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PREVALENCE OF PULMONARY EMBOLISM IN ACUTE EXACERBATION OF COPD : AN OBSERVATIONAL STUDY AT A TERTIARY CARE CENTER OF WESTERN RAJASTHAN

Respiratory Medicine

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KEYWORDS

INTRODUCTION

Chronic obstructive pulmonary disease (COPD) is a leading cause of morbidity and mortality worldwide, with acute exacerbations (AECOPD) representing critical events that accelerate lung function decline, drive hospital admissions, and increase the risk of death. According to the GOLD 2025 report, COPD remains a major global health burden, particularly in low- and middle-income countries, where the frequency and severity of exacerbations are higher and access to advanced diagnostic resources is limited [1].

Pulmonary embolism (PE) has emerged as a clinically important but frequently underrecognized comorbidity in patients with AECOPD. The overlap in clinical manifestations-such as dyspnea, hypoxemia, and chest discomfort-creates significant diagnostic uncertainty. Multiple large-scale studies and systematic reviews have demonstrated a variable but consistently substantial prevalence of PE among patients hospitalized with AECOPD, ranging between 5% and 29%, depending on the population studied and the diagnostic strategies employed [2–8]. This underlines the necessity for heightened clinical vigilance and tailored diagnostic approaches.

Recent evidence indicates that the coexistence of PE in AECOPD is associated with increased morbidity, longer hospital stay, greater use of intensive care resources, and higher short-term and long-term mortality [9–12]. In addition to clinical overlap, systemic inflammation, immobility during severe exacerbations, and frequent glucocorticoid use may predispose patients to venous thromboembolism (VTE), further complicating management [13,14].

Despite advances in diagnostic imaging and biomarker development, uncertainty remains regarding optimal strategies to balance timely detection of PE with judicious use of resources in this high-risk population. Furthermore, meta-analyses suggest that reported prevalence may vary substantially depending on whether all patients undergo systematic imaging versus selective imaging guided by clinical suspicion [4,13]. These findings highlight the need for region-specific data to guide evidence-based practice, particularly in resource-constrained settings.

Against this background, the present study was designed to investigate the prevalence of PE in patients admitted with AECOPD at a tertiary care center in Western Rajasthan. By addressing diagnostic challenges and contextualizing findings within the latest global evidence, this work aims to provide insights that may inform clinical decision-making and resource allocation in similar health care environments.

MATERIALS AND METHODS

Study Design And Duration

This hospital-based observational study was conducted in the Department of Respiratory Medicine, Kamla Nehru Chest Hospital, Jodhpur, from February 2020 to February 2021, after approval from the Institutional Ethics Committee.

Inclusion Criteria

Patients were eligible if they met the following:

- Diagnosis of AECOPD according to GOLD guidelines.
- Hospital admission due to acute exacerbation of COPD.
- Provision of written informed consent.

Exclusion Criteria

- Patients were excluded if they had:
- Refusal to provide written informed consent.
- Renal failure (serum creatinine >150 µmol/L).
- Known allergy to intravenous contrast medium.
- Ongoing long-term anticoagulant therapy.
- Pregnancy.
- Known malignancy.
- Surgery within the preceding 4 weeks.
- Recent bone fracture.
- Past or active pulmonary tuberculosis.

Study Protocol

All hospitalized patients with AECOPD fulfilling eligibility criteria were enrolled after informed consent. Each patient underwent D-dimer testing and arterial blood gas (ABG) analysis at admission. Those with elevated D-dimer levels (>0.5 µg/mL) were further evaluated using two-dimensional echocardiography (2D ECHO) and spiral computed tomographic pulmonary angiography (CTPA) within 48 hours of admission.

Sample Size

Sample size was calculated at a 95% confidence level, assuming a 25% prevalence of pulmonary embolism in AECOPD based on prior studies, with a 10% absolute allowable error. The minimum required sample was 72 patients. To enhance study power, the final sample size was increased to 80.

Statistical Analysis

Categorical variables were expressed as frequencies and percentages, and comparisons were performed using the Chi-square test. Continuous variables were expressed as mean ± standard deviation (SD) and compared using the Student's t-test. A two-tailed p-value <0.05 was considered statistically significant.

RESULTS

Baseline Characteristics and Prevalence of Pulmonary Embolism. A total of 80 patients admitted with AECOPD were included. Of these, 66 (82.5%) were male and 14 (17.5%) were female. The sex distribution did not differ significantly between patients with and without pulmonary embolism. Pulmonary embolism was detected in 7 patients, yielding a prevalence of 8.75%.

