Original Article

Characteristics of pulmonary infarction in patients with acute pulmonary embolism in China: a single-center retrospective observational study

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Background: Pulmonary infarction (PI) is an uncommon complication of pulmonary embolism (PE). The risk factors of PI are still relatively unclear.

Methods: This was a single-center retrospective review conducted on 500 patients with PE. After applying the inclusion and exclusion criteria, 386 patients diagnosed with PE were enrolled in our study. These patients were then categorized into the PI group (n=64) and the non-PI group (n=322). A comparison was conducted between the two groups regarding the clinical characteristics.

Results: The occurrence of PI secondary to PE was 16.58%. In univariate analysis, recent trauma (21.9% *vs.* 9.9%, P=0.007), pleuritic chest pain (46.9% *vs.* 17.4%, P<0.001), hemoptysis (29.7% *vs.* 2.5%, P<0.001), fever (26.6% *vs.* 8.1%, P<0.001), lower limb edema/pain (37.5% *vs.* 14.0%, P<0.001), white blood cell (WBC) counts (37.5% *vs.* 24.5%, P=0.032), C-reactive protein (CRP) (65.6% *vs.* 41.3%, P<0.001), and pleural effusion (45.3% *vs.* 18.6%, P<0.001) were associated with an increased risk of PI. Multivariate analysis demonstrated that age [odds ratio (OR) 0.975, 95% confidence interval (CI): 0.951–0.999, P=0.045], pleuritic chest pain (OR 2.878, 95% CI: 1.424–5.814, P=0.003), hemoptysis (OR 10.592, 95% CI: 3.503–32.030, P<0.001), lower limb edema/pain (OR 2.778, 95% CI: 1.342–5.749, P=0.006) and pleural effusion (OR 3.127, 95% CI: 1.531–6.388, P=0.002) were independent factors of PI due to PE. No significant difference was recorded between the two groups in treatment and mortality.

Conclusions: Young patients were found to be a higher risk of PI. Pleural effusion was found to be a factor for PI. PI should be considered when pleuritic chest pain, hemoptysis, or lower limb edema/pain are present with peripheral opacity.

Keywords: Pulmonary embolism (PE); pulmonary infarction (PI); risk factor

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Introduction

Pulmonary embolism (PE) constitutes a perilous manifestation of venous thromboembolism (VTE) that can be challenging for diagnosis and treatment. In the United States, there are 100–200 cases of PE for every 100,000 people (1). PE ranks as the third most frequent contributor to cardiovascular mortality worldwide after heart attack and stroke (2). In diagnosed and treated PE, the death rate is 8%, while in untreated PE, it rises to nearly 30% (3). A lung section would suffer from a pulmonary infarction (PI) when its blood supply is cut off, causing the tissue to perish. The literature states that the prevalence of PI ranges from 16% to 31% (4-6).

The bronchial and pulmonary artery supply blood to the lungs, preventing ischemic injury (7). Due to the collateral pathway's alternate perfusion, an occlusive pulmonary vascular lesion typically does not result in infarction. Yet, when there is a reduced bronchial artery flow and increased pulmonary venous pressure, the visceral perfusion would be impaired severely, making the PE leading to PI (8). Previous studies reported for older individuals with comorbidities, particularly those with impaired heart function, were more likely to get PI (9,10) due to poor collateral circulation. In addition, other predisposing factors were pneumonia (11), malignancy (10), and advanced age (12). However, recent studies showed young and healthy patients had a higher risk for PI (5,6,13). According to the concept put forward by Islam *et al.* (5), chronic cardiopulmonary diseases caused long-term local tissue hypoxia and stronger vascular collateralization, which protected lung parenchyma from infarction.

PI patients experience pleuritic chest pain and hemoptysis more frequently. It is possible to miss a small PI if no clinical symptoms appear. Chest CT scans are more commonly used to diagnose PI; even small PIs can be seen. A typical sign of PI is peripheral wedge-shaped, pleural-based consolidation (14).

