

# Frequency of Pulmonary Embolism in Patients with COPD Exacerbation and Clinical and Laboratory Predictive Factors

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## Abstract

**Background:** Chronic obstructive pulmonary disease (COPD) is the leading cause of respiratory morbidity and mortality. Patients with COPD have an increased risk of pulmonary thromboembolism (PTE), which complicates clinical management. This study aimed to evaluate the frequency of PTE in patients with COPD exacerbations and to identify clinical and laboratory predictive factors associated with PTE.

**Materials and Methods:** A retrospective cross-sectional study was conducted at Al-Zahra Hospital, Isfahan, Iran, between 2021-2022. Patients diagnosed with COPD exacerbation and shortness of breath were assessed for PTE by using computed tomography pulmonary angiography. Clinical data, including demographics, blood gas levels, and ECG and echocardiography findings, were collected and analyzed using SPSS 24 software.

**Results:** Among the 260 COPD patients, 26 (10%) were diagnosed with PTE. Patients with PTE exhibited significantly higher body mass index (BMI), smoking and D-dimer levels, and lower oxygen saturation. Logistic regression analysis identified older age, BMI, smoking, and abnormal electrocardiogram (ECG) as independent predictors of PTE ( $P < 0.001$  for all).

**Conclusion:** This study highlights the notable prevalence of PTE in patients with COPD exacerbations. Factors such as obesity, smoking, and abnormal ECG findings are critical in predicting the risk of PTE. These findings underscore the necessity for vigilant assessment and management of PTE in COPD patients to improve clinical outcomes in this vulnerable population.

**Keywords:** Chronic obstructive pulmonary disease, exacerbation, pulmonary embolism, risk factors

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## INTRODUCTION

Chronic obstructive pulmonary disease (COPD) is the most common cause of respiratory-related mortality and disability.<sup>[1]</sup> COPD is a broad classification that includes a group of diseases associated with the chronic obstruction of airflow inside or outside the lungs. Airway obstruction is the diffuse narrowing of the airways, which increases the resistance to air passage. Diseases such as chronic bronchitis, bronchiectasis, emphysema, and asthma are included in COPD.<sup>[2]</sup>

Although some lung functions, such as vital capacity and expiratory volume, decrease with age, COPD exacerbates

many of these changes and eventually blocks the airway. Signs of overexpansion of the lungs, use of accessory respiratory muscles, decreased breath sounds on examination, and diffuse wheezing (especially during deep exhalation) have been observed.<sup>[3]</sup> The appearance of patients differs from a thin and even cachectic appearance to an edematous and cyanotic face. Arterial blood gas tests usually show hypoxia with variable intensities, and hypercapnia is also observed in the advanced stages of the disease.<sup>[4,5]</sup> The most common cause of COPD is smoking, although other factors, such as air pollution and heredity, play a lesser role. Diagnosis of this disease is based

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on shortness of breath, which is measured using lung function tests. It can be prevented by reducing exposure to known factors of this disease. This is accompanied by efforts to reduce smoking and increase indoor and outdoor air quality. Treatments for COPD include smoking cessation, vaccination, rehabilitation, and inhaled bronchodilators and corticosteroids. Long-term oxygen therapy or lung transplantation may be appropriate in some patients.<sup>[6]</sup>

According to this definition, acute exacerbation of COPD is defined as any change in the natural course of the disease in the form of a change or an increase in the amount of sputum that has occurred acutely and is out of the usual daily changes and increases the patient's need for daily medications, increasing the level of basic shortness of breath, increasing the cough, and changing its nature. In acute exacerbation of COPD, oxygen is prescribed to maintain  $\text{PaO}_2 > 60$  mmHg and  $\text{SaO}_2 > 90\%$ .<sup>[7]</sup> The prescribed oxygen concentration varies according to the severity of the patient's symptoms and underlying diseases. Arterial blood gases are routinely checked at the beginning and 30 min after oxygen administration to check the adequacy of oxygenation as well as the amount of  $\text{CO}_2$  retention.<sup>[8]</sup>

