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Clinical outcomes and factors associated with pulmonary infarction following acute pulmonary embolism: a retrospective observational study at a US academic centre

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ABSTRACT

Objective Pulmonary infarction is a common clinical and radiographic finding in acute pulmonary embolism (PE), yet the clinical relevance and prognostic significance of pulmonary infarction remain unclear. The study aims to investigate the clinical features, radiographic characteristics, impact of reperfusion therapy and outcomes of patients with pulmonary infarction.

Design, setting and participants A retrospective cohort study of 496 adult patients (≥18 years of age) diagnosed with PE who were evaluated by the PE response team at a tertiary academic referral centre in the USA. We collected baseline characteristics, laboratory, radiographic and outcome data. Statistical analysis was performed by Student's t-test, Mann-Whitney U test, Fischer's exact or χ^2 test where appropriate. Multivariate logistic regression was used to evaluate potential risk factors for pulmonary infarction.

Results We identified 143 (29%) cases of pulmonary infarction in 496 patients with PE. Patients with infarction were significantly younger (52±15.9 vs 61±16.6 years, p<0.001) and with fewer comorbidities. Most infarctions occurred in the lower lobes (60%) and involved a single lobe (64%). The presence of right ventricular (RV) strain on CT imaging was significantly more common in patients with infarction (21% vs 14%, p=0.031). There was no significant difference in advanced reperfusion therapy, in-hospital mortality, length of stay and readmissions between groups. In multivariate analysis, age and evidence of RV strain on CT and haemoptysis increased the risk of infarction.

Conclusions Radiographic evidence of pulmonary infarction was demonstrated in nearly one-third of patients with acute PE. There was no difference in the rate of reperfusion therapies and the presence of infarction did not correlate with poorer outcomes.

STRENGTHS AND LIMITATIONS OF THIS STUDY

⇒ This study represents the largest cohort describing the clinical characteristics and outcomes of patients with pulmonary infarction.
⇒ This study highlights the potential role of pulmonary infarction in the risk stratification of acute pulmonary embolism.
⇒ Cases of pulmonary infarction were identified based on CT findings and there was no histological correlation to differentiate between true necrosis (infarction) versus alveolar haemorrhage.
⇒ Not all patients underwent CT imaging postdischarge and thus we are unable to comment on the precise timing of the resolution of infarction and its long-term significance.

INTRODUCTION

Acute pulmonary embolism (PE) is the third-leading cause of cardiovascular death following myocardial infarction and stroke, with approximately 100 000 annual deaths in the USA.[1 2] Pulmonary infarction is a common complication of acute PE with a reported radiographic prevalence of up to 36%.[3] The dual blood supply of lungs has been thought to be protective against ischaemic insults, with the bronchial circulation and other collateral vessels undergoing hypertrophy or remodelling to maintain blood flow to ischaemic lung tissues through anastomoses at the level of alveoli and respiratory bronchioles.[4 5] Nevertheless, obstruction of the pulmonary artery by acute PE can cause infarction. In the past, the presence of infarction has been regarded as a sign of poor outcomes due to its association with compromised cardiac function, which leads to increased pulmonary venous pressure and impairment of forwarding flow through the bronchial circulation.[6 7] Contrarily, recent studies have suggested that younger, healthier patients are at the highest risk for infarction, because of their less robust collateral blood supply.[8 9] The clinical relevance and prognostic significance of pulmonary infarction remain unclear. The aim of

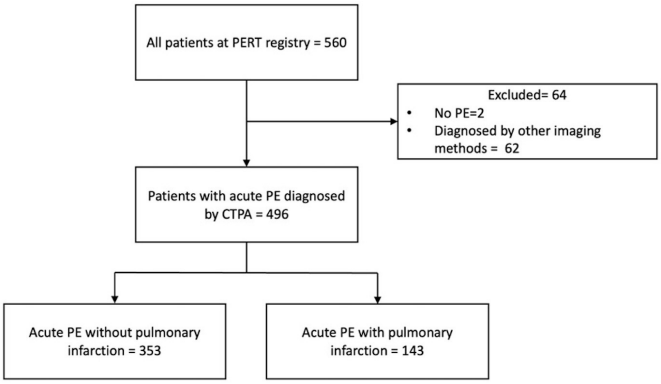

Figure 1 Patient selection flow diagram. CTPA, CT pulmonary angiography; PERT, pulmonary embolism response team.

this study is to investigate the clinical features, radiographic characteristics, impact of reperfusion therapy and outcomes of patients with pulmonary infarction.

