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Predictors of residual thrombus in patients with pulmonary embolism

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Abstract:

BACKGROUND AND AIM: Residual thrombus is a common sequela of acute pulmonary thromboembolism (PTE), potentially contributing to long-term complications such as chronic thromboembolic pulmonary hypertension or persistent dyspnea in the absence of pulmonary hypertension. This study aimed to identify baseline clinical and radiological predictors of residual thrombus in patients treated for PTE, with a specific focus on thrombus density measured by computed tomography (CT).

METHODS: In this single-center retrospective cohort study, 103 patients who received anticoagulation therapy for at least three months following a PTE diagnosis were included. Patients with malignancy, pulmonary hypertension, antiphospholipid syndrome, or recurrent embolism were excluded. Clinical variables, treatment details, and radiologic parameters, including thrombus burden (Qanadli score) and density (Hounsfield units, HU), were recorded. Residual thrombus was evaluated by follow-up pulmonary CT angiography. Multivariate logistic regression was used to identify independent predictors.

RESULTS: Residual thrombus was detected in 30 of 103 patients (29.1%). Chronic obstructive pulmonary disease (COPD) (odds ratio [OR]=6.29; p=0.017) and right ventricular dysfunction (RVD) (OR=8.01; p=0.026) emerged as independent predictors of thrombus persistence. No significant association was found between residual thrombus and initial Qanadli score or HU values.

CONCLUSIONS: Chronic obstructive pulmonary disease and right ventricular dysfunction are key predictors of residual thrombus after acute PTE, whereas baseline thrombus density and burden do not appear to influence thrombus resolution. These findings highlight the importance of individual clinical and hemodynamic factors over radiologic metrics in identifying high-risk patients who may benefit from closer follow-up.

Keywords:

Hounsfield unit, pulmonary thromboembolism, residual thrombus

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Introduction

Acute pulmonary thromboembolism (PTE) is a critical condition with high mortality, usually caused by deep vein thrombosis (DVT) in the lower extremities.[1] Beyond its mortality risk, complications arising during the chronic phase are

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also significant. This condition, commonly referred to as post-pulmonary embolic syndrome, includes chronic thromboembolic pulmonary hypertension (CTEPH) accompanied by persistent dyspnea and residual thrombus, as well as cases of residual thrombus without pulmonary hypertension. Identifying predictors of these two important conditions at the time of PTE diagnosis is crucial. The risk factors for CTEPH are relatively well known; however, studies on the risk factors for the development of residual thrombus, particularly in patients without pulmonary hypertension, remain limited.

Residual perfusion defects following PTE can be observed in 25–50% of patients.[2] The 2022 European Society of Cardiology (ESC) and European Respiratory Society (ERS) guidelines for the diagnosis and treatment of pulmonary hypertension recommend lifelong anticoagulation for patients with residual thrombus without pulmonary hypertension, particularly if they are at high risk of PTE recurrence.[3] To date, studies investigating potential risk factors for residual thrombus have demonstrated associations with initial D-dimer levels at the time of diagnosis, age ≥65 years, unprovoked PTE, and chronic respiratory failure.[4] Additionally, various laboratory markers have been evaluated for their prognostic value in PTE. For example, red cell distribution width (RDW) has been shown to differentiate low- and moderate-risk groups in hemodynamically stable patients, with higher RDW levels associated with increased in-hospital mortality.[5] Although the relationship between initial thrombus burden and residual thrombus has been demonstrated, there is evidence that the nature of the thrombus may also contribute to this process.[6] Fibrin properties have been identified as a novel risk factor, particularly in young and middle-aged patients with unexplained arterial and venous thrombotic events.[7] In patients with malignancies, denser and stiffer clots that are relatively resistant to lysis are often observed, and these patients are known to have higher rates of residual thrombus formation.[8]