Previous studies (5,6,15) have researched on the risk factors for PI with PE. However, risk factors that contribute to PI are still relatively unclear and controversial. Meanwhile, little is known about PI's clinical relevance and characteristics in Chinese PE patients. Consequently, we aimed to ascertain the incidence of PI associated with PE, analyze the clinical features of patients with PI, and identify the PI risk factors from different potential factors. We present this article in accordance with the STROBE reporting checklist (available at https://jtd.amegroups.com/article/view/10.21037/jtd-23-944/rc).

Highlight box

Key findings

- Young patients were more likely to experience pulmonary infarction (PI). Pleural effusion was a risk factor for PI.

What is known and what is new?

- Young patients have a higher risk of developing PI.
- Patients with PI were more likely to present with pleuritic chest pain and hemoptysis.
- This manuscript is the first Chinese study of PI.

What is the implication, and what should change now?

- Doctors should improve awareness of PI and reduce missed and misdiagnosed cases.
- The diagnosis of PI could potentially prevent unnecessary antibiotic use.

Methods

It is a retrospective observational study conducted at a single center between January 1, 2017, and December 31, 2021, by reviewing the electronic medical record system and searching for the terms 'pulmonary thromboembolism', 'pulmonary embolism', or 'pulmonary thrombosis' to obtain patients with acute PE in the Department of Pulmonary and Critical Care Medicine, Beijing Luhe Hospital, Capital Medical University, Beijing, China. The study was conducted in accordance with the Declaration of Helsinki (as revised in 2013). The retrospective study was approved by the Ethics Committee of Beijing Luhe Hospital, Capital Medical University (No. 2021-LHKY-119-02). Individual consent was waived due to the retrospective nature of this study.

Patients who had acute PE were diagnosed according to the standards of the 2008 European Society of Cardiology (ESC) recommendations on acute PE diagnosis and treatment (16). The definitive diagnosis required computed tomography pulmonary angiography (CTPA) or ventilation-perfusion scan (V/Q scan). Partial or complete endoluminal filling defects in the pulmonary arteries showed by CTPA or normal ventilation in hypoperfused segments (perfusion-ventilation mismatch) showed by the V/Q scan would be considered as PE. Patients had to be at least 18 years of age at the time of diagnosis for being enrolled in the study. The inclusion criteria were patients with acute PE diagnosed with CTPA. Patients whose diagnoses were not confirmed by radiography or who were previously diagnosed with PE were excluded. Patients diagnosed by V/Q scan were also excluded (In V/Q scan, the main signs are well-defined

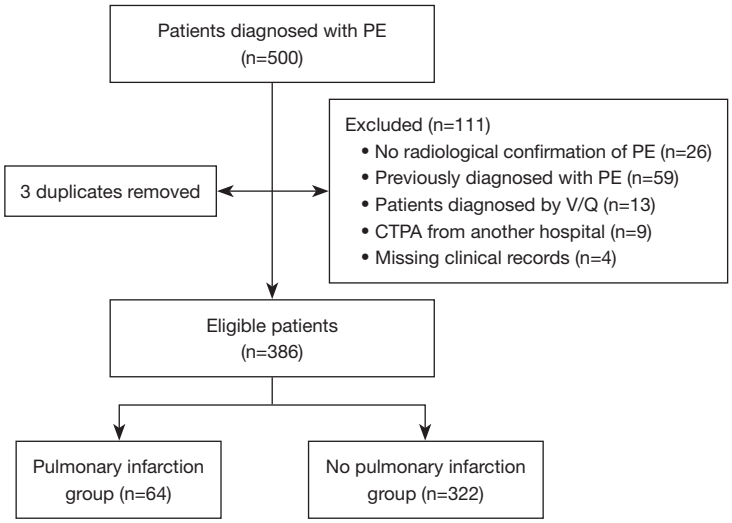

Figure 1 Flowchart of patient selection. PE, pulmonary embolism; V/Q, ventilation-perfusion scintigraphy; CTPA, computed tomography pulmonary angiography.

pulmonary lobe and segmental perfusion defects. But the specific location of pulmonary artery thromboembolism cannot be observed). Furthermore, CTPA from another hospital and lack of complete clinical data were excluded. PI in PE was defined according to the following criteria (17-20): (I) Hampton hump; (II) peripheral consolidation with nearby subsegmental PE or more central PE; (III) presence of one or more of the following imaging features: peripheral opacity with central lucencies; peripheral opacity with thickened arteries leading directly to the opacity's apex; peripheral opacity combined with ground-glass opacity; peripheral ground-glass opacity; peripheral opacity with cavitation; (IV) no other attributable causes, except for PI, including pneumonia, interstitial lung disease, drug or radiation-induced pneumonitis, or cancer. Depending on the presence or absence of PI, the enrolled patients were allocated into the PI and non-PI groups. *Figure 1* shows a patient inclusion flow chart.

