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The accuracy of the Hounsfield unit in pulmonary embolism diagnostics

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Objective Pulmonary embolism (PE) is a vascular disease that is most frequently diagnosed using the radiological imaging technique computed tomography pulmonary angiography (CTPA). In this study, we aimed to demonstrate the diagnostic accuracy of the Hounsfield unit (HU) for PE based on the hypothesis that acute thrombosis causes an increase in HU value on CT.

Methods This research was a single-center, retrospective study. Patients presenting to the emergency department diagnosed with PE on CTPA were enrolled as the study group. Patients admitted to the same emergency department who were not diagnosed with PE and had noncontrast CT scans were included as the control group. A receiver operating curve was produced to determine the diagnostic accuracy of HU values in predicting PE.

Results The study population (n=74) consisted of a study group (n=46) and a control group (n=28). The sensitivity and specificity of the HU value for predicting PE on thoracic CT were as follows: for the right main pulmonary artery, 61.5% and 96.4% at a value of 54.8 (area under the curve [AUC], 0.690); for the left main pulmonary artery, 65.0% and 96.4% at a value of 55.9 (AUC, 0.736); for the right interlobar artery, 44.4% and 96.4% at a value of 62.7 (AUC, 0.615); and for the left interlobar artery, 60.0% and 92.9% at a value of 56.7 (AUC, 0.736).

Conclusion HU may exhibit high diagnostic specificity on CT for thrombi up to the interlobar level. An HU value exceeding 54.8 up to the interlobar level may raise suspicion of the presence of PE.

Keywords Hounsfield unit density; Pulmonary embolism; Noncontrast thorax computed tomography

Capsule Summary

What is already known

Pulmonary embolism is often diagnosed in the emergency department. Pulmonary angiography is the gold standard for pulmonary embolism diagnosis; however, computed tomography pulmonary angiography (CTPA), chest magnetic resonance imaging, chest x-ray, echocardiography, limb ultrasonography, and nuclear medicine imaging modalities are also used for diagnosis. CTPA is the most frequently used radiological imaging in clinical practice. An intravenous contrast agent is used for CTPA. In cases of contrast allergy or severe renal insufficiency, alternative diagnostic tools are needed.

What is new in the current study

We know that Hounsfield unit values can be used for radiological diagnosis of several diseases. In our study, noncontrast thoracic CT may exhibit high diagnostic specificity with Hounsfield unit density, especially for thrombi up to the interlobar level.

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INTRODUCTION

Pulmonary embolism (PE), a vascular disease with heightened morbidity and mortality, is often diagnosed in the emergency department (ED). PE is a difficult diagnosis for clinicians since it has no characteristic physical examination sign or symptom [1]. Pulmonary angiography is the gold standard for diagnosis. However, computed tomography pulmonary angiography (CTPA), chest magnetic resonance imaging, chest x-ray, echocardiography, limb ultrasonography, and nuclear medicine imaging modalities are also used for diagnosis [2,3], with CTPA as the most frequently used radiological imaging technique in clinical practice to diagnose PE [4–6]. An intravenous contrast agent is usually used for CTPA, but in cases of contrast allergy, severe renal insufficiency, and pregnancy, CT imaging can be performed without intravenous contrast to establish the diagnosis by indirect methods [7].

The Hounsfield unit (HU) is a relative quantitative measurement of radiodensity used by radiologists to interpret CT images. The linear transformation of radiodensity creates an HU scale that shows gray tones. Dense tissue, with better x-ray beam absorption, has positive values and appears bright; less dense tissue, with weaker x-ray beam absorption, has negative values and appears dark [8]. Using HU helps radiologists interpret images and diagnose diseases [9–12].

CT attenuation of whole blood and its parts has been studied [13,14]. Increases in clotted blood hematocrit cause a proportional increase in density that can be measured in HU. Therefore, acute thrombosis usually has an HU of 60 to 80 [13].

Previous studies have examined the diagnostic accuracy of HU values on CT for cranial venous thrombosis and deep vein thrombosis (DVT) [10,15]. In this study, we aimed to demonstrate the diagnostic accuracy of HU values for PE based on the hypothesis that acute thrombosis causes an increase in HU values on CT.

METHODS

Ethics statement

This study was approved by the Institutional Review Board of Recep Tayyip Erdoğan University T raining and Research Hospital (No. E-40465587-050.01.04-657) and the Ethics Committee of Recep Tayyip Erdoğan University (No. 2023/84). Informed consents for publication of the research details and clinical images were obtained from patients before starting the study. The study followed the principles outlined in the Declaration of Helsinki.

Study population and design

This research was conducted as a single-center, retrospective study. The study group included patients presenting to the ED of Recep Tayyip Erdoğan University Training and Research Hospital (Rize, Turkiye), a tertiary training and research hospital, between January 1 and December 31, 2021, who were diagnosed with PE on CTPA. The control group included patients presenting to the same ED between September 1 and October 1, 2022, who were not diagnosed with PE based on clinical and laboratory findings, who had undergone noncontrast thorax CT imaging, and with no prior history of PE.