Although the nature of the thrombus is classically determined by histopathological analysis, studies have suggested a correlation between the radiological density of thrombi detected through imaging methods and these histopathological characteristics.[9] It is well established that thrombi with higher red blood cell content exhibit greater Hounsfield unit (HU) values.[9,10] Previous studies have demonstrated that thrombi with higher HU values on computed tomography (CT) imaging are more susceptible to intravenous thrombolysis or mechanical thrombectomy, suggesting that HU-based radiological assessment may serve as a non-invasive surrogate for thrombus composition and treatment response prediction.[11]

In parallel, thrombus burden has not been reported to have a decisive effect on determining the duration of treatment, but thrombus density may play a crucial role in residual thrombus development and in guiding treatment duration. To our knowledge, there is no data evaluating the relationship between thrombus density and residual thrombus in patients with pulmonary thromboembolism. This study aimed to explore the clinical and radiological characteristics associated with the persistence of residual thrombus in patients who completed at least three months of anticoagulant therapy for acute PTE, including an evaluation of the potential relationship between radiological thrombus density and thrombus persistence.

Materials and Methods

Study design and population

This single-center, retrospective cohort study was conducted at Yedikule Chest Diseases and Thoracic Surgery Training and Research Hospital between January 2022 and June 2024 on adult patients diagnosed with PTE through pulmonary CT angiography who received the necessary treatment for at least three months. The study was conducted in accordance with the ethical principles of the Declaration of Helsinki and was approved by the University of Health Sciences Hamidiye Scientific Research Ethics Board (Approval Number: 12/29, Date: 17.10.2024). The requirement for written informed consent was waived due to the retrospective nature of the study. Patients with a diagnosis of pulmonary hypertension, malignancy, antiphospholipid antibody syndrome, or recurrent embolism were excluded from the study. Patients with chronic obstructive pulmonary disease (COPD) were included only if they did not meet the diagnostic criteria for pulmonary hypertension according to echocardiographic and clinical evaluation, in line with the 2022 ESC/ERS guidelines.[3] Additionally, patients with high early mortality risk per the 2019 ESC/ERS PTE severity classification,[12] as well as those who received thrombolysis, were excluded.

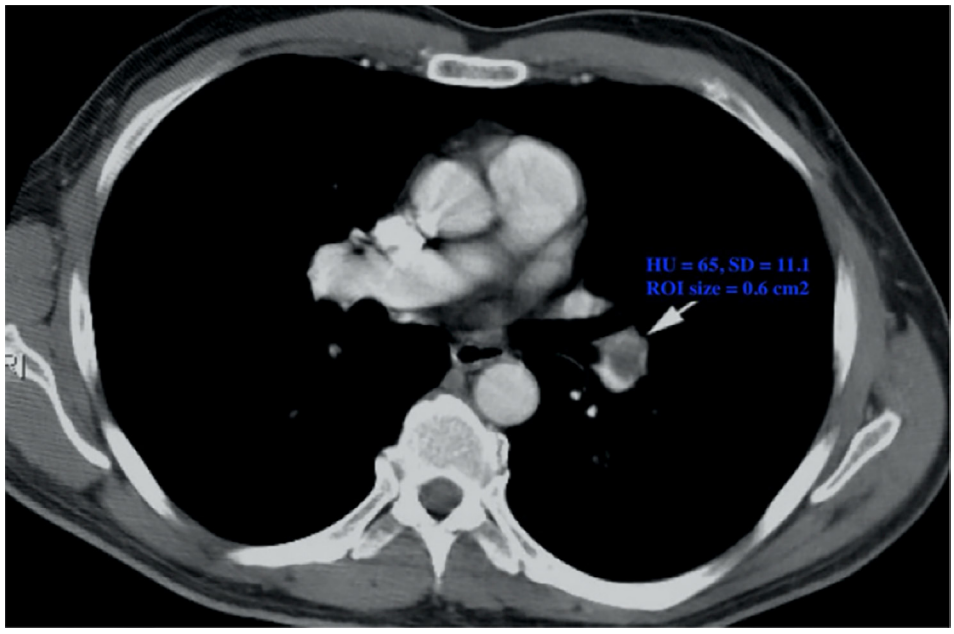

Figure 1: Hounsfield unit (HU) density measurement in computed tomography. Circular regions of interest (ROIs) were placed within the most homogeneous and centrally located part of the thrombus on axial computed tomography (CT) slices

