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Original article

CT PULMONARY ANGIOGRAPHY IN ACUTE PULMONARY EMBOLISM: THE IMPACT OF CONCOMITANT DEEP VEIN THROMBOSIS ON THE IMAGING BIOMARKERS OF RIGHT HEART STRAIN

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Abstract

Objective: To assess the influence of concomitant deep vein thrombosis (DVT) on CT pulmonary angiography (CTPA) markers of right heart strain (RHS) in patients with acute pulmonary embolism (APE).

Material and Methods: A retrospective analysis was conducted on 119 patients diagnosed with APE using CTPA. Key imaging parameters evaluated included the pulmonary artery obstruction index (PAOI), diameters of the right and left atria and ventricles, pulmonary artery (PA) and aorta (Ao), the RV/LV and RA/LA diameter ratios, interventricular septal (IVS) deviation, and inferior vena cava (IVC) contrast reflux. Patients were stratified based on the presence of DVT confirmed by clinical symptoms or ultrasound.

Results: DVT was identified in 49 of 119 patients (41.2%). Compared to those without DVT, this group demonstrated significantly elevated PAOI values (61.5 ± 29.2 *vs*. 49.3 ± 26.3; $p = 0.023$), larger RA/LA diameter ratios (1.44 ± 0.44 *vs*. 1.25 ± 0.42; $p = 0.016$), and higher RV/LV ratios (1.49 ± 0.53 *vs*. 1.24 ± 0.46; $p = 0.017$). While PA/Ao ratios and IVC diameters were numerically greater in the DVT group, differences were not statistically significant. IVS shift and IVC contrast reflux were more frequently observed in the DVT subgroup (53.1% and 69.4%, respectively), although without statistical significance ($p > 0.5$).

Conclusions: The presence of DVT in patients with acute PE is associated with more pronounced CTPA markers of right heart strain, particularly elevated PAOI and increased ventricular and atrial diameter ratios. These findings suggest a greater hemodynamic burden in PE patients with concomitant DVT. CTPA evaluation of RHS markers may aid in risk stratification and clinical decision-making in this population.

Keywords: CT pulmonary angiography, acute pulmonary embolism, deep vein thrombosis, right heart strain, PAOI, RV/LV ratio, IVC reflux

Introduction

Acute pulmonary embolism (APE) is a critical clinical manifestation of venous thromboembolism (VTE), frequently presenting as a life-threatening complication of deep vein thrombosis (DVT)[1,2]. Together, PE and DVT form a pathophysiological continuum and are considered two interrelated expressions of the same disease process -VTE -which ranks as the third

most common cause of acute cardiovascular morbidity and mortality after myocardial infarction and stroke[3]. Despite advancements in diagnostic imaging and therapeutic strategies over the past three decades, the early mortality associated with APE has remained largely unchanged[4]. Autopsy data suggest that a significant proportion of fatal cases remain undiagnosed during life, underscoring the persistent challenges in timely diagnosis[5].

In approximately 65-70% of cases, pulmonary emboli originate from thrombi located in the deep venous system of the lower extremities or pelvis[6]. The embolic occlusion of pulmonary arteries precipitates a cascade of pathophysiological responses, including increased alveolar dead space, ventilation-perfusion mismatch, arterial hypoxemia, and pulmonary infarction[7]. The most critical hemodynamic consequence is the abrupt elevation in pulmonary vascular resistance, resulting in right ventricular (RV) strain and, potentially, acute RV failure - a condition associated with poor short-term outcomes, including cardiogenic shock and sudden death[8,9].

The clinical presentation of APE is highly variable, ranging from mild dyspnea to sudden circulatory collapse, and may be entirely asymptomatic, further complicating diagnosis[10]. Given these diagnostic challenges, imaging plays a pivotal role in both confirming the presence of emboli and stratifying clinical risk. Computed tomography pulmonary angiography (CTPA) has emerged as the gold standard imaging modality due to its high sensitivity and specificity, rapid acquisition time, and capability to visualize not only intravascular thrombi but also secondary signs of right heart strain (RHS)[11,12].