All patients not meeting the exclusion criteria were included in the study (Fig. 1). Patients under 18 years of age, pregnant women, patients with a history of hematological malignancy, with bleeding findings, with histories of severe anemia (hemoglobin level, <8 g/dL), with CTPA and noncontrast thoracic CT images not suitable for measurements due to the presence of artifacts, and patients who died in the ED were excluded from both the study group and the control group. In addition, patients with clinically suspected PE but incomplete CTPA imaging and patients for whom CTPA did not diagnose PE were excluded from the study group.

Study protocol

The study population was formed after the exclusion criteria were applied to the study and control groups. All patient data were obtained from the hospital's digital archive. Examination of demographic data, comorbidities, admission symptoms, hematocrit index, and noncontrast thorax CT and CTPA findings (study and control groups) was performed.

CTPA and noncontrast thoracic CT findings were recorded at the initial presentation, and the two imaging modalities were evaluated by separate radiologists (one radiologist for each group). The radiologists evaluating the images had 3 years of experience in cardiothoracic CT imaging and were unaware of demographic data, comorbidities, presenting symptoms, and hematocrit index. Nevertheless, the radiologist who performed the CTPA evaluation was not blinded to the diagnosis of PE because they saw the contrast transmission. Further, radiologists were blind to each other's assessments.

Measurements

All patient CT scans were obtained with a 16-slice multidetector CT scanner (Toshiba Alexion, Toshiba Medical Systems Corp) with 1-mm-thick slices and 120 kVp. The radiologists independently evaluated the CT scans using the hospital's digital picture ar-

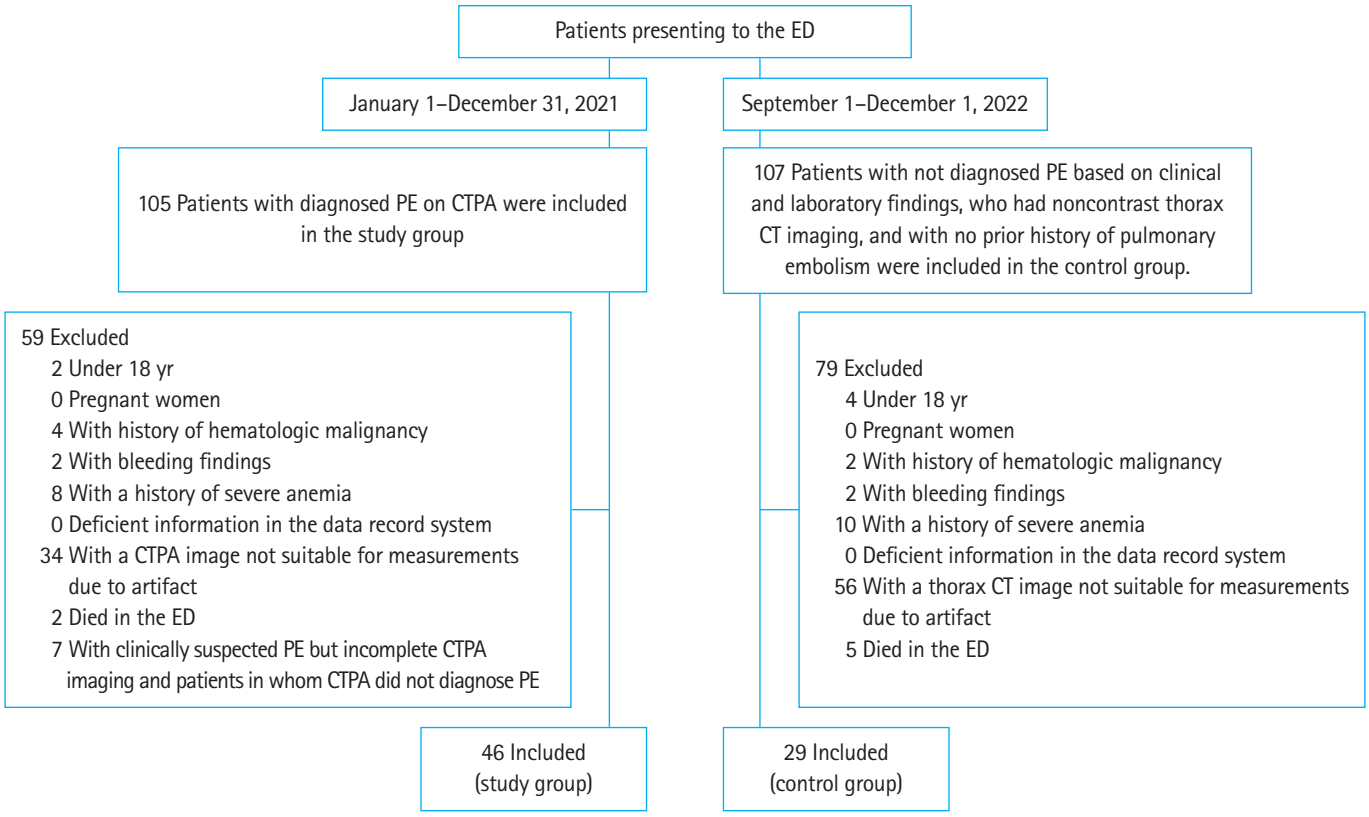

Fig. 1. Patient selection flowchart. ED, emergency department; PE, pulmonary embolism; CT, computed tomography; CTPA, computed tomography pulmonary angiography.

chiving and communication system. Images with artifacts that could impact measurement were eliminated from the assessment. Acute embolism was defined as a clot in the pulmonary arteries on CTPAs. This definition refers to areas where there was no contrast pass-through, in which all measurements in the study and control groups were performed.