Data collection

Data extracted from the hospital's electronic medical records included patients' age, sex, comorbidities, anticoagulant treatment received, duration of treatment, presence of residual thrombus after six months of treatment (evaluated by pulmonary CT angiography), tricuspid jet flow velocity measured by echocardiography at six months, and the presence of chronic thromboembolic disease or CTEPH at six months. Thrombus density at diagnosis and, in patients with residual thrombus, after treatment, as well as thrombus burden on pulmonary CT angiography, were assessed by a radiologist with 20 years of experience.

Definitions

Hounsfield unit is a standardized, quantitative measure of radiodensity that facilitates the assessment and interpretation of CT images by radiologists, contributing to enhanced diagnostic accuracy. Measurements were performed by a single experienced radiologist who was blinded to the clinical outcomes. For each thrombus, attenuation values were measured in HU by placing circular regions of interest within the most homogeneous and centrally located part of the thrombus on axial CT slices [Fig. 1]. Measurements were taken from at least three different slices per thrombus, and the average value was used for analysis.[13] This approach ensured consistency in radiodensity assessment and minimized the influence of partial volume effects.

Right ventricular dysfunction (RVD) was defined according to the 2019 ESC/ERS Guidelines for the Diagnosis and Management of Pulmonary Hypertension. In line with guideline recommendations, RVD was considered present if at least one of the following echocardiographic findings was identified: tricuspid annular plane systolic excursion (TAPSE) <17 mm, right ventricular dilatation (right/left ventricular area ratio >1), interventricular septal flattening, or elevated pulmonary artery pressure estimated by a tricuspid regurgitation velocity (TRV) >2.8 m/s.[12]

Outcomes

The primary outcome of this study was the identification of clinical, laboratory, and radiologic factors associated with the persistence of residual thrombus in patients who completed at least three months of anticoagulant therapy for acute PTE. Residual thrombus was assessed by follow-up pulmonary CT angiography. All patients underwent imaging at a fixed six-month interval without variation in timing. The secondary outcome was to evaluate the potential relationship between initial thrombus density, measured in HU on pulmonary CT angiography, and the likelihood of residual thrombus formation.

Data analysis and statistical methods

Statistical analyses of the findings obtained in this study were performed using IBM SPSS Statistics, version 25 (Armonk, NY, IBM Corp.). The normality of parameter

distributions was assessed with the Shapiro-Wilk test. Descriptive statistics were applied, and for normally distributed parameters, the mean and standard deviation were reported, while for non-normally distributed parameters, the median and interquartile range were provided. Categorical variables were expressed as frequencies and percentages. To identify predictors of residual thrombus, categorical data were analyzed using the chi-square test, continuous variables with a normal distribution were compared using the Student's t-test, and non-normally distributed variables were assessed with the Mann-Whitney U test. In the univariate analysis, parameters that were statistically significant at $p<0.2$ or deemed clinically relevant were included in the logistic regression model. Correlations between continuous variables included in the multivariate analysis were examined, and if a high correlation ($r>0.7$) was observed between any variables, the parameter considered to have greater clinical significance was retained for further analysis. The goodness of fit for the logistic regression model was evaluated using the Hosmer-Lemeshow test, with a p-value>0.05 indicating an acceptable fit. Values of $p<0.05$ were considered statistically significant.

Results

A total of 103 patients were included in the study, with a mean age of 50.4 years; 40.8% of the cohort were female. The most frequently observed comorbidities were hypertension (29.1%), diabetes mellitus (15.5%), and chronic obstructive pulmonary disease (11.7%). Right ventricular dysfunction was identified in 7.8% of the patients, while DVT was present in 20.4% at the time of diagnosis. Regarding anticoagulant therapy, 16.5% of patients received warfarin, 19.4% were treated with low-molecular weight heparin (LMWH), and 64.1% received direct oral anticoagulants (DOACs). The mean HU value was 44.1. The median Qanadli score was 7, with an interquartile range of 3 to 13 (Table 1).