CTPA-derived markers, such as the pulmonary artery obstruction index (PAOI), right-to-left ventricular (RV/LV) diameter ratio, right-to-left atrial (RA/LA) diameter ratio, interventricular septal deviation, and contrast reflux into the inferior vena cava (IVC), are widely validated indicators of hemodynamic impairment in APE and have been associated with increased risk of adverse clinical outcomes[13,14]. These imaging parameters now play a critical role in prognostic stratification and may guide decisions regarding intensive therapy or hospital admission[15].

Given the anatomical and physiological link between DVT and PE, it is plausible that the presence of DVT may reflect a more extensive thrombotic burden and contribute to increased embolic load, thereby worsening RHS markers on CTPA. However, the impact of concomitant DVT on these imaging indicators remains under-investigated and somewhat controversial[16,17].

This study aimed to assess the influence of confirmed DVT on CTPA-based RHS markers in patients with acute PE, with the objective of enhancing our understanding of the interplay between these conditions and contributing to the refinement of diagnostic and prognostic protocols in clinical practice.

Material and methods

This was a cross-sectional analytical study conducted at the University Institute of Radiology in Skopje. The study involved retrospective review and detailed radiological analysis of computed tomography pulmonary angiography (CTPA) scans in patients with confirmed acute pulmonary embolism (APE). Echocardiographic findings and clinical data were obtained from patient medical records and supplemented by a structured questionnaire administered at the time of imaging.

The cohort comprised 119 consecutive patients, aged 19 to 91 years, referred with a working diagnosis of APE. All patients had undergone echocardiographic evaluation within 48 hours prior to or following initial CTPA and completed both baseline and follow-up CTPA scans. Participants were stratified based on the presence or absence of deep vein thrombosis (DVT), as clinically or sonographically confirmed.

Inclusion Criteria

- Age 19-91 years
- Positive CTPA findings for acute PE
- Available transthoracic echocardiogram (TTE) within 48 hours of CTPA
- Informed consent to participate in the study

Exclusion Criteria

- Age <19 or >91 years
- Absence of echocardiographic data
- Known pre-existing cardiac or pulmonary diseases (e.g., chronic thromboembolic disease, chronic pulmonary hypertension)
- Suboptimal or non-diagnostic CTPA image quality

All CTPA examinations were performed using a 128-slice Somatom Definition AS+ scanner (Siemens Healthineers). A bolus of 70-100 mL of iodinated contrast (300 mg/mL) was injected into the left antecubital vein at 3-5 mL/sec, using automated bolus tracking. Scans were acquired caudo-cranially during deep inspiration, with 1 mm slice thickness and 0.5 mm reconstruction intervals. Optimal opacification of the pulmonary arteries was defined by attenuation values between 210-300 Hounsfield units (HU).

Images were post-processed on a dedicated workstation with multi-planar reformats and evaluated by a thoracic radiologist with more than 11 years of experience. Quantification included calculation of the pulmonary artery obstruction index (PAOI) using the Qanadli scoring method, and assessment of right heart strain (RHS) markers: RV and LV diameters, RA/LA ratios, PA/Ao ratio, superior vena cava (SVC) diameter, interventricular septal shift, and IVC contrast reflux.

Echocardiographic data were used to confirm right ventricular dysfunction (RVD), defined by at least one of the following criteria: RV dilatation (end-diastolic diameter >30 mm), RV/LV ratio >1, interventricular septal flattening, RV hypokinesia, or estimated pulmonary hypertension.

All patients completed a standardized questionnaire documenting symptom onset, risk factors, and comorbidities. Imaging parameters and clinical data were recorded in a structured form. Statistical analyses were performed using SPSS v. 26.0. Descriptive and inferential statistics included: Chi-square tests, Mann-Whitney U, Wilcoxon tests, Pearson and point-biserial correlation, and ROC curve analysis. A p-value of <0.05 was considered statistically significant.