Physicians conducted a review of the electronic medical records and extracted data pertaining to the patient's age, gender, past medical history, history of trauma (lower limb injuries such as femoral neck fractures, patellar fractures, etc. leading to recent bed rest) and surgery (occurring within a month), smoking history, medication use, clinical presentation, laboratory data, length of stay in hospitals, and incidence of inpatient mortality. Calculations were conducted for the pulmonary embolism severity index (PESI) and its simplified version. Computed tomography (CT) sign analyses were independently performed in a blinded manner by a senior radiologist and a senior respiratory specialist without knowledge of the clinical data. This procedure was performed to obtain a consensus among the experts. In addition, an attending sonographer performed color Doppler echocardiography to quantify the pulmonary artery systolic pressure (PASP) and assess the right ventricular (RV) dilation. The frequency of PI was the study's main outcome. The connection between clinical factors and PI, shown as odds ratios, was the secondary outcome.

Statistical analysis

The Kolmogorov-Smirnov test was employed for the assessment of the continuous variable normality. The median [interquartile range (IQR)] was carried out to describe the non-normally distributed data, whereas the mean and standard deviation were used to describe the normally distributed data. The *t*-test and Mann-Whitney *U* test were utilized for comparing continuous variables with normal and non-normal distributions, respectively. The study employed the Chi-squared or Fisher's exact test to compare categorical variables that were provided as frequency (percentages). The criteria for statistical significance were two-tailed P values <0.05. The odds ratio (OR) with 95% confidence intervals (CIs) assessing the risk of PI with PE was calculated using multivariable binomial logistic regression models. Data analysis was performed through SPSS, version 26.0 (IBM Corporation, Armonk, NY, USA).

Results

This study analyzed a consecutive sample of 386 patients enrolled between January 1, 2016, and December 31, 2020. Of these patients, 64 (16.58%) were PE patients with PI, and 322 (83.42%) were PE patients without PI. The mean age was 67.54±13.58 years. Moreover, no significant differences were found between the two groups in terms of age distribution, gender, or comorbid diseases, including chronic obstructive pulmonary disease (COPD); asthma; chronic bronchitis; interstitial lung disorder; sleep apnea-hypopnea syndrome (SAHS); malignancy; coronary heart disease; hypertension; heart failure; atrial fibrillation; cerebrovascular disease; diabetes mellitus; and connective tissue disease, as well as other PE risk factors (*Table 1*). The distribution of PE and PI in different age groups was seen in *Figure 2A*. The PI incidence showed a gradual decline with advancing age (*Figure 2B*). Recent trauma exhibited a significant increase (21.9% *vs.* 9.9%, P=0.007).

The time period between the symptom appearance and confirmed PE diagnosis was 7.13±7.55 *vs.* 10.76±25.56 days. The time period between the symptom appearance and the initial visit was 4.14±5.16 *vs.* 8.72±24.80 days. The time duration from the initial visit to the definitive diagnosis of PE was 4.14±5.16 *vs.* 8.72±24.80 days.

The incidence of pleuritic chest pain, hemoptysis, fever, and lower limb edema/pain was significantly higher than in no-PI patients. Both groups showed no significant difference in other clinical symptoms as well as in the simplified PESI score, hospitalization duration, and in-hospital deaths (*Table 1*). There were two in-hospital deaths. One patient died from severe pneumonia, and one patient died suddenly of food suffocation during a hospital stay. Of the 64 PI patients, 16 (25%) patients were misdiagnosed with pneumonia. And this group presented more frequently with the antibiotic application.