In cases of PE with no contrast passage in the pulmonary arteries in contrast-enhanced CT, the HU values were measured by selecting the area with the most extensive filling defect for the region of interest (ROI). In the same way, similar-sized ROIs were used to obtain measurements from comparable levels in noncontrast CT images of patients with no prior PE. For standardization of measurements, an ROI size of 0.5 cm^2 was used for the main pulmonary artery (MPA), right main pulmonary artery (RMPA), and left main pulmonary artery (LMPA). Similarly, an ROI size of 0.3 cm^2 was used for the right interlobar artery (RILA) and left interlobar artery (LILA); and an ROI size of 0.05 cm^2 was used for the right upper lobe segmental branch (RULSB), right middle lobe segmental branch (RMLSB), right lower lobe segmental branch (RLLSB), left upper lobe segmental branch (LULSB), and left lower segmental branch (LLLSB).

In the study group (PE group), contrast-enhanced thorax CT HU measurements were performed in an area (thought to be a thrombus) without contrast passage. HU measurements were determined by standardized ROI size immediately distal to the area without contrast passage. Likewise, in the control group (non-PE group), noncontrast thorax CT HU value measurements were performed from the MPA to the distal segmental branches. HU measurements were determined by standardized ROI size. Noncontrast field measurements on contrast-enhanced CT planned in this way will likely include HU values of thrombus areas (which may also be normal). Conversely, the noncontrast area measurements on noncontrast CT include HU values of regular areas. The measurements of CT scans are shown in Figs. 2 and 3.

End points

The end point of this study is the presence of PE on thorax CT. We evaluated the diagnostic accuracy of the HU value for PE.

Statistical analysis

All statistical analyses were performed on Jamovi ver.1.6 (The Jamovi Project). Type I errors were accepted as 5% for all com-

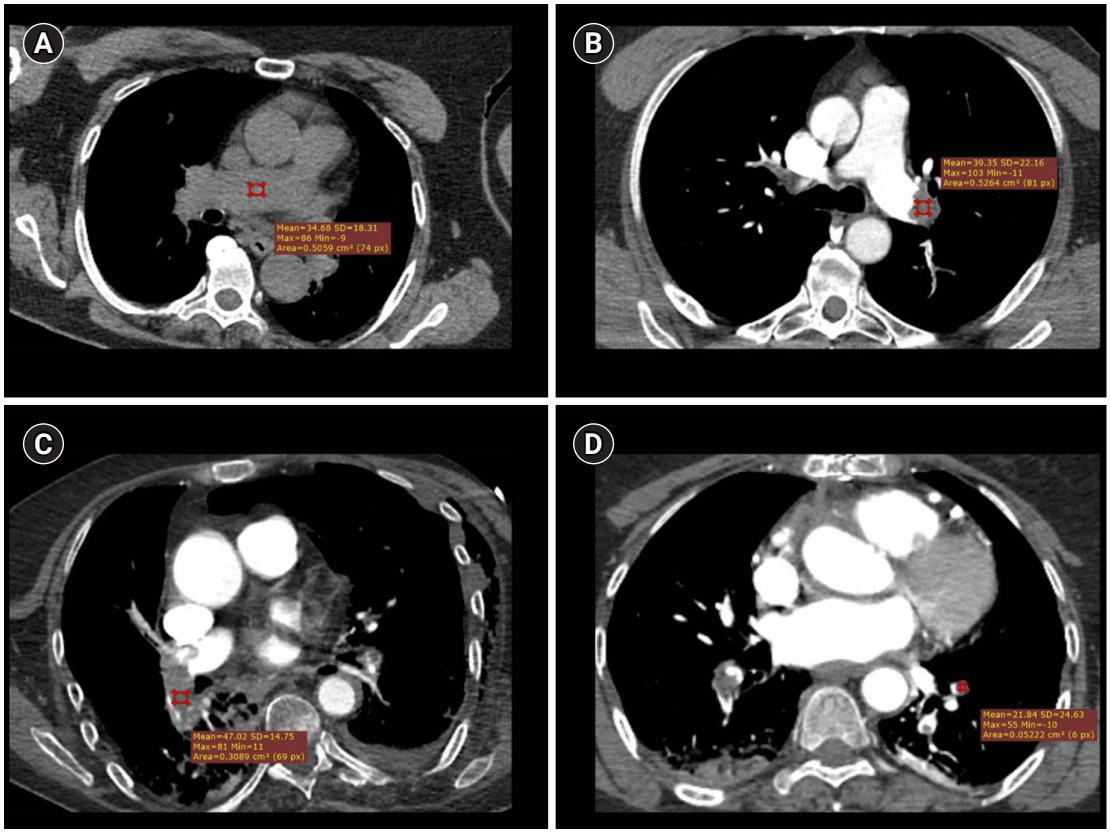

Fig. 2. Measurements of computed tomography scans. (A) Hounsfield unit (HU) density measurement in noncontrast computed tomography. (B) HU density measurement of left main pulmonary artery thrombus. (C) HU density measurement of right interlobar artery thrombus. (D) HU density measurement of left lower lobe segmental branches thrombus.