When patients with residual thrombus (n=30, 29.1%) and those without residual thrombus (n=73, 70.9%) on pulmonary CT angiography were compared, the mean ages were 51.6 and 49.9 years, respectively. Although no statistically significant differences were observed in the distribution of comorbidities between the two groups, the prevalence of COPD was significantly higher in the residual thrombus group (p=0.002). Analysis of laboratory parameters revealed no significant differences between the groups (p>0.05). The distribution of anticoagulant therapies, including warfarin, LMWH, and DOACs, was similar between patients with and without residual thrombus, with no statistically significant differences observed (p=0.23, p=0.92, and p=0.31, respectively) (Table 1).

The presence of right ventricular dysfunction was significantly more frequent in patients with residual thrombus (p=0.003). However, no statistically significant difference was observed in the prevalence of DVT at baseline between the two groups (p=0.12). Similarly, there were no significant differences between patients with and without residual thrombus in terms of Qanadli scores and HU values (p=0.20 and p=0.74, respectively) (Table 1).

Multivariate logistic regression analysis was conducted using variables that were either clinically relevant or demonstrated a p-value <0.20 in the univariate analysis. Among these, the presence of COPD (odds ratio [OR]=6.29, 95% confidence interval [CI]: 1.38–28.62, p=0.017) and right ventricular dysfunction (OR=8.01, 95% CI: 1.28–49.90, p=0.026) were identified as independent predictors of residual thrombus development. Although DVT (OR=1.83, 95% CI: 0.57–5.85, p=0.30) showed a trend toward significance, it did not reach statistical significance. Similarly, no significant associations were found for Qanadli score (OR=1.01, 95% CI: 0.93–1.09, p=0.80) or HU value (OR=1.01, 95% CI: 0.97–1.04, p=0.53) (Table 2).

Discussion

This study explored the clinical and radiological characteristics associated with the persistence of residual thrombus in patients who completed at least three months of anticoagulant therapy for acute PTE. Our findings revealed that the most distinctive features in patients with residual thrombus were the presence of COPD and right ventricular dysfunction. These two variables were identified as independent predictors of residual thrombus formation in multivariate analysis, while other parameters, including thrombus density, thrombus burden, and laboratory findings, did not show significant associations.

Residual thrombus is a frequent outcome after acute PTE, with studies reporting rates as high as 25%–50%, depending on patient selection, imaging modality, and follow-up timing.[2] The clinical relevance of residual thrombus lies in its association with long-term sequelae, both in patients with residual thrombus without pulmonary hypertension and in those with CTEPH. Both conditions can lead to progressive

Table 1: Demographic and clinical variables of patients with and without residual thrombus