Results

A total of 119 patients (mean age 58.96±15.6 years; 55% female) diagnosed with acute pulmonary embolism (APE) via computed tomography pulmonary angiography (CTPA) were included. Of these, 49 patients (41.2%) had confirmed concomitant deep vein thrombosis (DVT), while 70 patients (58.8%) did not.

Patients with DVT exhibited significantly higher pulmonary artery obstruction index (PAOI) values compared to those without DVT (61.54±29.24 *vs*. 9.29±26.27; *p=0.023*) (Table 1, Table 2, Figure 1).

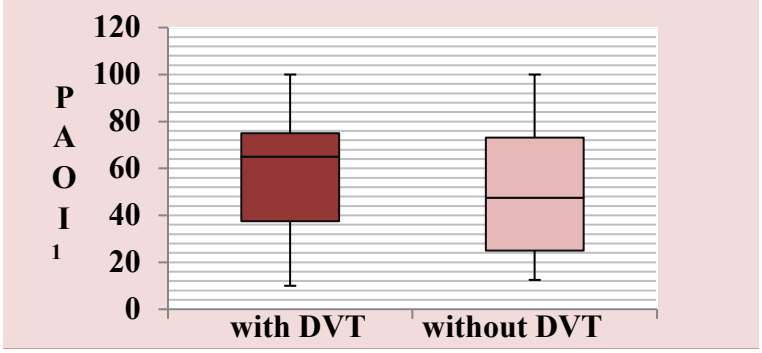

Fig. 1. Graphical Representation of PAOI in Relation to the Presence or Absence of DVT

Table 1. Differences of PAOI and CT Imaging Markers of RHF by DVT Presence

	Deep Vein Thrombosis				p-level
	With DVT (N = 49)		Without DVT (N = 70)		
	Mean (SD)	Median	Mean (SD)	Median	
PAOI	61.54 (29.24)	65.0	49.29 (26.27)	47.5	z = -2.276 p* = .023
RA	6.80 (1.90)	7.40	6.06 (1.90)	6.60	z = -2.227 p* = .026
LA	4.73(0.48)	4.70	4.81(0.47)	4.90	z = 1.169 p = .242
RA/LA	1.44(0.44)	1.60	1.25(0.42)	1.40	z = -2.411 p* = .016
RV	5.34(1.10)	5.20	4.92(0.90)	4.80	z = -2.333 p* = .020
LV	3.86(0.86)	3.50	4.26(0.90)	4.10	z = 2.497 p* = .012
RV/LV	1.49(0.53)	1.50	1.24(0.46)	1.10	z = -2.378 p* = .017
PA	3.00(0.50)	3.00	2.98(0.50)	2.90	z = –0.486 p = .624
Ao	3.26(0.50)	3.20	3.35(0.50)	3.40	z = 0.969 p = .332
PA/Ao	0.93(0.10)	0.90	0.89(0.10)	0.90	z = -1.147 p = .250
SVC	2.14(0.30)	2.10	2.03(0.30)	2.00	z = -1.901 p = .057

z (Mann Whitney U test). p* < .05

Similarly, markers of right heart strain (RHS) were more pronounced in the DVT group. Specifically, the RA/LA diameter ratio was significantly elevated (1.44±0.44 *vs*. 1.25±0.42; *p=0.016*), as was the RV/LV ratio (1.49±0.53 *vs*. 1.24±0.46; *p=0.017*). Other significant differences included larger diameters of the right atrium (6.80±1.90 mm *vs*. 6.06±1.90 mm;

p=0.026) and right ventricle (5.34±1.10 mm *vs*. 4.92±0.90 mm; *p=0.020*), and smaller left ventricular diameters in the DVT group (3.86±0.86 mm *vs*. 4.26±0.90 mm; *p=0.012*) (Table 1).