METHODS

Study population

We conducted a single-centre retrospective review of all patients between January 2017 and June 2020 at Temple University Hospital who underwent evaluation by the PE response team (PERT) and included all cases of acute PE diagnosed by CT pulmonary angiography (CTPA). Cases of acute PE diagnosed by other imaging modalities were excluded from the study. Cases of pulmonary infarction were identified by review of the final CT reports by board-certified thoracic radiologists. Figure 1 demonstrates patient selection.

Patient and public involvement

Patients or the public were not involved in the design, recruitment, conduct, reporting or dissemination of our research.

Data collection

Clinical data

Patient demographic, clinical features, laboratory data, echocardiographic data, radiographic characteristics and patient outcomes were extracted from electronic medical records. Demographics included age, gender, race and body mass index. Clinical features included symptoms at presentation, comorbid conditions, calculated simplified Pulmonary Embolism Severity Index (sPESI) and PE severity per the European Society of Cardiology guideline at the time of diagnosis (ie, low, intermediate-low, intermediate-high and high risk).[10] Laboratory data included B-type natriuretic peptide (BNP; positive if ≥100 pg/mL) and troponin I (positive if ≥0.1 ng/mL). Data regarding treatment modalities, length of stay, in-hospital mortality, readmission within 30 days, new oxygen requirement on discharge, complications including major and minor bleeding using the International Society on Thrombosis and Haemostasis criteria,[11] need for transfusion, access site haematoma and pulmonary follow-up were recorded.

Radiographic data

All CTPAs performed at the time of acute PE diagnosis were retrieved. The diagnosis of infarction was based on generally accepted criteria with the presence of a peripheral wedge-shaped consolidation within the region of an obstructed vessel, with or without the presence of the other suggestive findings including: (1) central lucency; (2) vessel signs and (3) air bronchogram.[12] In case of discrepancies, a final decision was reached by consensus. A central PE was defined as the presence of thrombus in the main trunk of the pulmonary artery (PA) or the left or the right main PA. A peripheral PE was defined as thrombus in the lobar, segmental or subsegmental PA. Signs of right ventricular (RV) strain were defined as the presence of one, or a combination of the following signs: (1) right-to-left ventricular ratio >0.9; (2) pulmonary artery enlargement; (3) abnormal interventricular septum (flattening of septum or leftward septal bowing) and (4) inferior vena caval contrast reflux. Parameters including PE distribution (central vs distal), infarct location and burden, signs of RV strain and other parenchymal abnormalities were collected. All available follow-up CT chest imaging up to 1-year postdischarge were reviewed by board-certified thoracic radiologists to determine the resolution of infarctions or residual abnormalities on available scans. All echocardiograms were reviewed, and data regarding left ventricular ejection fraction, RV dilation and RV dysfunction were collected.

Statistical analysis

All continuous variables were tested for normality and presented as mean with an SD, or median with an IQR if distribution was skewed. Categorical variables were presented as absolute number (percentage). Comparisons between patients with and without infarction were performed using Student's t-test or Mann-Whitney U for continuous variables or Fischer's exact or χ^2 test for categorical variables, as appropriate. Univariable and multivariable logistic regression models were used to evaluate risk factors associated with pulmonary infarction. The software SPSS Statistics for Mac, V.26.0 (IBM) was used for statistical analysis. P values of <0.05 (two-sided) were considered statistically significant.

RESULTS

Patient characteristics

Out of 560 patients in the PERT registry, 62 cases were excluded due to diagnosis by other imaging modalities and 2 cases were excluded after diagnosis of PE was ruled out. Hence, 496 patients were included in the study with a mean age of 58 years and 48% were female. Twenty-nine per cent (143 of 496) of patients had evidence of pulmonary infarction on CTPA. Patients with pulmonary infarction were younger (52±15.9 vs 61±16.6 years, p<0.001)