Variables	All patients (n=103)		Residual thrombus + (n=30)		Residual thrombus – (n=73)		p
	n	%	n	%	n	%	
Age, years	50.4±14		51.6±12.8		49.9±14.5		0.59
Female sex	42	40.8	15	50	27	37	0.22
Comorbidity							
COPD	12	11.7	8	26.7	4	5.5	**0.002**
Asthma	10	9.7	5	16.7	5	6.8	0.12
Chronic heart failure	5	4.9	2	6.7	3	4.1	0.58
Atrial fibrillation	4	3.9	2	6.7	2	2.7	0.34
Hypertension	30	29.1	11	36.7	19	26	0.28
Coronary artery disease	9	8.7	3	10	6	8.2	0.77
Diabetes mellitus	16	15.5	6	20	10	13.7	0.42
Malignancy	8	7.8	2	6.7	6	8.2	0.78
Laboratory parameters							
D-dimer, ng/mL	84 (6–213)		52 (3.4–205)		91.5 (14.7–223)		0.37
Pro-BNP, pg/mL	81 (10–269)		114 (23–476)		65.5 (10–247.7)		0.33
Troponin, ng/mL	5 (3–9)		4 (3–9)		5.4 (3–9)		0.82
Leukocyte, $10^3/\mu L$	10.5±3.6		10.6±4.6		10.4±3.2		0.86
Hemoglobin, g/dL	13.5 (11.8–14.5)		13.1 (11.2–13.9)		13.5 (12.4–14.6)		0.19
Platelet, $10^3/\mu L$	284±89		271±77		290±93		0.34
BUN, mg/dL	25 (22–32)		25 (18.5–35.5)		26 (22–30.5)		0.75
Creatinine, mg/dL	0.76±0.18		0.78±0.23		0.76±0.16		0.59
AST, U/L	19 (14–32)		19 (13–33)		19 (24.5–31)		0.95
ALT, U/L	19 (13–29)		17 (12.2–30.2)		19 (13.5–28.5)		0.96
Treatments							
Warfarin	17	16.5	7	23.3	10	13.7	0.23
LMWH	20	19.4	6	20	14	19.2	0.92
DOACs	66	64.1	17	56.7	49	67.1	0.31
Rivaroxaban	36	35	10	33.3	26	35.6	
Apixaban	21	20.4	5	16.7	16	21.9	
Edoxaban	9	8.7	2	6.7	7	9.6	
Right ventricular dysfunction	8	7.8	6	20	2	2.7	0.003
DVT	21	20.4	9	30	12	16.4	0.12
Hounsfield unit	44.1±15.2		44.9±15.8		43.8±15.1		0.74
Qanadli score	7 (3–13)		9.5 (3–14)		6 (3–13)		0.20

Normally distributed (parametric) variables are presented as mean±standard deviation (SD); non-normally distributed (non-parametric) variables are presented as median (IQR). Statistically significant values are shown in bold. COPD: Chronic obstructive pulmonary disease, BNP: B-type natriuretic peptide, BUN: Blood urea nitrogen, AST: Aspartate aminotransferase, ALT: Alanine aminotransferase, LMWH: Low-molecular-weight heparin, DOAC: Direct oral anticoagulant, DVT: Deep vein thrombosis.

dyspnea, reduced exercise tolerance, and right ventricular failure, underscoring the importance of early identification of patients at higher risk for thrombus persistence.

The most striking result in our analysis was the strong association between COPD and residual thrombus (p=0.002). The mechanisms underlying this relationship are likely multifactorial. COPD is known to promote systemic inflammation, endothelial dysfunction, and procoagulant activity, all of which can impair thrombus resolution.[14] Previous studies have shown that patients with COPD have increased levels of fibrinogen and circulating microparticles, as well as reduced fibrinolytic activity.[15] Additionally, structural changes in the pulmonary vasculature and hypoxic vasoconstriction in COPD may further impede thrombus clearance.[16] A recent study by Ząbczyk et al.[17]

Table 2: Multivariate logistic regression analysis demonstrating independent predictors of residual thrombus development

Variables	OR	CI (95%)	p
COPD	6.52	1.43–29.69	0.015
Right ventricular dysfunction	7.20	1.19–43.4	0.031
DVT	1.82	0.57–5.73	0.30
Qanadli Score	1.01	0.94–1.09	0.67

Hosmer-Lemeshow goodness-of-fit test: p=0.38. OR: Odds ratio, CI: Confidence interval, COPD: Chronic obstructive pulmonary disease, DVT: Deep vein thrombosis

emphasized that patients with chronic lung disease tend to generate dense and stiff fibrin clots that are less susceptible to enzymatic lysis, a factor that may explain our findings.