Table 2. PAOI Score Values in Patients Stratified by DVT Presence

PAOI1	Statistical parameters				
	N	mean ± SD	min-max	Median (IQR)	p-level
With DVT	49	61.54±29.24	10-100	65(37.5-75)	U = 1.293 z = -2.276
Without DVT	70	49.29±26.27	12.5-100	47.5(25-73.13)	p* = .023

z (Mann Whitney U test). p* < .05

No statistically significant differences were observed in pulmonary artery (PA) or aortic (Ao) diameters, PA/Ao ratios, or SVC diameters between the groups. Although contrast reflux into the inferior vena cava (IVC) and interventricular septal shift were more frequent in patients with DVT (69.4% and 53.1%, respectively), these associations did not reach statistical significance (*p = 0.674* and *p = 0.525*, respectively) (Table 3).

Table 3. Comparative Analysis of VCI Reflux and IVS Shift by DVT Status

	With DVT (N = 49) N (%)		Without DVT (N = 70) N (%)		p-level
VCI reflux	no	15(30.6)	no	24(34.3)	χ^2 = .1765
	yes	34(69.4)	yes	46(65.7)	p = .6744
IVS shift	no	23(46.9)	no	37(52.9)	χ^2 = .4039
	yes	26(53.1)	yes	33(47.1)	p = .5251

χ^2 (Pearson Chi-square)

There were no significant differences in age (58.0 ± 15.5 *vs*. 57.6 ± 16.0 years; *p = 0.711*), sex distribution (*p = 0.393*), or body weight (*p = 0.904*) between DVT and non-DVT groups (Table 4).

Table 4. Demographic and Anthropometric Differences by DVT Status

		With DVT N (49)		Without DVT N (70)		p-level
		Mean (SD)	Median	Mean (SD)	Median	
Gender	**male**	27 (55.10%)		33 (47.14)		χ^2 = .730
	female	22 (44.88%)		37 (52.86%)		p = .393
Age (years)		58,04 (15,51)	62	57.6 (15.96)	60	z = -0.373 p = .711
Weight (kg)		82,12 (11,57)	83	81.64 (12.21)	85	z = -0.121 p = .904

χ^2 (Pearson Chi-square), z (Mann Whitney U test)

Discussion

Pulmonary embolism (PE) and deep vein thrombosis (DVT) are pathophysiologically linked conditions within the broader clinical spectrum of venous thromboembolism (VTE). While isolated DVT typically follows a more benign course, approximately one-third of clinically

manifested DVT cases may be complicated by silent PE, highlighting the close interplay between the two entities[18,19]. Our study, which included 119 patients with acute PE confirmed by CTPA, demonstrated that 49 patients (41.2%) had concomitant DVT, diagnosed either clinically or via lower limb ultrasound. These findings are consistent with recent literature reporting concomitant DVT rates ranging from 56% to 61% in symptomatic PE[20], although earlier reports varied widely, between 10% and 93%.

A key focus of this study was the impact of coexisting DVT on CTPA-derived markers of right heart strain (RHS), an imaging correlate known to be associated with adverse clinical outcomes in PE. We observed that patients with PE and concomitant DVT had significantly higher pulmonary artery obstruction index (PAOI) values (mean 61.5 *vs*. 49.3; $p = 0.023$) and elevated RA/LA and RV/LV diameter ratios compared to those without DVT. These imaging features, indicative of acute right ventricular pressure overload, suggest a greater hemodynamic burden in patients with coexisting DVT, likely reflecting a larger or more proximal embolic source.

These results are in line with findings from the REMOTEV study by Kordeanu *et al*., which reported that patients with PE and DVT presented more frequently with severe PE ($p < 0.0001$), especially in cases with proximal DVT, although no significant differences were found in 3-month all-cause mortality or recurrence rates[21]. Similarly, the meta-analysis by Becattini *et al*., encompassing over 8,800 patients, confirmed a significantly higher 30-day mortality rate in patients with PE and DVT (6.2%) *versus* PE alone (3.8%)[20]. This supports the interpretation that concomitant DVT may act as a proxy for thrombotic load and disease severity, even if it does not consistently alter long-term prognosis.