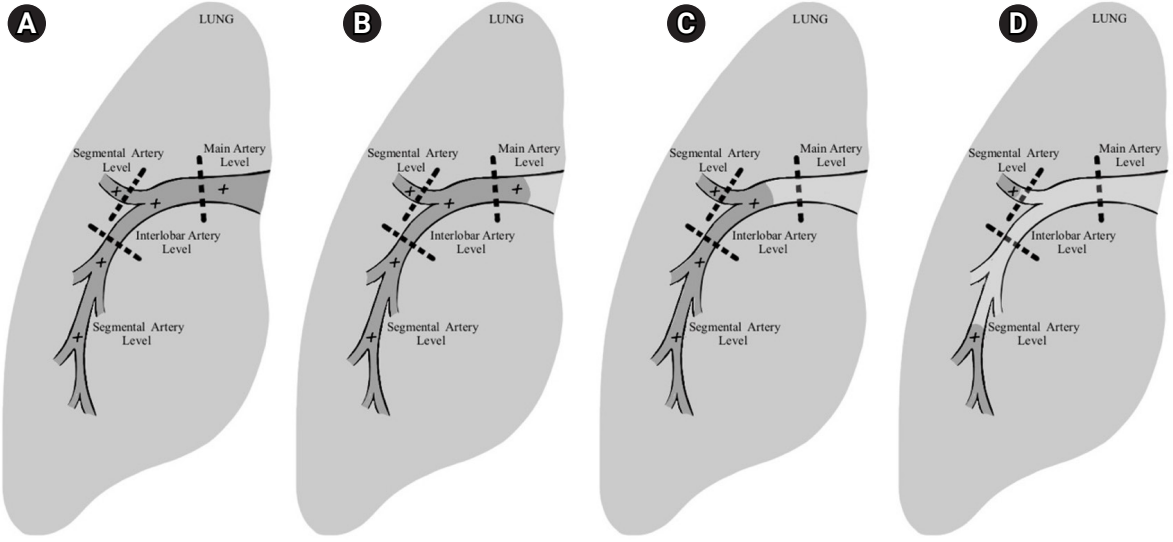

Fig. 3. Illustrations of measurements. (A) A noncontrast thorax computed tomography (CT) evaluation was performed, starting from the main pulmonary artery (MPA) to the distal segmental branches. For standardization of measurements, region of interest (ROI) size of 0.5 cm^2 was used for the MPA and right and left MPAs; ROI size of 0.3 cm^2 for the right and left interlobar arteries; ROI size of 0.05 cm^2 for the right upper, middle, and lower lobe segmental branches and left upper and lower segmental branches. (B–D) In CT pulmonary angiography evaluation, assessment was performed starting from the adjacent part where there was no contrast passage to the distal segmental branches. Similar-sized ROIs were used for the same segments as in noncontrast thorax CT evaluation.

parisons. The Shapiro-Wilk test was applied to evaluate whether the data were normally distributed. Continuous variables were expressed as mean and standard deviation (range, minimum–maximum) if they followed a normal distribution. Continuous variables were expressed as median and interquartile range (IQR) if they did not follow a normal distribution. Categorical data are represented as the frequency and percentage. In comparing the continuous variables, groups with normal distribution were compared with the t-test, and those lacking such a distribution were compared with the Mann-Whitney U-test. The chi-square test was used to compare the categorical variables between groups. A receiver operating curve (ROC) was produced to determine the cutoff levels of HU values for PE for the RMPA, LMPA, RILA, and LILA. Youden index (maximum value) in ROC analysis was used to select the cutoff value. Finally, sensitivity, specificity, likelihood ratios (+LR and –LR), and positive and negative predictive values were calculated for the RMPA, LMPA, RILA, and LILA HU values.

RESULTS

The study population included 74 patients, which fulfilled the needed 46 (62.2%) in the study group and 28 (37.8%) in the control group. Among the patients, 29 (39.2%) were men and 45 (60.8%) were women. The median age of the patients was 74 years (IQR, 66–81 years). The patients in the study were similar in age and sex distribution in the two groups. The most common comorbid diseases were hypertension (70.3%) and stroke (20.3%), and the most common admission symptoms at the ED were dyspnea (32.4%) and chest pain (21.6%). The mean hematocrit of the patients was 38.5% (range, 25.4%–54.0%). The patients in the two groups had similar hematocrit values. Patient demographic data, admission symptoms, and hematocrit values are shown in Table 1.

