5.42, .817), and PAD/AoD >1 (73 (70.2), 12 (92.3), .110), IVSD (16 (15.4), 0 (0), .214), IVCR (19 (18.3), 1 (8.3), .688). Thrombolytic therapy was administered to 5 patients (4.8%) in the surviving group and one patient (7.7%) in the deceased group (p>.515).

A comparison of clinical and radiologic findings with CTOI in CTPA is shown in Table 5. CTOI was higher in those with IVSD (p<.001) and those who received thrombolytic therapy (.013). There was no difference in CTOI in aorta/MPA ratio (.147), IVCR (.438), infarcts (.818), mortality (.242), and recurrent embolism (.556).

Table 5. The relationship between computed tomography obstruction index and other indexes.

		n	CTOI	*p*-value
PAD/AoD	<1	91	25 (12.5 – 50)	.147
	≥1	26	37.5 (25 – 50)	
IVSD	No	100	25 (11.88 – 50)	**<.001**
	Yes	16	56.25 (45.63 – 75)	
IVCR	No	96	27.5 (12.5 – 50)	.438
	Yes	20	33.75 (16.25 – 55)	
Infarct	No	84	30 (12.5 – 50)	.818
	Yes	32	26.25 (12.5 – 50)	
Thrombolytic therapy	Not given	111	27.5 (12.5 – 50)	**.013**
	Given	6	56.25 (50 – 71.88)	
Mortality	No	104	31.25 (12.5 – 50)	.242
	Yes	13	20 (12.5 – 27.5)	
Relapsing Embolism	No	107	27.5 (12.5 – 50)	.556
	Yes	10	37.5 (15.63 – 50)	

Abbreviations: CTOI: computed tomography obstruction index ratio; AoD: Aortic diameter; PAD: Pulmonary artery diameter; IVSD: Interventricular septal deviation; IVCR: Inferior vena cava reflux; PAD/AoD: Ratio of pulmonary artery diameter to aortic diameter.

4. Discussion

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Effective treatment of PE in the acute phase depends on an accurate assessment of the patient's prognosis Aujesky et al. created a clinical prediction rule (PESI) that includes 11 clinical parameters [8]. High-risk PE is characterized by arterial hypotension or shock associated with PE [9]. Risk stratification for normotensive patients diagnosed with PE should aim to distinguish the low-risk group of patients from the intermediate-high-risk group of patients with preserved systemic arterial pressure and a high risk of a complicated course. Clinicians may use separate prognostic approaches to identify low-risk and intermediate-high-risk patients [4]. Numerous studies have confirmed the prognostic accuracy of PESI [10–12]. The sPESI, which was created because the PESI scoring is highly parametric and impractical to use, has been shown to successfully predict 30-day mortality after acute symptomatic PE and when compared with the PESI, the sPESI has similar prognostic accuracy [4]. Patients with a low-risk sPESI are unlikely to have an early adverse outcome and do not need additional imaging examinations or laboratory tests to predict low risks for adverse outcomes. Studies support the idea that compared with imaging and laboratory biomarkers or combinations thereof, standardized clinical scores more accurately identify patients at low risk for fatal and non-fatal adverse medical outcomes in the acute phase after PE diagnosis [13–16]. It has also been reported that the sPESI score is associated with long-term mortality [17]. In our study, 89 patients were in the high-risk group according to the sPESI risk classification. The mortality rate was significantly higher in the high-risk group, as reported in the literature. Our findings suggest that clinical scoring may be more effective than laboratory and imaging methods in identifying high-risk patients and detecting possible adverse events related to them.

Signs known to be associated with increased mortality risk in patients with pulmonary embolism include persistent hypotension and signs of right ventricular dysfunction. Enzymes, markers of cardiac damage, and echocardiography were independent risk factors in these patients [9,18]. However, it is important to use standardized and easily accessible radiological parameters in such patients because it is not clear how to make a prognostic classification in the case of normal blood pressure, echocardiographic demonstration of signs of right ventricular dysfunction is not always possible, and there are differences in personal interpretation.