Table 1 Baseline characteristics and symptoms of presentation

	All (496)	Without infarction (353)	With infarction (143)	P value
Gender (female)	238 (48%)	168 (48%)	70 (49%)	0.784
Age, mean (±SD)	58±16.9	61±16.6	52±15.9	<0.001
Race, n (%)				0.017
Black	249 (50)	168 (48)	81 (57)	
White	98 (20)	81 (23)	17 (12)	
Other	149 (30)	104 (30)	45 (32)	
BMI, mean (±SD)	31.8±9.3	31.8±9.5	32.0±8.7	0.421
Symptoms at presentation, n (%)				
Dyspnoea	297 (60)	207 (59)	90 (63)	0.376
Hypoxia	237 (49)	173 (50)	64 (46)	0.371
Pleuritic chest pain	114 (23)	69 (20)	45 (32)	0.004
DVT symptoms	50 (10)	40 (11)	10 (7)	0.146
Syncope	48 (10)	40 (11)	8 (6)	0.05
Haemoptysis	21 (4)	9 (3)	12 (8)	0.04
Altered mental status	38 (8)	28 (8)	10 (7)	0.722
Cardiac arrest	22 (4)	15 (4)	7 (5)	0.752
Comorbidities, n (%)				
Cardiac diseases	191 (39)	148 (42)	43 (30)	0.014
COPD/asthma	71 (14)	54 (15)	17 (12)	0.326
CKD	49 (10)	42 (12)	7 (5)	0.018
Diabetes mellitus	115 (23)	91 (26)	24 (17)	0.032
Hypothyroidism	36 (7)	31 (9)	5 (4)	0.039
Malignancy	111 (22)	88 (25)	23 (16)	0.032
Recent surgery	68 (14)	52 (15)	16 (11)	0.299
Current anticoagulation	59 (12)	41 (12)	18 (13)	0.762

BMI, body mass index; CKD, chronic kidney disease; COPD, chronic obstructive pulmonary disease; DVT, deep vein thrombosis; n, number; SD, standard deviation.

and with a significantly lower prevalence of comorbidities including cardiac disease (30% vs 42%, p=0.014), chronic kidney disease (CKD) (5% vs 42%, p=0.018), diabetes mellitus (17% vs 26%, p=0.032), hypothyroidism (4% vs 9%, p=0.039) and malignancy (16% vs 25%, p=0.032). Although results did not reach statistical significance, chronic obstructive pulmonary disease was less prevalent in the infarction group. Patients with infarction were more likely to present with pleuritic chest pain (32% vs 20%, p=0.004) and haemoptysis (8% vs 3%, p=0.04), while patients without infarction were more likely to present with syncope (6% vs 11%, p=0.05). The baseline characteristics and symptoms of presentation of the study population are reported in table 1.

Risk stratification of PE

Troponin elevation (>0.1 ng/mL) was less frequently observed in patients with infarction (39% vs 49%, p=0.031). The presence of RV strain on CT was significantly higher in patients with infarction (58% vs 45%, p=0.009), although no differences in signs of RV dilation or dysfunction on echocardiogram between infarction and non-infarction group were noted. There was no significant difference in sPESI risk group, BNP elevation, PE severity or evidence of RV dysfunction on electrocardiography and echocardiogram between groups. Table 2 describes factors associated with PE severity.

Radiographic characteristics

There was no difference in PE distribution and clot burden between groups and 50% of patients with infarction had thrombus located at the main PAs including those who had saddle PE. Most infarctions occurred in the lower lobe (60%) and involved a single lobe (64%). Patients with infarction were more likely to have parenchymal abnormalities including consolidation (21% vs 12%, p=0.007), pulmonary oedema (13% vs 7%, p=0.036), pleural effusion (33% vs 24%, p=0.038) and ground glass opacity (20% vs 13%, p=0.042). Table 3 demonstrates the radiographic characteristics of patients with acute PE. Table 4 demonstrates the radiographic characteristics of pulmonary infarction. Fifty-eight (41%) patients had follow-up

Table 2 PE severity indices

	All (496)	Without infarction (353)	With infarction (143)	P value
sPESI score				0.108
Low risk (0 points)	89 (21%)	57 (19%)	32 (26%)	
High risk (≥1 points)	327 (79%)	238 (81%)	89 (74%)	
Elevated BNP	242 (54%)	169 (53%)	73 (55%)	0.629
Elevated troponin	228 (46%)	173 (49%)	55 (39%)	0.031
Evidence of RV strain on ECG	77 (16%)	48 (14%)	29 (21%)	0.137
PE severity				
Low risk	132 (27%)	93 (26%)	39 (27%)	0.994
Intermediate low risk	173 (35%)	124 (35%)	49 (34%)	
Intermediate high risk	144 (29%)	103 (29%)	41 (29%)	
High risk	47 (10%)	33 (9%)	14 (10%)	
ECG evidence of RV strain	77 (16%)	48 (14%)	29 (21%)	0.137
CT evidence of RV strain	242 (49%)	159 (45%)	83 (58%)	0.009
Echocardiogram				
RV dilation	251 (54%)	173 (52%)	78 (57%)	0.302
RV dysfunction	235 (50%)	162 (49%)	73 (54%)	0.338
LVEF	38 (8%)	23 (7%)	15 (11%)	0.306
LE DVT	227 (50%)	161 (51%)	66 (50%)	0.928
UE DVT	39 (22%)	27 (22%)	12 (21%)	0.916