In contrast-enhanced CT, HU measurements were made in RMPA 19, LMPA 20, RILA 18, LILA 16, RULSB 5, RMLSB 6, RLLSB 4, LULSB 5, and LLLSB 4 in areas without contrast transmission. Since there was no area without contrast passage in MPA, MPA HU measurement could not be performed on contrast-enhanced CT. Similarly, HU was measured in all 28 segments on noncontrast CT. The mean HU values of noncontrast areas (thought to be a thrombus) measured in the study group and the mean HU values of noncontrast areas measured in the control group included a statistically significant difference at levels of RMPA (P = 0.006), LMPA (P = 0.005), RILA (P = 0.034), and LILA (P = 0.014). In addition, there was a significant difference in the mean HU value to hematocrit ratio in levels of RMPA (P = 0.006), LMPA (P = 0.007), RILA (P = 0.047), and LILA (P = 0.003) between the study and con-

Table 1. Patient demographic data and baseline characteristics

Characteristic	All patients (n = 74)	Study group (n = 46)	Control group (n = 28)	P-value
Sex				0.633
Male	29 (39.2)	19 (25.7)	10 (13.5)	
Female	45 (60.8)	27 (36.5)	18 (24.3)	
Age (yr)	74.0 (66.0–81.0)	76.5 (65.0–85.8)	72.0 (67.0–78.3)	0.475
Comorbidity				
Hypertension	52 (70.3)	35 (47.3)	17 (23.0)	0.161
Diabetes	9 (12.2)	5 (6.8)	4 (5.4)	0.722
Coronary artery disease	13 (17.6)	10 (13.5)	3 (4.1)	0.347
Atrial fibrillation	8 (10.8)	7 (9.4)	1 (1.4)	0.245
Stroke	15 (20.3)	12 (16.2)	3 (4.1)	0.111
Congestive heart failure	5 (6.8)	3 (4.1)	2 (2.7)	0.999
COPD	5 (6.8)	3 (4.1)	2 (2.7)	0.999
Dementia	10 (13.5)	8 (10.8)	2 (2.7)	0.301
Neoplasm	7 (9.5)	5 (6.8)	2 (2.7)	0.703
Admission symptom				0.381
Dyspnea	24 (32.4)	18 (24.3)	6 (8.1)	
Chest pain	16 (21.6)	10 (13.5)	6 (8.1)	
Syncope	9 (12.2)	5 (6.8)	4 (5.4)	
Cough	7 (9.5)	2 (2.7)	5 (6.8)	
Back pain	11 (14.8)	7 (9.4)	4 (5.4)	
Hemoptysis	7 (9.5)	4 (5.4)	3 (4.1)	
Hematocrit (%)	38.5 ± 5.6	38.1 ± 6.0	39.1 ± 4.7	0.456

Values are presented as number (%), median (interquartile range), or mean±standard deviation.
COPD, chronic obstructive pulmonary disease.

trol groups. The summary statistics of HU and HU value to hematocrit ratio between the study and control groups are shown in Table 2.

The cutoff HU values for RMPA, LMPA, RILA, and LILA were calculated to predict PE. The area under the curve (AUC) value for RMPA HU was 0.690 (95% confidence interval [CI], 0.457–0.922; P = 0.005), and the cutoff value for RMPA HU was 54.8, exhibiting 61.5% sensitivity and 96.4% specificity. The AUC value for LMPA HU was 0.736 (95% CI, 0.563–0.909; P = 0.001), and the cutoff value for LMPA HU was 55.9, exhibiting 65.0% sensitivity and 96.4% specificity. The AUC value for RILA HU was 0.615 (95% CI, 0.364–0.866; P = 0.030), and the cutoff value for RILA HU was 62.7, exhibiting 44.4% sensitivity and 96.4% specificity. The AUC value for LILA HU was 0.736 (95% CI, 0.475–0.996; P = 0.009), and the cutoff value for LILA HU was 56.7, exhibiting 60.0% sensitivity and 92.9% specificity. The cutoff values of HU of RMPA, LMPA, RILA, and LILA for PE with ROC analysis are shown in Table 3 and Fig. 4.