The PI group exhibited elevated inflammatory marker levels, including white blood cell (WBC) counts and C-reactive protein (CRP), compared to the other group. No significant differences in WBC, D-dimer, troponin I, brain-type natriuretic peptide (BNP), RV dilation, pulmonary hypertension, deep venous thrombosis, and main pulmonary artery involvement were found. Pleural effusion and CRP exhibited significant differences (*Table 2*). *Table 3* demonstrates the radiographic characteristics of PI. Most infarctions were located in the lower lobes (60.75%).

The dependent variable of whether PI occurs was utilized, while other variables were employed as independent variables. The study incorporated statistically significant variables into a multivariable logistic regression model and subsequently conducted a multivariable logistic regression analysis, revealing that PI had an independent

Table 1 Baseline characteristics of patients with pulmonary embolism (n=386)

Characteristics	Pulmonary infarction (n=64)	No pulmonary infarction (n=322)	P value
Age (years)	64 [52–73]	70 [62–77]	<0.001
Male gender	35 (54.7)	146 (45.3)	0.171
Comorbidities			
COPD	4 (6.3)	35 (10.9)	0.263
Asthma	1 (1.6)	9 (2.8)	>0.99
Chronic bronchitis	1 (1.6)	11 (3.4)	0.435
Interstitial lung disease	0 (0.0)	13 (4.0)	0.138
SAHS	1 (1.6)	7 (2.2)	>0.99
Lung cancer	2 (3.1)	11 (3.4)	>0.99
Coronary heart disease	9 (14.1)	77 (23.9)	0.084
Hypertension	33 (51.6)	170 (52.8)	0.857
Atrial fibrillation	4 (6.3)	25 (7.8)	0.800
Heart failure	3 (4.7)	27 (8.4)	0.445

Table 1 *(continued)*

Table 1 *(continued)*

Characteristics	Pulmonary infarction (n=64)	No pulmonary infarction (n=322)	P value
Cerebrovascular disease	14 (21.9)	58 (18.0)	0.469
Diabetes mellitus	5 (7.8)	51 (15.8)	0.096
Connective tissue disease	0 (0.0)	4 (1.2)	>0.99
Other system tumors	2 (3.1)	13 (4.0)	>0.99
Risk factors			
History of DVT	6 (9.4)	54 (16.8)	0.136
Varicose veins of lower limbs	10 (15.6)	34 (10.6)	0.244
Recent surgery	7 (10.9)	24 (7.5)	0.349
Recent trauma	14 (21.9)	32 (9.9)	0.007
Recent immobilisation	10 (15.6)	28 (8.7)	0.089
Hormone therapy	0 (0.0)	1 (0.3)	>0.99
Oestrogen use, women	1 (1.6)	0 (0.0)	0.166
History of smoking	24 (37.5)	100 (31.1)	0.313
Symptoms			
Dyspnoea	48 (75.0)	269 (83.5)	0.103
Pleuritic chest pain	30 (46.9)	56 (17.4)	<0.001
Hemoptysis	19 (29.7)	8 (2.5)	<0.001
Fever	17 (26.6)	26 (8.1)	<0.001
Cough	23 (35.9)	65 (20.2)	0.006
Palpitation	7 (10.9)	32 (9.9)	0.809
Hypotension	2 (3.1)	13 (4.0)	>0.99
Syncope	6 (9.4)	41 (12.7)	0.453
Lower limb edema/pain	24 (37.5)	45 (14.0)	<0.001
sPESI (≥1)	29 (45.3)	182 (56.5)	>0.99
Risk stratification			0.150
Low risk	26 (40.6)	87 (27.0)	
Intermediate-low risk	24 (37.5)	133 (41.3)	
Intermediate-high risk	11 (17.2)	77 (23.9)	
High risk	3 (4.7)	25 (7.8)	
Treatment			>0.99
Thrombolysis	3 (4.7)	15 (4.7)	
Anticoagulation	61 (95.3)	307 (95.3)	
Hospital length of stay (day)	10.5 [8–15]	10 [8–14]	0.412
In-hospital mortality	0 (0.0)	2 (0.6)	>0.99

Data are presented as median [interquartile range] or n (%). COPD, chronic obstructive pulmonary disease; SAHS, sleep apnea hypopnea syndrome; DVT, deep venous thrombosis; sPESI, simplified pulmonary embolism severity index.