Contrary to these findings, Gérard *et al*. reported no significant difference in three-month mortality or recurrence in a prospective study of over 1,000 patients with symptomatic PE, regardless of DVT status[22]. Their results challenge the routine screening for DVT in PE patients, although they do suggest increased utility in selected high-risk populations such as the elderly. The Montreal study also highlighted that patients with PE alone were younger and more often female, while those with coexisting DVT had higher rates of malignancy, cardiopulmonary comorbidities, and prior VTE - factors that could confound prognostic assessments[19].

In our cohort, despite a higher prevalence of IVC contrast reflux and interventricular septal shift in the DVT group (69.4% and 53.1%, respectively), these differences did not reach statistical significance. This may be attributable to sample size limitations or the inherent variability of these qualitative CTPA findings. However, the statistically significant increase in PAOI, RV and RA diameters, and RHS ratios (RV/LV, RA/LA) in the DVT subgroup strongly supports the hypothesis that DVT contributes to a more severe hemodynamic presentation of PE.

Dudzinski *et al.* examined CTPA and TTE correlations in right heart dysfunction (RHD), concluding that CTPA has high sensitivity (88%) but modest specificity (39%) for RHD compared to echocardiography. Importantly, the combination of both modalities was more predictive of early clinical deterioration than either alone[23]. In our study, echocardiographic evidence of RV dysfunction was available and corroborated with CTPA findings, enhancing the validity of our results.

The heterogeneity in the literature regarding the prognostic implications of DVT in PE highlights the complexity of this association. While some studies suggest worsened outcomes[20,19], others report no significant prognostic influence[22,21]. These discrepancies may be due to methodological differences, varying inclusion criteria, and the diversity of treatment protocols. However, the consistency in our findings with studies that report increased thrombotic burden and

RHS in the presence of DVT reinforces the potential utility of DVT as a surrogate marker for PE severity.

It is also worth noting that in our population, 44.9% of patients with PAOI > 45 had concomitant DVT, compared to only 35.3% in those with PAOI < 45. This aligns with the concept that DVT may contribute to a more extensive embolic load. Although our study did not directly assess clinical outcomes such as mortality or recurrence, the significant differences in imaging biomarkers suggest a potential impact on risk stratification and early management decisions.

Given the above, the routine evaluation of DVT in patients with APE, particularly in those with high PAOI or signs of RV strain, may offer additional prognostic value. Future prospective studies with outcome-based endpoints are needed to better elucidate the prognostic implications of coexisting DVT in acute PE.

Conclusion

This study demonstrates that the presence of concomitant deep vein thrombosis (DVT) in patients with acute pulmonary embolism (APE) is significantly associated with more severe imaging markers of right heart strain (RHS) on computed tomography pulmonary angiography (CTPA). Specifically, patients with DVT exhibited higher pulmonary artery obstruction index (PAOI) values and increased right atrial (RA/LA) and right ventricular (RV/LV) diameter ratios - parameters that are well-established indicators of hemodynamic compromise and adverse prognosis in APE.

Although interventricular septal bowing and inferior vena cava (IVC) contrast reflux were more frequently observed in the DVT subgroup, these qualitative features did not reach statistical significance, likely reflecting interobserver variability or limited sample size. Nevertheless, the consistent elevation of quantitative RHS markers in the presence of DVT supports the hypothesis that DVT contributes to a larger embolic burden and more substantial right-sided cardiac strain.

Our findings align with several multicenter trials and meta-analyses suggesting that concomitant DVT may serve as a surrogate marker for PE severity, though its independent impact on mortality and recurrence remains debated. These results reinforce the potential value of integrating CTPA-derived RHS markers with DVT assessment for more refined prognostic stratification.

In clinical practice, early identification of RHS and detection of coexisting DVT may help guide therapeutic intensity, especially in normotensive patients at intermediate risk. Prospective, outcome-driven studies are warranted to further define the prognostic implications of DVT in APE and to clarify whether its routine identification should alter standard diagnostic or management algorithms.

Conflict of interest statement. None declared.

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