BNP, B type natriuretic peptide; DVT, deep vein thrombosis; ECG, electrocardiography; LE, lower extremity; LVEF, left ventricular ejection fraction; PE, pulmonary embolism; RV, right ventricular; sPESI, simplified Pulmonary Embolism Severity Index; UE, upper extremity.

CT-chest imaging for a variety of reasons performed up to 1-year postdischarge. Resolution of pulmonary infarction was observed in 53% (17 out of 32), 85% (12 out of 14), and 92% (11 out of 12) of patients who had imaging at 3-month, 6-month and 1-year intervals.

Treatment and outcomes

There was no significant difference in the number of patients receiving advanced reperfusion therapy between groups. Most patients with pulmonary infarction received anticoagulation alone (69%), followed by catheter-directed thrombolysis (12%) and systemic thrombolysis (11%). There were more patients with infarction who received antimicrobial therapy compared with those without infarction (15% vs 5%, p<0.001). Among the 47 patients who had concomitant infarction and pleural effusion, 6 patients (13%) underwent diagnostic and/or therapeutic thoracocentesis and five had an exudative pleural effusion. Patients with infarction were less likely to require oxygen on discharge (11% vs 19%, p=0.031), and those who required oxygen on discharge were more likely to have multiple lobe infarctions than single lobe infarction (19% vs 7%, p=0.032). There was no significant difference between patients with infarction and without infarction regarding length of stay (10.7±14.7 vs 9.5±12.3, p=0.698), in-hospital death (7% vs 8%, p=0.801), disposition (home: 78% vs 75%, p=0.759), bleeding complications (24% vs 12%, p=0.089) and readmission within 30 days (18% vs 16%, p=0.584) (online supplemental table 1).

Patients with infarction who underwent CDT had a longer length of stay (15±24.4 vs 7±8.4, p=0.044) and a higher rate of readmission within 30 days (18% vs 2%, p=0.047), compared with those without infarction. Readmission diagnoses of patients with infarction who underwent CDT were as follows: one patient developed vaginal bleeding from anticoagulant use, one patient developed acute hypoxic respiratory failure due to multifocal pneumonia, and one patient was admitted for chest pain. There was no significant difference in in-hospital mortality (6% vs 6%, p=0.973) and complications including minor and major bleeding (24% vs 21%, p=0.676), access site haematoma (13% vs 2%, p=0.134) and need for transfusion (18% vs 13%, p=0.649) between those with and without infarction (online supplemental table 2).

In univariable regression analyses, we identified several factors independently associated with pulmonary infarction. These included age, history of cardiac diseases, malignancy, hypothyroidism, diabetes mellitus, CKD, elevated troponin, pleuritic chest pain, haemoptysis and RV strain on CT. A multivariable regression analysis was subsequently performed, and four factors remained significant: Both haemoptysis (OR 3.034; 95% CI 1.162 to 7.924) and presence of RV strain on CT (OR 2.142; 95% CI 1.365 to 3.360) significantly increased the risk of

Table 3 Radiographic characteristics of patients with acute pulmonary embolism

	All (496)	Without infarction (353)	With infarction (143)	P value
RV strain on CT	242 (49%)	159 (45%)	83 (58%)	0.009
RV to LV ratio ≥1	155 (31%)	106 (30%)	49 (34%)	0.64
Dilated PA	99 (20%)	59 (17%)	40 (28%)	0.011
Septal position				0.03
Rightward bowing	131 (27%)	102 (29%)	29 (20%)	
Flattened	112 (23%)	66 (19%)	46 (32%)	
Leftward bowing	50 (10%)	32 (9%)	18 (13%)	
PE distribution				0.052
Central PE	216 (44%)	144 (41%)	72 (50%)	
Distal PE	280 (57%)	209 (59%)	71 (50%)	
Clot burden				0.2
Main, saddle or proximal	215 (43%)	143 (41%)	72 (50%)	
Interlobar to lobar	124 (25%)	94 (27%)	30 (21%)	
Segmental	150 (30%)	110 (31%)	40 (28%)	
Subsegmental	7 (1%)	6 (2%)	1 (1%)	
Parenchymal abnormalities				
Pulmonary oedema	42 (9%)	24 (7%)	18 (13%)	0.036
Consolidation	71 (14%)	41 (12%)	30 (21%)	0.007
Emphysema	65 (13%)	48 (14%)	16 (11%)	0.469
Fibrosis	42 (9%)	30 (9%)	12 (8%)	0.969
Pleural effusion	128 (26%)	84 (24%)	47 (33%)	0.038
Atelectasis	161 (33%)	113 (32%)	48 (34%)	0.738
Ground glass opacity	72 (15%)	44 (13%)	28 (20%)	0.042