Right ventricular dysfunction was another independent predictor of residual thrombus (p=0.026). Right ventricular dysfunction reflects increased pulmonary artery pressures and is often indicative of a more extensive embolic burden at baseline.[18] Sustained right-sided pressure overload may impair pulmonary perfusion, limiting the endogenous mechanisms responsible for thrombus degradation. Furthermore, right ventricular dysfunction could serve as a surrogate for delayed hemodynamic recovery and compromised vascular remodeling, both of which may hinder thrombus resolution. Prior studies have also shown that right ventricular dysfunction at the time of PTE diagnosis is associated with poor long-term outcomes, including persistent perfusion defects and reduced functional capacity.[19]

Although the presence of DVT did not reach statistical significance, it was more frequent among patients with residual thrombus (30% vs. 16.4%). This trend suggests that DVT may reflect a more extensive or systemic thrombotic phenotype. Other studies have also indicated that concomitant DVT may increase thrombus volume and promote clot propagation, thereby increasing the likelihood of residual thrombus.[20]

Notably, thrombus burden, as assessed by the Qanadli score, and thrombus density, measured in HU, did not differ significantly between the groups. The lack of association between thrombus burden and thrombus persistence may indicate that anatomical extent alone is insufficient to predict clot resolution. Although HU has been proposed as a non-invasive indicator of thrombus composition, particularly in ischemic stroke, its value in predicting long-term outcomes in PTE remains uncertain.[21] In our cohort, the mean HU values were nearly identical between the two groups, suggesting that thrombus radiodensity may not capture the complex interplay of biological, inflammatory, and hemodynamic factors that influence thrombus persistence.

In terms of anticoagulant therapy, we did not observe any differences in the distribution of warfarin, LMWH, or DOACs between the groups. While some data suggest differences in fibrin structure or thrombus remodeling depending on the anticoagulant used, our results indicate that in real-world clinical practice, the type of anticoagulation may be less important than patient-specific factors.[22] Early recognition of high-risk subgroups, particularly patients with COPD or right ventricular dysfunction, has important clinical implications. These patients may benefit from shorter follow-up intervals and individualized imaging schedules to detect persistent thrombus earlier. Moreover, careful reassessment of Prolonged anticoagulation or closer monitoring may be warranted to mitigate long-term complications.

This study has several limitations that should be acknowledged. First, its retrospective design may have introduced selection bias and limited the ability to establish causal relationships. Second, the study was conducted at a single center with a relatively modest sample size, which may restrict the generalizability of the findings. The limited sample size may also have reduced the statistical power of the analyses, potentially obscuring smaller effect sizes, particularly in the evaluation of thrombus density. Third, the assessment of thrombus density was based on radiologic measurements performed by a single radiologist, which, although blinded to outcomes, may still carry observer-related variability. Interobserver variability was not assessed and should be considered an additional limitation of our study. Another limitation of our study is the lack of detailed anatomical classification of residual thrombus based on its location within the pulmonary arterial tree (e.g., main, lobar, or segmental arteries). This limitation may have provided additional insights into thrombus resolution patterns and their clinical significance. In addition, specific clot properties, such as histopathological characteristics and fibrin content, could not be evaluated. Another methodological consideration is that all follow-up imaging was performed at a fixed six-month interval.

Conclusion

This study identified COPD and right ventricular dysfunction as independent predictors of residual thrombus in patients treated for acute PTE. These findings suggest that patient-specific clinical and hemodynamic characteristics may play a more critical role in thrombus persistence than radiological parameters such as thrombus burden or density. Early recognition of high-risk subgroups, particularly those with chronic lung disease or right ventricular dysfunction, may help guide individualized follow-up strategies and optimize treatment plans. Further prospective, multicenter studies with larger cohorts and longer follow-up are warranted to validate these findings and explore additional predictive markers.

Ethics Committee Approval

The study was approved by the University of Health Sciences Hamidiye Scientific Research Ethics Committee (No: 12/29, Date: 17/10/2024).

Informed Consent

The requirement for written informed consent was waived due to the retrospective nature of the study.

Conflicts of Interest Statement

The authors have no conflicts of interest to declare.

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No AI technologies utilized.

Author Contributions

Concept – A.E.; Design – A.E., C.S.; Supervision – C.S.; Resource – A.E., A.K.; Materials – A.E.; Data Collection and/or Processing - A.E., A.K.; Analysis and/or Interpretation - A.K., F.Ç.; Literature Review – A.K., F.Ç.; Writing – A.E., A.K., C.S..; Critical Review – A.E., C.S.

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