Pulmonary artery obstruction index (CTOI) determined by pulmonary arterial computed tomography may be important in determining the severity of embolism, indicating thrombus burden and determining treatment protocols [19]. The cardiovascular consequences of APE should be assessed not only as a consequence of the degree of pulmonary vascular obstruction but also by the degree to which it requires the right ventricle to function as a high-pressure pump. Therefore, obstruction and right ventricular reserve determine the clinical outcome. Our study investigated the relationship between CTPA parameters and clinical and laboratory data and the relationship between CTOI and patient outcomes. Our results suggest that although quantitative determination of thrombus burden in the pulmonary artery with the CTOI score is valuable in evaluating PE and guiding treatment strategies, its predictive value alone may be insufficient to determine the patient's prognosis. Studies have failed to show an association between a high obstruction index and mortality and an unstable hemodynamic picture, and there is limited predictive ability for mortality [20–26]. Akhoundi et al. also stated that the Qanadli score should be modified because of its inadequacy in predicting mortality [27]. In a study using the Mastora score, an alternative method to assess pulmonary vascular occlusion, it was emphasized that thrombotic occlusion was not associated with the clinical severity of PE [24]. Studies are showing that CTOI helps predict mortality. Kumamaru et al. reported that CTPA-based thrombus burden scoring was superior to PESI in predicting 30-day mortality in their study to predict prognosis [28]. CTOI has been reported to be strongly predictive for high-risk patients [29,30]. In another study, it was reported that a high CTOI score significantly predicted 30-day mortality and high CTOI (> 46.2; $p = 0.004$) was independently associated with mortality in multivariable regression analysis [31]. In a study in which 30-day mortality was followed, it was reported that the mean CTOI was significantly higher in the deceased group [32]. Although the results of the studies are contradictory, recent publications show that the CTOI score alone is insufficient in predicting mortality, similar to our study. However, when combined with

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clinical data, it may help physicians choose the most appropriate approach for the management of patients.

The interventricular septum may shift toward the LV due to increased right ventricular pressure with severe pulmonary arterial obstruction [33]. IVSD has been reported to indicate RV dysfunction [32,34–38]. IVSD is predictive for adverse outcomes such as endotracheal intubation, vasopressor therapy, thrombolytic therapy, cardiopulmonary resuscitation and surgical embolectomy but has not been reported to be associated with 30-day mortality [39]. Although one study reported that IVSD was associated with short-term mortality, defined as in-hospital death or death within 30 days, with low sensitivity [40], most studies have reported that IVSD does not detect death from acute PTE [41–44]. In our study, there was no difference in mortality in 16 (13.8%) patients with IVSD at 6-month follow-up, as reported in most studies. There was no difference in receiving thrombolytic therapy in this group. Since IVSD is not specific to acute PE and can be found in many diseases that cause increased pulmonary artery pressure, a more careful evaluation of IVSD may be a helpful finding in the patient's follow-up. However, using it with other findings to predict clinical outcomes may be more appropriate, as in CTOI.

The ratio of pulmonary artery diameter (PAD) to aortic diameter (AoD) (PAD/AoD) is known to be an important factor in determining pulmonary hypertension, regardless of its etiology [45]. In patients with APE, mean pulmonary artery pressure (PAP) increases due to increased resistance in the pulmonary circulation. Pulmonary arterial pressure is an indirect indicator of hemodynamic damage and the degree of circulatory decompensation. The higher the PAP, the more significant the impact on prognosis becomes [17,46]. Some reports have shown that pulmonary trunk diameter may predict severity and mortality in PE [25,32,47,48]. There are also publications identifying only high left PAD as an independent risk factor for PE-related 30-day mortality [31]. In our study, there was a significant difference between the low and high-risk groups in terms of LPA. The correlation between pulmonary trunk diameter on CT and pulmonary artery pressure was analyzed. He reported that patients with severe PE had a larger pulmonary trunk diameter than patients with non-severe PE [49]. There is no consensus on the threshold value of the PAD/AoD ratio to predict the presence of pulmonary hypertension. Cut-off values of 0.83 and above have been recommended to determine the presence of pulmonary hypertension [50]. The PAD/AoD ratio was significantly higher in the group followed up with a diagnosis of APE and mortality compared to the healthy group [17]. In another study investigating the PAD/AoD ratio in acute PTE, the adequate accuracy of the PAD/AoD ratio in diagnosing moderate or severe pulmonary hypertension could not be demonstrated [51]. It was also reported that there was no correlation between pulmonary artery diameter and echocardiographically estimated PE pressure [34]. In many studies, pulmonary trunk diameter was not associated with mortality and pulmonary hypertension on CTPA alone cannot be used to diagnose severe PE [34,40,44,52,53]. We think the PAD/AoD ratio should not be used alone because there is no cut-off value for PAD/AoD ratio and no correlation between PAD/AoD ratio and ECHO used for pulmonary artery pressure measurement. The prognostic effect of the isolated high LPA diameter found in our study will need to be supported by further studies.