Pulmonary thromboembolism (PTE) is defined as the presence of a blood clot or fat in one of the pulmonary arteries or lung tissue. This blood clot first forms in one of the deep veins of the lower limbs or pelvis. Fat embolisms are mostly based on the area of the bone fracture. A blood clot travels through the bloodstream, passes through the heart to one of the pulmonary arteries, and settles there.<sup>[9]</sup> This phenomenon causes the artery to close, and therefore, the ability to breathe decreases, and sometimes the lung tissue is lost. One of the most important symptoms found in patients with pulmonary embolism (PE) is shortness of breath.<sup>[10]</sup>

Studies have shown that patients with COPD may develop PTE more frequently than other patients.<sup>[11]</sup> Because dyspnea is common in patients with COPD exacerbation as well as in patients with PTE, the diagnosis of PTE in these patients is more difficult than in others. Other symptoms such as pain in one of the lower limbs, hemoptysis, tachycardia, unilateral edema of the lower limb, clinical symptoms of DVT, hemoptysis, presence of other differential diagnoses that justify the symptoms, pregnancy and D-dimer level, tachycardia and immobility for more than 3 days, or recent surgery can help distinguish the diseases.<sup>[12]</sup> Considering the importance of COPD and its high prevalence in the elderly, as well as the overlap of its symptoms with PTE and the high mortality of PTE, epidemiological data have indicated a wide range of prevalence in different populations. Various studies have evaluated the prevalence of PTE and its clinical and laboratory risk factors in different populations. However, to our knowledge, very few studies have investigated these results in our country. Therefore, we determined the prevalence of PTE and its clinical and laboratory risk factors to investigate COPD exacerbations in Iran.

## MATERIALS AND METHODS

### Study design

This retrospective cross-sectional study was performed between 2021 and 2022 in Al-Zahra Hospital affiliated with Isfahan University of Medical Science. This study was conducted on patients with COPD. The Research Committee of Isfahan University of Medical Sciences and Ethics Committee approved the study (Ethics code: IR.MUI.MED.REC.1401.093).

### Inclusion and exclusion criteria

The inclusion criteria were diagnosis of COPD based on signs and symptoms, spirometry and previous records, admission with shortness of breath, symptoms of COPD exacerbation, and written informed consent to participate in this study.

The diagnosis of COPD was confirmed, and its severity was determined according to the criteria of the Global Initiative for Chronic Obstructive Lung Disease (GOLD).<sup>[13]</sup> Moreover, COPD exacerbation occurs when a patient's respiratory symptoms (dyspnea, cough, sputum, and fever) worsen beyond daily expectations and necessitates adjustments to their medication treatment regimens.<sup>[14]</sup>

Patients with the following criteria did not enter the study: (1) taking anticoagulant drugs such as warfarin, (2) active cancer, (3) having surgery or prolonged immobility in the last 2 months, and (4) patients who are not allowed to receive contrast for imaging. The exclusion criteria were interstitial lung diseases or congenital pulmonary diseases, such as cystic fibrosis.

### Study population selection and data gathering

In this study, all patients with COPD referred to the emergency department of Al-Zahra Hospital with respiratory problems and suspected COPD exacerbation in 2021–2022 were investigated using the census method.

Information regarding physical examinations, medical history, and other required items was collected from the patients' medical records. Additionally, the amount of arterial blood gas was measured using laboratory methods. In addition, all patients were requested to have an electrocardiogram (ECG) upon admission to detect cardiac abnormalities, such as blocks, atrial fibrillation (AF), ST changes, or S1Q3T3 pattern. Echocardiography was also performed to check for evidence of PTE, such as right ventricular hypokinesis or intraventricular septum deviation. Pulmonary function tests were also performed.

### PTE diagnosis

Patients with COPD underwent computed tomography pulmonary angiography (CTPA) in cases of strong clinical suspicion and based on the diagnostic algorithm of PE (combination of clinical probability and D-dimer) in cases of (high probability) need for a definitive diagnosis of PTE. PTE was diagnosed as a defect in intraluminal filling or closure of the lumen of the pulmonary vessels. Patients were divided into two groups based on the diagnosis and absence of PTE.