Table 2 Laboratory and radiographic characteristics

Laboratory and radiographic findings	Pulmonary infarction (n=64)	No pulmonary infarction (n=322)	P value
WBC (>10×10^9/L)	24 (37.5)	79 (24.5)	0.032
D-dimer (>0.243 μg/mL)	57 (89.1)	280 (87.0)	0.644
CRP (>10 mg/L)	42 (65.6)	133 (41.3)	<0.001
Troponin I (>0.034 ng/mL)	11 (17.2)	59 (18.3)	0.830
BNP (>100 pg/mL)	20 (31.3)	120 (37.3)	0.360
RV dilation	39 (60.9)	179 (55.6)	0.431
Pulmonary hypertension	26 (40.6)	155 (48.1)	0.271
Pleural effusion	29 (45.3)	60 (18.6)	<0.001
Deep venous thrombosis	40 (62.5)	175 (54.3)	0.230
Main pulmonary artery involvement	21 (32.8)	108 (33.5)	0.910

Categorical data are presented as n (%). WBC, white blood cell; CRP, C-reactive protein; BNP, brain-type natriuretic peptide; RV, right ventricular.

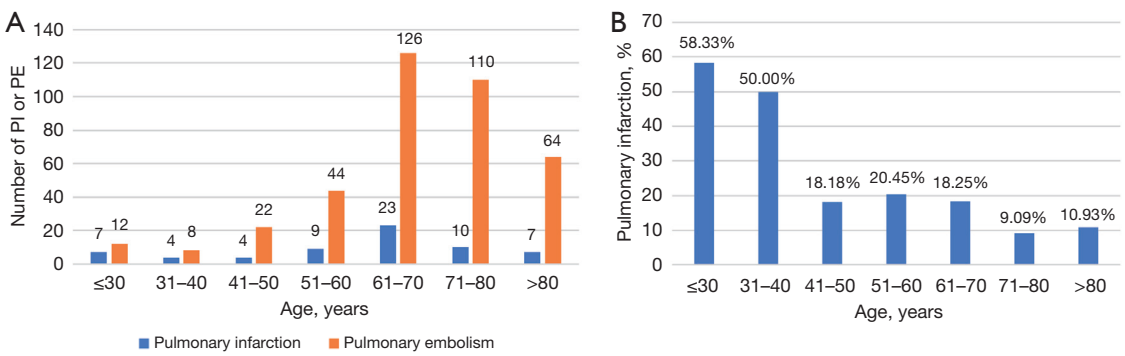

Figure 2 Age distribution of pulmonary embolism and pulmonary infarction (A) and prevalence of pulmonary infarction as a function of age (B) in 386 patients with acute pulmonary embolism. PI, pulmonary infarction; PE, pulmonary embolism.

Table 3 Radiographic characteristics of pulmonary infarction

Infarct location	Infarcts (N=107)
Left upper lobe	17 (15.89)
Left lower lobe	28 (26.17)
Right upper lobe	13 (12.15)
Middle	12 (11.21)
Right lower lobe	37 (34.58)

Data are presented as n (%).

correlation to pleuritic chest pain (OR 2.878, 95% CI: 1.424–5.814, P=0.003), hemoptysis (OR 10.592, 95% CI: 3.503–32.030, P<0.001), and lower limb edema/pain (OR 2.778, 95% CI: 1.342–5.749, P=0.006) (*Table 4*). Moreover, pleural effusion (OR 3.127, 95% CI: 1.531–6.388, P=0.002) was an independent risk factor correlated to PI.

Discussion

PI represents a PE complication. As the lung tissue has a dual circulation through pulmonary and bronchial vasculatures and directly absorbs oxygen through alveolar ventilation, the lung has partial resistance to ischemic damage. Recent research reported that the prevalence of PI was 16% to 31% (4-6). In our research, 16.58% of PE patients had PI.

Firstly, one interesting finding is that individuals with PI were generally younger than those without. This is consistent with findings recorded in other literature (5,6,15).