CT, computed tomography; LV, left ventricular; PA, pulmonary artery; PA, pulmonary artery; PE, pulmonary embolism; RV, right ventricular.

infarction, while age (OR 0.973, 95% CI 0.959 to 0.987) and presence of elevated troponin (OR 0.629; 95% CI 0.398 to 0.993) decreased risk (table 5).

DISCUSSION

In this study, the estimated prevalence of pulmonary infarction was 29%. Patients with infarction were more likely to present with pleuritic chest pain and haemoptysis. Additionally, those with pulmonary infarction were younger and had a lower prevalence of comorbidities. While the presence of RV strain on CT imaging was more common in patients with pulmonary infarction, the rate of reperfusion therapies, complications and outcomes was similar in both groups. The presence of haemoptysis and RV strain on CT significantly increased the risk of infarction, whereas age and elevated troponin decreased the risk. Pulmonary infarction resolved in the majority of patients for whom follow-up imaging was available, which is in concordance with newer studies.[8 9 13]

Table 4 Radiographic characteristics of pulmonary infarction

	Infarcts, no (%)
Infarct location	
Upper lobe	31 (22)
Middle lobe	8 (6)
Lower lobe	86 (60)
Multiple	18 (12)
Infarct burden	
Single lobe	91 (64)
Multiple lobes	52 (36)

The prevalence of pulmonary infarction in our cohort is in keeping with previously reported rates ranging from 9% to 36%.[3 13] Regarding clinical presentation, the higher presence of pleuritic chest pain and haemoptysis in patients with infarction likely represents a result of alveolar haemorrhage, leading to pleural inflammation, irritation and necrosis. In addition, the presence of pleural effusion was more prevalent in patients with infarction due to pleural inflammation following infarction, however, most of these effusions were not intervened on and had resolved on follow-up imaging. Although patients with infarction were more likely to present with pleuritic chest pain and haemoptysis, these symptoms were only present in 32% and 8% of patients with infarction, respectively.

Table 5 Univariate and multivariate analysis of potential risk factors

Variables			Univariate analysis			Multivariate analysis		
	Without infarction (353)	With infarction (143)	OR	P value	95% CI	OR	P value	95% CI
Age, years	61±16.6	52±15.9	0.968	<0.001	0.956 to 0.980	0.973	<0.001	0.959 to 0.987
Cardiac diseases	148 (42%)	43 (30%)	0.596	0.014	0.393 to 0.902			
CKD	42 (12%)	7 (5%)	0.381	0.022	0.167 to 0.870			
DM	91 (26%)	24 (17%)	0.581	0.033	0.352 to 0.957			
Malignancy	88 (25%)	23 (16%)	0.577	0.034	0.348 to 0.958			
Hypothyroidism	31 (9%)	5 (4%)	0.375	0.047	0.143 to 0.985			
Elevated troponin	173 (49%)	55 (39%)	0.647	0.032	0.435 to 0.962	0.629	0.047	0.398 to 0.993
Pleuritic CP	69 (20%)	45 (32%)	1.890	0.005	1.217 to 2.935			
Haemoptysis	9 (3%)	12 (8%)	3.491	0.006	1.437 to 8.479	3.034	0.023	1.162 to 7.924
RV strain on CT	159 (45%)	83 (58%)	1.688	0.009	1.140 to 2.500	2.142	<0.001	1.365 to 3.360

CKD, chronic kidney disease; CP, chest pain; CT, computed tomography; DM, diabetes mellites; RV, right ventricular.

Thus, the majority did not have these symptoms may not be useful in identifying pulmonary infarction. Interestingly, patients with pulmonary infarction were more likely to be treated with antimicrobial therapy than those without. One possible explanation is that other lung processes, such as pneumonia, pulmonary oedema or atelectasis can produce consolidative changes similar to infarction on CT imaging,[14] and as a result, pneumonia cannot be excluded especially when combined with a clinically compatible presentation.