Demographic information of patients such as age and sex and information such as systolic and diastolic blood pressure and blood oxygen saturation, history of DVT or PTE, pain in one of the lower limbs, hemoptysis, tachycardia and unilateral edema of the lower limb, and the level of D-dimer were also collected from the patients' medical records.

### Statistical analysis

The recorded data were analyzed and compared using the SPSS 24 software for the two groups of patients with and without PTE. The mean/standard deviation was used to describe quantitative variables, and frequency/percentage was used for qualitative variables. We used an independent *t*-test to compare quantitative variables between the two groups and a Chi-square test to compare qualitative variables between the two groups. Logistic regression analysis was used to determine the predictive power of different factors for the development of PE. Statistical significance was set at  $P < 0.05$ .

## RESULTS

Evaluation of medical documents revealed 260 cases of COPD exacerbation that were admitted to our medical center. Of these, 26 were diagnosed with PTE and were considered the main group. We randomly selected 234 patients with COPD and no PTE as the control group (9:1 ratio) that were matched with the main group regarding demographic data.

Data from 260 patients were analyzed. The study population consisted of 174 females (66.9%) and 86 males (33.1%) with a mean age of  $59.52 \pm 11.18$  years. Primary analysis revealed that 26 patients (10%) were diagnosed with PTE. Patient data are shown in Table 1.

We compared different data between patients with and without PTE. Data showed that patients with PTE had higher body mass index (BMI), smoking level, lower  $\text{PCO}_2$  levels, limb pain, higher D-dimer level, higher rates of hemoptysis, and higher systolic blood pressure ( $P < 0.001$  for all). Patients with PTE had lower rates of normal ECG and echocardiography, blood pH, and oxygenation saturation ( $P < 0.001$  for all). Data are shown in Table 2.

By evaluating different risk factors of PTE in COPD patients, it was observed that higher age (OR = 1.622), higher BMI (OR = 1.824), smoking (OR = 1.841), and abnormal ECG (OR = 1.153) increased the risks of PTE ( $P < 0.001$  for all). Data are shown in Table 3.

## DISCUSSION

The frequency of PTE in COPD patients is an important aspect to consider in clinical practice. Understanding PTE's clinical and laboratory predictive factors can help identify high-risk patients and facilitate timely diagnosis and treatment. In a recent study, several factors were significantly associated with PTE in COPD patients.<sup>[15]</sup>

**Table 1: Demographic and clinical data of patients**

| Variables                       | n (%) / mean $\pm$ SD |
|---------------------------------|-----------------------|
| Age (year)                      | 59.52 $\pm$ 11.18     |
| Gender                          |                       |
| Male                            | 86 (33.1%)            |
| Female                          | 174 (66.9%)           |
| PTE                             | 26 (10%)              |
| BMI (kg/m <sup>2</sup> )        | 29.19 $\pm$ 6.33      |
| Smoking (pack*year)             | 19.17 $\pm$ 7.21      |
| Normal ECG                      | 91 (35%)              |
| Normal echocardiography         | 210 (80%)             |
| Abnormal ECG                    |                       |
| Tachycardia (>100/min)          | 16 (6.1%)             |
| Bradycardia (<50/min)           | 8 (3%)                |
| PVC                             | 13 (5%)               |
| Right axis deviation            | 10 (3.8%)             |
| Left axis deviation             | 14 (5.3%)             |
| Undetermined axis               | 7 (2.6%)              |
| P-pulmonale                     | 8 (3%)                |
| AV block                        | 10 (3.4%)             |
| RBBB                            | 11 (3.8%)             |
| LBBB                            | 8 (3%)                |
| SIQ3T3 pattern                  | 14 (5.3%)             |
| Atrial fibrillation             | 11 (4.2%)             |
| Poor R-wave progression         | 14 (5.3%)             |
| ST/T-wave changes               | 16 (6.1%)             |
| Myocardial infarction           | 9 (3.4%)              |
| Echocardiographic findings      |                       |
| Right atrial dilatation         | 9 (3.4%)              |
| Right ventricular dilatation    | 16 (5.5%)             |
| Pulmonary hypertension          | 15 (5.7%)             |
| Right ventricular hypertrophy   | 10 (3.8%)             |
| Lower limb pain                 | 39 (15%)              |
| Blood pH                        | 7.37 $\pm$ 0.08       |
| D- Dimer (mg/L)                 | 1.51 $\pm$ 0.79       |
| Hemoptysis                      | 16 (6.1%)             |
| Systolic blood pressure (mmHg)  | 137.82 $\pm$ 22.32    |
| Diastolic blood pressure (mmHg) | 91.91 $\pm$ 12.62     |
| Oxygenation saturation (%)      | 88.20 $\pm$ 4.84      |
| PCO <sub>2</sub> levels (mmHg)  | 62.38 $\pm$ 25.19     |