DISCUSSION

In the present study, we found significant differences in HU val-

Table 2. Patient HU values and HU/H statistics

Variable	All patients			Study group			Control group			P_1-value	P_2-value
	No. of measurements	HU value	HU/H	No. of measurements	HU value	HU/H	No. of measurements	HU value	HU/H		
MPA	28	43.5 ± 9.7 (24.9–61.1)	1.1 ± 0.3 (0.7–1.6)	0	-	-	28	43.5 ± 9.7 (24.9–61.1)	1.1 ± 0.3 (0.7–1.6)	-	-
RMPA	47	46.7 ± 17.8 (20.9–122)	1.2 ± 0.5 (0.5–3.4)	19	57.6 ± 27.3 (20.9–122)	1.5 ± 0.8 (0.5–3.4)	28	41.7 ± 7.3 (24.9–64.7)	1.1 ± 0.2 (0.5–1.4)	0.006*	0.006*
LMPA	48	53.4 ± 18.5 (17.5–101)	1.4 ± 0.6 (0.5–3.1)	20	62.0 ± 25.1 (17.5–101)	1.6 ± 0.8 (0.5–3.1)	28	47.2 ± 7.6 (32.6–70.3)	1.2 ± 0.2 (0.7–1.7)	0.005*	0.007*
RILA	46	50.2 ± 13.0 (27.9–89.8)	1.3 ± 0.4 (0.7–2.6)	18	58.2 ± 19.4 (37.5–89.8)	1.5 ± 0.6 (0.9–2.6)	28	47.7 ± 9.3 (27.9–62.7)	1.2 ± 0.2 (0.7–1.6)	0.034*	0.047*
LILA	44	46.8 ± 11.7 (27.2–89.1)	1.2 ± 0.3 (0.8–2.4)	16	58.3 ± 19.1 (40.2–89.1)	1.6 ± 0.5 (1.1–2.4)	28	44.7 ± 8.9 (27.2–62.1)	1.2 ± 0.2 (0.8–1.6)	0.014*	0.003*
RULSB	33	43.1 ± 13.3 (11.6–75.2)	1.1 ± 0.3 (0.4–1.6)	5	34.6 ± 32.5 (11.6–57.6)	1.0 ± 0.8 (0.4–1.6)	28	43.7 ± 12.0 (24.5–75.2)	1.1 ± 0.3 (0.7–1.6)	0.359	0.567
RMLSB	34	40.4 ± 13.3 (20.5–67.8)	1.0 ± 0.3 (0.5–1.7)	6	33.8 ± 2.3 (32.2–35.5)	0.9 ± 0.2 (0.8–1.1)	28	40.8 ± 13.7 (20.5–67.8)	1.1 ± 0.3 (0.5–1.7)	0.483	0.659
RLLSB	32	41.3 ± 14.2 (12.3–73.0)	1.1 ± 0.3 (0.4–1.7)	4	37.6 ± 19.1 (12.3–73.0)	1.0 ± 0.4 (0.4–1.5)	28	43.0 ± 11.3 (25.2–66.1)	1.1 ± 0.3 (0.5–1.7)	0.263	0.278
LULSB	33	39.5 ± 10.5 (18.7–66.7)	1.0 ± 0.3 (0.4–1.8)	5	40.5 ± 10.5 (40.5–40.5)	0.9 ± 0.3 (0.9–0.9)	28	39.5 ± 10.7 (18.7–66.7)	1.0 ± 0.3 (0.4–1.8)	0.932	0.62
LLLSB	32	39.2 ± 14.0 (15.8–85.0)	1.0 ± 0.4 (0.4–2.1)	4	47.3 ± 24.6 (25.8–85.0)	1.2 ± 0.6 (0.7–2.1)	28	37.4 ± 10.5 (15.8–54.6)	1.0 ± 0.3 (0.4–1.5)	0.120	0.13

Values are presented as number only or mean±standard deviation (range). P_1-value, P-value for HU value; P_2-value, P-value for HU/H.
HU, Hounsfield unit; HU/H, HU value to hematocrit ratio; MPA, main pulmonary artery; RMPA, right main pulmonary artery; LMPA, left main pulmonary artery; RILA, right interlobar artery; LILA, left interlobar artery; RULSB, right upper lobe segmental branch; RMLSB, right middle lobe segmental branch; RLLSB, right lower lobe segmental branch; LULSB, left upper lobe segmental branch; LLLSB, left lower lobe segmental branch.
*P<0.05.

Table 3. Receiver operating curve analysis

Variable	RMPA for PE	LMPA for PE	RILA for PE	LILA for PE
Area under the curve (95% CI)	0.690 (0.457–0.922)	0.736 (0.563–0.909)	0.615 (0.364–0.866)	0.736 (0.475–0.996)
Cutoff	54.8	55.9	62.7	56.7
Sensitivity (%) (95% CI)	61.5 (31.6–86.1)	65.0 (40.8–84.6)	44.4 (13.7–78.8)	60.0 (14.7–94.8)
Specificity (%) (95% CI)	96.4 (81.7–99.9)	96.4 (81.7–99.9)	96.4 (81.7–99.9)	92.9 (76.5–99.1)
+Likelihood ratio (95% CI)	17.3 (2.4–123.8)	18.2 (2.6–128.1)	12.4 (1.6–97.5)	8.4 (1.9–38.2)
–Likelihood ratio (95% CI)	0.4 (0.2–0.8)	0.4 (0.2–0.7)	0.6 (0.3–1.0)	0.4 (0.2–1.3)
Positive predictive value (%) (95% CI)	88.9 (52.7–98.3)	92.9 (64.9–98.9)	80.0 (33.8–96.9)	60.0 (24.8–87.2)
Negative predictive value (%) (95% CI)	84.4 (73.1–91.5)	79.4 (67.9–87.6)	84.4 (75.0–90.7)	92.9 (81.6–97.5)
Accuracy (%) (95% CI)	85.4 (70.8–94.4)	83.3 (69.8–92.5)	83.8 (68.0–93.8)	87.9 (71.8–96.6)

RMPA, right main pulmonary artery; PE, pulmonary embolism; LMPA, left main pulmonary artery; RILA, right interlobar artery; LILA, left interlobar artery; CI, confidence interval.