In our study, patients with infarction were significantly younger and with fewer comorbid conditions, specifically a lower prevalence of cardiac disease and malignancy that have traditionally been regarded as major risk factors for infarction. This discrepancy may be due to a lack of efficient collateral circulation to lung tissues, which presumably develops in the setting of longstanding local tissue hypoxia,[8 15] and is unlikely to happen in otherwise healthy, young individuals in the absence of cardiopulmonary diseases.

Our cohort also found that patients with infarction did have higher rates of RV strain, dilated PA and flattened or leftward bowing of the interventricular septum, although signs of RV strain were not reproducible on echocardiography. A potential explanation for this finding is that patients with infarction were younger with less comorbid diseases, thus without pre-existing RV hypertrophy allowing for tolerance of acute RV afterload elevation. This may also explain our finding that troponin elevation was less likely to be observed in patients with infarction. While troponin elevation is indicative of RV strain, RV myocardium might not necessarily be its only source.[16] A mismatch between oxygen demand and supply (demand ischaemia) and a decrease in renal clearance could contribute to the higher rates of troponin elevation observed in patients without infarction, as evidenced by their higher prevalence of comorbidities.

Regarding the discrepancy between CT and echocardiographic findings, unlike CTPA which is often the first diagnostic and confirmatory modality for PE, most echocardiography is performed after the initial diagnosis and potentially after reperfusion therapy. In our study, the mean and median time lapse between the availability of CT results to echocardiogram results was 29 hours 40 min and 20 hours 11 min, respectively. Thus, it is possible that the initial signs of RV strain shown on CTPA could have improved or resolved through several potential mechanisms; administration of supplemental oxygen leading to a reversal of hypoxic vasoconstriction, clot burden reduction using reperfusion therapy, or intrinsic thrombolytic activity with anticoagulation support alone.

Importantly, we demonstrated no differences in complication rates and in-hospital mortality in patients who underwent CDT with pulmonary infarction compared with those without, although patients with infarction had a longer length of stay and a higher rate of 30-day readmission for non-PE or CDT-related diagnoses. There remains a theoretical risk of increased bleeding complications in the area of pulmonary infarction during catheter manipulation and local installation of thrombolytics, however, this was not demonstrated in our cohort.

Study limitations

To the best of our knowledge, this study represented the largest cohort of patients with acute PE complicated by pulmonary infarction, however, there are several limitations in our study. Cases of pulmonary infarction were identified based on CT findings and there was no histological correlation to differentiate between true necrosis versus alveolar haemorrhage. Patients with conditions that led to pulmonary hypertension could have pre-existing parenchymal changes that resemble acute RV strain caused by PE. In those scenarios, it would be technically difficult to differentiate whether these CT features

are related to pre-existing pulmonary hypertension itself, or the combination. Owing to the retrospective nature of this study, not all patients underwent CT imaging postdischarge, thus we were unable to comment on the precise timing of the resolution of pulmonary infarction. We acknowledge the fact that not all patients in our cohort had echocardiography, in particular, patients with low-risk PE when echocardiography was not deemed necessary to change management or patients with high-risk PE who expired prior to the completion of echocardiography. Other limitations included the retrospective, single-centre nature of the data, which may be prone to selection bias.

In conclusion, pulmonary infarction was demonstrated on CT in nearly one-third of acute PE, and patients with infarction were younger, with fewer comorbidities, and more likely to present with pleuritic chest pain and haemoptysis. Overall, there was no difference in length of stay, in-hospital death, bleeding complication and readmission rate between patients with and without infarction, and patients with infarction were less likely to require oxygen on discharge. The presence of pulmonary infarction did not correlate with poorer outcomes and should not be considered a supporting factor nor a contraindication for advanced reperfusion therapy for PE.

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Contributors KUL is the primary author, collected and analysed the data, and is the guarantor of the article, taking responsibility for the integrity of the work as a whole from inception to the published article. OO'C and PR are responsible for the study concept and helped write the manuscript. RB, GC, VL and JP reviewed the imaging studies and treated the patients who underwent catheter-directed thrombolysis. SB and BR-L helped write the manuscript.

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Provenance and peer review Not commissioned; externally peer reviewed.

Data availability statement All data relevant to the study are included in the article or uploaded as online supplemental information.

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