ECG=electrocardiogram, PVC=premature ventricular contraction, AV block=atrioventricular block, RBBB=right bundle branch block, LBBB=left bundle branch block

One of the observed findings of this study was that patients with PTE had higher BMI than those without PTE. Obesity has been identified as a risk factor for PTE due to its association with inflammation, impaired lung function, and altered coagulation pathways.<sup>[16]</sup> The increased BMI in patients with PTE suggests that weight management should be considered in COPD patients to potentially reduce the risk of PTE.<sup>[17]</sup>

Another significant factor associated with PTE in COPD exacerbation was the smoking level. Smoking is a well-known risk factor for both COPD and PTE. The detrimental effects of smoking on lung function and its prothrombotic properties contribute to the development of PTE. Therefore, smoking

**Table 2: Comparison of demographic and clinical data between patients with and without PTE**

| Variables                       | With PTE<br>(n=26) | Without PTE<br>(n=234) | P        |
|---------------------------------|--------------------|------------------------|----------|
| Age (year) (mean±SD)            | 62.27±7.20         | 62.19±9.87             | 0.711*   |
| Gender (n (%))                  |                    |                        |          |
| Male                            | 8 (30.8%)          | 74 (31.6%)             | 0.632**  |
| Female                          | 18 (69.2%)         | 160 (68.4%)            |          |
| BMI (kg/m <sup>2</sup> )        | 30.16±6.44         | 28.13±6.83             | <0.001*  |
| Smoking (pack*year)             | 24.07±8.67         | 18.91±8.25             | <0.001*  |
| Normal ECG                      | 6 (23.1%)          | 85 (36.3%)             | <0.001** |
| Normal echocardiography         | 17 (65.4%)         | 183 (78.2%)            | <0.001** |
| Lower limb pain                 | 21 (80.8%)         | 18 (7.7%)              | <0.001** |
| Blood pH                        | 7.36±0.08          | 7.37±0.08              | 0.833*   |
| D-Dimer (mg/L)                  | 1.86±0.62          | 0.67±0.43              | <0.001*  |
| Hemoptysis                      | 9 (34.6%)          | 7 (2.9%)               | <0.001** |
| Systolic blood pressure (mmHg)  | 144.35±18.71       | 136.37±22.20           | <0.001*  |
| Diastolic blood pressure (mmHg) | 91.55±11.12        | 92.64±13.51            | 0.331*   |
| Oxygenation saturation (%)      | 86.25±2.37         | 88.15±3.19             | <0.001*  |
| PCO2 levels (mmHg)              | 44.37±6.82         | 69.21±12.07            | <0.001*  |

BMI=body mass index, ECG=electrocardiogram. \*Independent *t*-test, \*\*Chi-square test

**Table 3: Logistic regression for different drug use and risks of PTE**

| Variables                 | P*     | Odds ratio (OR) | CI (95%) |       |
|---------------------------|--------|-----------------|----------|-------|
|                           |        |                 | Lower    | Upper |
| Higher age                | <0.001 | 1.622           | 1.26     | 1.47  |
| Higher BMI                | <0.001 | 1.824           | 1.18     | 1.63  |
| Smoking                   | <0.001 | 1.841           | 1.16     | 1.42  |
| Abnormal ECG              | <0.001 | 1.153           | 1.22     | 1.65  |
| Abnormal echocardiography | 0.082  | 1.068           | 0.83     | 1.25  |

BMI=body mass index, ECG=electrocardiogram. \*Logistic regression

cessation interventions should be implemented in COPD patients not only to improve their respiratory health but also to reduce the risk of PTE.