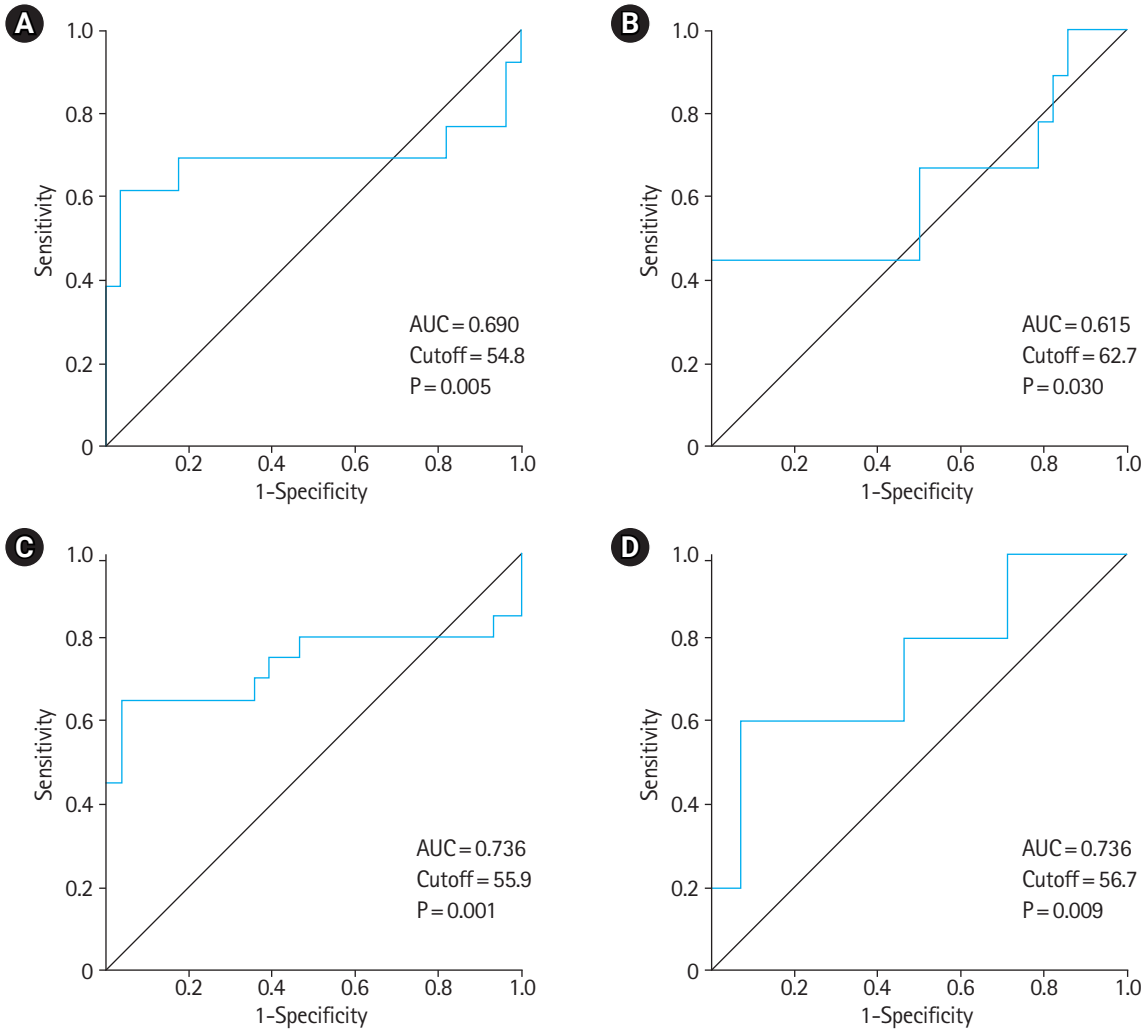

Fig. 4. Receiver operating curve. (A) Right main pulmonary artery for pulmonary embolism. (B) Right interlobar artery for pulmonary embolism. (C) Left main pulmonary artery for pulmonary embolism. (D) Left interlobar artery for pulmonary embolism. AUC, area under the curve.

ues at the RMPA, LMPA, RILA, and LILA levels. Between the study and control groups, there were statistically significant differences in HU values at levels of RMPA (57.6 vs. 41.7, P = 0.006), LMPA (62.0 vs. 47.2, P = 0.005), RILA (58.2 vs. 47.7, P = 0.034), and LILA (58.3 vs. 44.7, P = 0.014). However, there were no statistically significant differences in HU values between groups at the RULSB, RMLSB, RLLSB, LULSB, and LLLSB levels. Our data showed a statistically significant difference up to the level of the interlobar branch between the mean HU values of noncontrast areas (thought to be a thrombus) measured in the study group and the mean HU values of noncontrast areas measured in the control group. Another conclusion is that pulmonary thrombus may cause an increase in HU value, as seen in other thrombus studies [10,15].