Some CT parameters used for detecting right ventricular failure require reformatted images for measurement, require calculation and cannot be standardized due to reasons such as being affected by respiratory or cardiac cycles or requiring time for measurement. In contrast, venous cava inferior (VCI) contrast agent reflux (VCIR) can be evaluated quickly and quickly from the original axial sections, can be graded semiquantitatively, and does not require special reformatting. A high degree of contrast reflux may capture a higher degree of RV physiologic impairment and identify a more severe group of patients [54]. Identifying IVCR as a predictor of mortality in patients with PE [25,26,31,43,44,52] that despite the existence of publications, IVCR is insufficient to identify patients at risk of early death [39,54] there are also publications stating that severe PTE cannot be differentiated from non-severe PTE [34]. Again, various factors such as heart failure, tricuspid regurgitation (TR), pulmonary hypertension and atrial fibrillation may contribute to contrast reflux to the IVC. In addition, the amount of contrast given during the evaluation of reflux and the speed at

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which it is given may change the results of this finding. Significant interindividual variability in the reproducibility of CT findings of RV dysfunction has also been reported. This may partly explain the variability in published results for predicting adverse outcomes based on findings on pulmonary CTA [42,55]. In our study, the clinical course of 20 (17.2%) patients with IVCR did not differ from those without IVCR. We think that IVCR alone may be insufficient to show the course of the disease and mortality and may be affected by many reasons, as mentioned above.

One of the limitations of our study is that it was retrospective. Another was that the evaluated CTPA findings were not correlating with a reference method such as echocardiography. However, considering that previous studies have established a good correlation between echocardiographic and CT findings, we assumed that our results correlated with echocardiographic findings [26,34–36,41,52,56]. In addition, different generations of CT scanners were used during our study, which can be considered a limitation. Finally, although our study reports mortality rates, it does not attempt to identify the causes of these deaths. Investigating causal relationships will require a more focused and forward-looking research methodology.

5. Conclusions

In conclusion, our study showed that CTOI might help identify high-risk patients but has no contribution to predicting mortality. We also found no association between CTOI and right ventricular dysfunction findings. Determining the definitive utility of the CTOI in managing acute PE and the optimal cut-off value to predict mortality in comprehensive randomized controlled trials may contribute to improving risk assessment and, thus, outcomes in PE

Abbreviations

PE: Pulmonary Embolism
CTOI: Computed Ttomography Obstruction Index Ratio
sPESI: Simplified Pulmonary Embolism Severity Index
CTPA: Computed Tomography Pulmonary Angiography
AoD: Aortic Diameter
LPA: Left Pulmonary Artery
RPA: Right pulmonary artery
VTE: Venous Thromboembolism
BNP: Brain Natriuretic Peptide
H-FABP: Heart-Type Fatty Acid-Binding Protein
RV: Right Ventricular
TTE: Transthoracic Echocardiography
PAD: Pulmonary Artery Diameter
PAD/AoD: Pulmonary Artery Diameter (PAD), Pulmonary Artery to Aortic Diameter Ratio
IVSD: Interventricular Septal Deviation
IVCR: Inferior Vena Cava Reflux
V/Q: Ventilation-Perfusion scintigraphy
MDCT: Multidetector Computed Tomography
Kv: Kilovolt
mm: millimeter
HT: Arterial hypertension
COPD: Chronic Obstructive Pulmonary Disease
BMI: Body Mass Index
DVT: Deep Vein Thrombosis
AF: Atrial Fibrillation
CHF/CAD: Congestive Heart Failure / Coronary Artery Disease
BA: Bronchial Asthma
CRF: Chronic Renal Failure
RA: Rheumatoid Arthritis

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CK-MB: Creatine Kinase MB Isoenzyme
CRP: C-Reactive Protein;
LDH: Lactate Dehydrogenase
CK-MB: Creatine Kkinase MB Isoenzyme
ALT: Alanine Aminotransferase
AST: Aspartate Transferase
MCHC: Mean Corpuscular Hemoglobin Concentration
RDW: Red Cell Distribution Width
WBC: White Blood Count
AT-3: Antithrombin III

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