Lower limb pain, D-dimer levels, hemoptysis, and systolic blood pressure were also identified as significant factors associated with PTE in patients with COPD. Lower limb pain may indicate the presence of DVT, which is a precursor of PTE. D-dimer level, a marker of fibrin degradation, is frequently elevated in the presence of thrombosis. Hemoptysis, the coughing of blood, is a symptom that may suggest underlying pulmonary vascular abnormalities. Systolic blood pressure may reflect the hemodynamic consequences of PTE. Recognizing these clinical features and performing appropriate diagnostic tests, such as lower-limb ultrasonography and CTPA, can aid in the early detection of PTE in COPD patients.

Furthermore, patients with PTE had lower rates of normal ECG and echocardiography findings, as well as lower blood

pH and oxygenation saturation. Abnormal ECG findings such as right ventricular strain patterns are commonly observed in PTE. Echocardiography can provide valuable information on cardiac function and the presence of pulmonary hypertension, which may be secondary to PTE. Lower blood pH and oxygenation saturation levels reflect respiratory compromise associated with PTE. These findings emphasize the importance of performing ECG, echocardiography, and blood gas analysis in patients with COPD with suspected PTE to guide appropriate management.

By evaluating different risk factors, the study identified older age, higher BMI, smoking, and abnormal ECG as independent predictors of PTE in COPD patients. Advanced age is a well-established risk factor for PTE, possibly because of age-related changes in the coagulation system and decreased mobility. The association between BMI, smoking status, and PTE has been discussed previously. Abnormal ECG findings such as right ventricular strain patterns can indicate the presence of PTE-related cardiac stress.

These factors have been evaluated in previous research. As described by Sadeghi and colleagues in 2021, PTE was confirmed in one-fifth of suspected patients who underwent CTPA imaging. Male sex, decreased oxygen saturation, and lower levels of hemoglobin and albumin were independent predictors of PTE in patients with COVID-19 pneumonia.<sup>[18]</sup> In another study by Cao, they showed that COPD patients with PE experienced an increased risk of death and prolonged length of hospital stay. Therefore, the thromboembolic risk in patients with acute exacerbation of COPD, especially in hospitalized patients, should be carefully evaluated.<sup>[19]</sup> Furthermore, Kim and colleagues<sup>[20]</sup> showed that mild-to-moderate COPD was not a significant risk factor for postoperative pulmonary complications. However, previous hospitalization for respiratory problems, emergency surgery, increased amount of red blood cell (RBC) transfusion, and laparoscopic surgery were independent predictors of PPCs. These findings are somehow in line with the results of our study. However, only limited studies have been conducted in our region. Thus, our findings could have high clinical values.

Although this study provides valuable insights into the frequency of PTE in patients with COPD exacerbation and the associated clinical and laboratory predictive factors, it has several limitations. First, its retrospective design may compromise data accuracy and introduce selection bias, as information was gathered post-treatment from the medical records. Second, this single-center study restricts the generalizability of the findings to other healthcare settings with varying patient populations and management practices. Additionally, the relatively small sample size may have limited the statistical power and detection of significant differences. The absence of long-term follow-up data restricts the understanding of the natural history of PTE in patients with COPD. Furthermore, the study lacked comprehensive information on COPD severity and did not employ standardized

diagnostic criteria, potentially affecting the reliability of the findings. Lastly, it did not consider all possible confounding factors, such as comorbidities and lifestyle influences, which might have impacted the observed association between COPD exacerbations and PTE.

## CONCLUSION

The results of the study highlight the significance of recognizing and understanding the clinical and laboratory predictive factors associated with PE in patients with COPD exacerbation. Higher BMI, smoking, lower PCO<sub>2</sub> levels, limb pain, D-dimer levels, hemoptysis, and systolic blood pressure were identified as potential indicators of increased risk for PTE in this patient population. Additionally, abnormal ECG, echocardiography, blood pH, and oxygenation saturation were found to be associated with PTE in COPD patients.

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## Conflicts of interest

There are no conflicts of interest.

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