In a previous study, Besachio et al. [16] examined the value of HU on noncontrast CT in diagnosing cerebral venous thrombosis. They found that when HU threshold values greater than 65 and a HU to hematocrit ratio greater than 1.7 were applied alone or in combination, most cases of venous thrombosis could be identified on a noncontrast head CT. Their study concluded that absolute HU values and the HU to hematocrit ratio might be helpful in noncontrast head CT evaluation of cerebral venous thrombosis. Likewise, Kim et al. [15] evaluated the HU value of deep femoral vein thrombosis before and after contrast for PE prediction. In a study of 94 patients, the HU value in the DVT-PE group was 53.5 before contrast and 67 after (P < 0.001), whereas the HU value in the DVT alone group was 44.1 before contrast and 57.1 after (P < 0.001). The study concluded that HU value intensity on before and after contrast CT may be a predictive factor for PE.

Jung et al. [17] investigated the value of the DVT HU value in

predicting PE on lower extremity venous CT. In ROC analysis, the AUC for the cutoff value of 63.0 for HU was 0.737, sensitivity was 72.2%, and specificity was 66.7%. As a result, they concluded that high HU values in lower extremity venous CTs may be predictive for PE. In a study by Alharbi and Alahmadi [18], the HU value and the HU value to hematocrit ratio were evaluated in acute cerebral venous sinus thrombus. The HU value of 56 had 100% sensitivity and specificity in the diagnosis. The HU to hematocrit ratio of 1.48 had 100% sensitivity and 65% specificity; the HU to hematocrit ratio of 1.77 had 85% sensitivity and 90% specificity; and the HU to hematocrit ratio of 1.88 was found to have 79% sensitivity and 93% specificity in the diagnosis. The HU value and its normalized ratio to hematocrit may be a diagnostic tool for acute cerebral venous thrombosis. In our study, the cutoff value for RMPA HU value to predict PE was 54.8, with a sensitivity of 61.5% and a specificity of 96.4%; the cutoff value for LMPA HU value was 55.9, with a sensitivity of 65.0% and a specificity of 96.4%; the cutoff value for RILA HU value was 62.7, with a sensitivity of 44.4% and a specificity of 96.4%; and the cutoff value for LILA HU value was 56.7, with a sensitivity of 60.0% and a specificity of 92.9%. According to our findings, the HU value up to the interlobar level may be a diagnostic tool with high specificity for diagnosing PE. Furthermore, our study data suggest that, for diagnosing PE, the use of the HU value in lower segmental branches seems inappropriate.

We found statistically significant differences in the HU value to hematocrit ratio between the study and control groups at the levels of RMPA (1.5 vs. 1.1, P=0.006), LMPA (1.6 vs. 1.2, P=0.007), RILA (1.5 vs. 1.2, P=0.047), and LILA (1.6 vs. 1.2, P=0.003). Similar hematocrit ratios between the two groups may have caused similar statistical differences at the same arterial levels. As a result, we can say that there is a difference between the study and control groups in terms of the HU value to hematocrit ratio up to the level of the interlobar branch.

The HU value and the HU value to hematocrit ratio were significant up to the interlobar level in both groups in our study. The fact that thrombi in the lower segments did not cause a statistically significant difference may be due to the few segmental emboli present and the shrinking measurement area, making it impossible to make a sufficiently sensitive evaluation.

There are some limitations to this study. In particular, the study was small in scope, single-centered, and retrospective. In addition, and similar to other retrospective studies, there was concern over the possibility of selection bias. However, to eliminate this concern, the study groups were formed by excluding factors that may cause HU differences and cases with images that may cause measurement bias. Another limitation was related to HU measurement, which can vary depending on the measurer and the measurement site, which is a limitation of the study regarding reproducibility. Also, the fact that a single radiologist performed the measurements is a limitation. There was also difficulty measuring HU values, especially in segmental branches. The thrombi in segmental branches may not be detected, and a clinical case of PE may be missed. However, it does not change the fact that clinically, noncontrast CT may be helpful as an indirect diagnostic tool in detecting thrombi up to the interlobar level. Finally, we accepted that the measured HU values were normal pulmonary artery HU values because we thought that there was no pulmonary thromboembolism clot starting from the main pulmonary to distal branches on noncontrast CT. Likewise, we accepted that HU values measured after pulmonary thromboembolism clot could be either thrombus or normal HU values. Since we could not make this distinction clearly, we wanted to state this as a limitation of the study. Further studies with more patients at more centers are needed to confirm our findings.

In conclusion, in cases of PE, HU values may exhibit high diagnostic specificity on CT, especially for thrombi up to the interlobar level. An HU value greater than 54.8 up to the interlobar level may serve as an alert for the presence of PE.

ARTICLE INFORMATION

Author contributions

Conceptualization: MMY, AÇ; Data curation: MMY, SS; Formal analysis: MMY, SS, NH; Investigation: ÖY, MMY; Methodology: AÇ, MMY; Supervision: ÖY, NH; Writing–original draft: MMY, AÇ; Writing–review & editing: all authors. All authors read and approved the final manuscript.

Conflicts of interest

The authors have no conflicts of interest to declare.

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Data availability

Data analyzed in this study are available from the corresponding author upon reasonable request.

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