Table 4 Multivariate logistic regression analysis for predictors of pulmonary infarction

Variable	β	Wald	P value	OR	95% CI
Age	−0.025	4.023	0.045	0.975	0.951–0.999
Dyspnoea	0.464	1.043	0.307	1.591	0.653–3.876
Pleuritic chest pain	1.057	8.677	0.003	2.878	1.424–5.814
Hemoptysis	2.360	17.475	<0.001	10.592	3.503–32.030
Fever	0.802	2.760	0.097	2.230	0.866–5.746
Lower limb edema/pain	1.022	7.582	0.006	2.778	1.342–5.749
Coronary heart disease	−0.276	0.348	0.555	0.759	0.304–1.895
Diabetes mellitus	−0.344	0.381	0.537	0.709	0.238–2.114
Recent trauma	0.944	3.032	0.082	2.598	0.853-7.919
Recent immobilisation	−0.617	0.908	0.341	0.539	0.152–1.920
WBC (>10×10^9/L)	0.075	0.040	0.841	1.078	0.517–2.250
CRP (>10 mg/L)	0.530	2.085	0.149	1.699	0.827–3.490
Pleural effusion	1.140	9.788	0.002	3.127	1.531–6.388

OR, odds ratio; CI, confidence interval; WBC, white blood cell; CRP, C-reactive protein.

Miniati *et al.* (6) thought that it might be connected to the effectiveness of collateral circulation in peripheral lung tissues. Our study showed the highest frequency of onset was in the age group of under 30 years (58.33%), followed by 30s (50.00%), 40s (18.18%), 50s (20.45%), and 60s (18.25%). Islam *et al.* (5) reported most patients with PI were in their 20s and 30s, and Miniati *et al.* (6) reported most patients were in their 40s.

Hampton and Castleman (17) found in a cohort with 370 patients with PE confirmed by autopsy that the highest PI prevalence was in patients with a long history of left heart failure. Previous studies (9,10) proposed the mechanism of raised pulmonary venous pressure and decreased flow of bronchial circulation for PI patients with cardiac disease. In contrast, recent studies (5,6) revealed that individuals with PI exhibited a significantly lower incidence of cardiovascular disease in comparison to those with PE but without PI and noted that bronchopulmonary collateral circulation was developed more frequently in people with chronic cardiopulmonary disorders. Our study found that PI was independent of comorbidities, such as chronic respiratory disease, cardiovascular disease (including heart failure), and malignancy. Moreover, our study was unable to demonstrate that smoking history was associated with PI. Prior literature (6,21) suggested smoking caused an inflammatory reaction and made the alveolar-capillary barrier more permeable, which might raise the risk of PI. However, we hypothesized that smoking produces decreased oxygen supply to tissues, which in turn leads to development of more vascular collateralization. Of note, several traditional PE risk factors, such as the history of DVT, varicose veins of lower limbs, recent surgery, hormone therapy, and oestrogen use, were not associated with PI.

The clinical symptoms and imaging features of PI lack specificity. It may be misdiagnosed as lung infection or tumor (22). The consolidation is not specific to PI, because it can also be observed in pneumonia (23), tuberculosis, and invasive fungal infection (IFI). The internal morphologic characteristics of consolidation could be differentiated from other causes. A reversed halo sign (RHS) is a typical sign of PI. PI is a peripheral wedge-shaped consolidation with central lucencies, low-attenuation areas (with or without reticulation), vessel sign, and the absence of air bronchograms (19). In the early phase, it can present ground-glass opacity (24). Likewise typical chest CT findings for pneumonia are patchy consolidations with air bronchograms. Tuberculosis is frequently accompanied by tree-in-bud signs and nodules. Reticulation inside the RHS is also very common in IFI. It should be interpreted with consideration of the overall clinical presentation and the patient's immune status. In an immunocompromised patient, the RHS with reticulation is highly suggestive of

IFI. With increasing awareness and improved diagnosis of PI, the rate of misdiagnosis decreases, and this can potentially prevent unnecessary antibiotic use. In our study, 25% of PI patients were administered with antibiotics. Peripheral vascular obstruction is more likely to lead to infarction than a large central blood clot burden, and PI is more common in pulmonary artery branch occlusion with a diameter of 3 mm or less (5). The infarct lesions are usually based on the pleura, causing hemoptysis due to lung parenchyma coagulative necrosis and chest pain owing to pleural irritation. The prevalence of pleuritic chest pain and hemoptysis exhibited a significant elevation in the PI group than in the non-PI group. Another important finding was that some PI patients were accompanied by fever (11.2%). Fever may be caused by inflammatory cascade and tissue necrosis, or it may occur secondary to the immune response of vascular and serosal remodeling (25). We assumed that immunity is an important mechanism of fever, similar to Dressler's syndrome. Some researchers have put forward the concept of Dressler-like syndrome (26).

PI is more common in the lower lobe of the lungs. Impaired alveolar oxygenation in the lower lobe of the lung is caused by lower alveolar oxygen tension at the base of the lung in the upright position, as well as basal atelectasis due to pleural pressure gradient (27). And pleural effusion was more frequently discovered in PE patients with PI than those without PI in the current investigation. The hemorrhagic necrosis of infarction causes more inflammatory mediators to be released and the permeability of pleural capillaries to be increased, thus inducing or aggravating pleural effusion, and a higher thrombus load is more likely to combine with pleural effusion (28). In addition, due to blood flow damage, the hydrostatic pressure inside the pleural capillaries increases, and lymphatic reflux is obstructed, resulting in pleural effusion. The amount of pleural effusion in PI patients is usually relatively small (with an average effusion layer of 10.6 mm), but the amount of pleural effusion may not necessarily be related to the size of the PI (29).

Although PI means necrosis of the lung parenchyma, the term is defined in a variety of ways in medical research. Histopathologic evidence, imaging findings, and clinical features are used as three different categories of criteria. Radiological findings are the most common method used for the diagnosis of PI. The peripheral wedge-shaped consolidation with nearby (sub) segmental PE is a frequently used criterion of PI in PE on CT. Apart from individual differences, the inconsistencies in understanding the diagnostic standards for PI may be responsible for the wide difference in the incidence rates. Therefore, it is imperative for clinicians to have a comprehensive understanding of PI features that can improve diagnosis precision.

PI may be an important diagnostic clue or even the only sign of acute PE. The management of PI patients is consistent with PE (30). These patients should receive anticoagulant or thrombolytic therapy as soon as possible. In addition, non-steroidal anti-inflammatory drugs can be administered to alleviate severe chest pain in patients with PE and PI. PE often causes a small amount of hemoptysis, which is usually treated symptomatically. However, in the case of severe hemoptysis, it can be quite challenging. If massive dyspnea has not been relieved or hypoxia has been aggravated, tracheal intubation should be done immediately to keep the respiratory tract open. In our study, patients with PE did not experience massive hemoptysis. High-risk patients with hemodynamic instability, including cardiac arrest, obstructive shock, or persistent hypotension should be immediately admitted to the relevant intensive care unit (31). And the presence of PI was not associated with mortality.

It should be noted that the current study is of a retrospective nature, and the sample size utilized is comparatively limited. Furthermore, certain variables were not assessed, including the pulmonary artery obstruction index. Eventually, the data were collected in a single institution. The differences in patient selection among experts could lead to bias.

Conclusions

Young patients were found to be of a higher risk of PI. PI should be considered when pleuritic chest pain, hemoptysis, or lower limb edema/pain are present with peripheral opacity. Pleural effusion was found to be a factor for PI.

Acknowledgments

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Footnote

Reporting Checklist: The authors have completed the STROBE reporting checklist. Available at https://jtd.amegroups.com/article/view/10.21037/jtd-23-944/rc

Data Sharing Statement: Available at https://jtd.amegroups.

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Conflicts of Interest: All authors have completed the ICMJE uniform disclosure form (available at https://jtd.amegroups.com/article/view/10.21037/jtd-23-944/coif). The authors have no conflicts of interest to declare.

Ethical Statement: The authors are accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved. The study was conducted in accordance with the Declaration of Helsinki (as revised in 2013). The retrospective study was approved by the Ethics Committee of Beijing Luhe Hospital, Capital Medical University (No. 2021-LHKY-119-02). Individual consent was waived due to the retrospective nature of this study.

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