Category	Total (N=80)	AECOPD without PE (N=73)	AECOPD with PE (N=7)
Male, n (%)	66 (82.5)	60 (82.2)	6 (85.7)
Female, n (%)	14 (17.5)	13 (17.8)	1 (14.3)
Prevalence of PE (%)	–	–	8.75

Pulmonary Hypertension

The mean pulmonary arterial pressure (PAP) was significantly higher in patients with PE (47.0 ± 10.3 mmHg) compared with those without PE (31.1 ± 12.9 mmHg, p = 0.002).

Spirometry Findings

Spirometric indices, including FEV1/FVC ratio, FEV1, and FVC,

were lower in patients with PE compared to those without; however, the differences were not statistically significant.

Spirometry indices	AECOPD without PE (Mean ± SD)	AECOPD with PE (Mean ± SD)	p-value	All patients (Mean ± SD)
FEV1/FVC	46.7 ± 11.9	41.2 ± 10.6	0.243	46.2 ± 11.9
FEV1 (% predicted)	49.4 ± 15.4	38.7 ± 15.6	0.084	48.4 ± 15.6
FVC (% predicted)	80.1 ± 15.2	71.3 ± 18.2	0.154	79.4 ± 15.5

GOLD Staging

The distribution of COPD severity based on GOLD staging is shown below. The proportion of patients with very severe COPD (GOLD stage 4) was significantly higher among those with PE (42.9% vs. 9.6%, p = 0.011).

GOLD Stage	AECOPD without PE (N=73) n (%)	AECOPD with PE (N=7) n (%)	p-value	Total (N=80) n (%)
GOLD 1 (Mild)	1 (1.4)	0 (0.0)	0.754	1 (1.3)
GOLD 2 (Moderate)	35 (47.9)	2 (28.6)	0.331	37 (46.3)
GOLD 3 (Severe)	30 (41.1)	2 (28.6)	0.300	32 (40.0)
GOLD 4 (Very severe)	7 (9.6)	3 (42.9)	0.011	10 (12.5)

Arterial Blood Gas (ABG) Analysis

Patients with PE had significantly worse hypoxemia and hypocapnia compared with those without PE. Mean PaO_2 was 44.1 ± 6.9 mmHg vs. 65.7 ± 11.2 mmHg ($p < 0.001$), and mean $PaCO_2$ was 37.9 ± 12.1 mmHg vs. 48.0 ± 9.7 mmHg ($p = 0.011$). No significant differences were observed in pH or HCO_3^-.

Parameter	AECOPD without PE (Mean ± SD)	AECOPD with PE (Mean ± SD)	p-value	All patients (Mean ± SD)
pH	7.40 ± 0.10	7.43 ± 0.07	0.864	7.40 ± 0.10
PaO_2 (mmHg)	65.7 ± 11.2	44.1 ± 6.9	<0.001	63.8 ± 12.4
$PaCO_2$ (mmHg)	48.0 ± 9.7	37.9 ± 12.1	0.011	48.6 ± 10.4
HCO_3^- (mmol/L)	25.9 ± 5.0	23.5 ± 6.4	0.230	26.2 ± 5.0

DISCUSSION

Pulmonary embolism (PE) is an under recognized but clinically important comorbidity in patients admitted with acute exacerbation of chronic obstructive pulmonary disease (AECOPD). The current study, conducted in a tertiary care center of western Rajasthan in India, demonstrated a PE prevalence of 8.75%, which, although lower than some international reports, underscores the need for heightened clinical suspicion.

Our findings align with previous evidence suggesting that the prevalence of PE in AECOPD varies widely, typically ranging between 5% and 30%, depending on study design, diagnostic modalities, and patient selection criteria (2,4–8). Couturaud et al. reported a prevalence of 5.9% using systematic CTPA in hospitalized COPD patients (8), whereas Wang et al. and Han et al. highlighted in their meta-analyses that pooled prevalence estimates often exceed 15% (4,5). These differences suggest that prevalence estimates are influenced by diagnostic strategies and patient cohorts, a finding further emphasized by Li et al. and de Miguel-Díez et al., who demonstrated variability across different healthcare systems (3,7).

Importantly, our study identified a significant association between PE and pulmonary hypertension/cor pulmonale. The mean pulmonary artery pressure was markedly higher in patients with PE compared to those without, consistent with the vascular alterations and right heart strain described in previous studies (10,12). COPD-related vascular remodeling, endothelial dysfunction, and hypoxic vasoconstriction may amplify the hemodynamic burden of even small emboli, predisposing patients to adverse outcomes (2,5).

The spirometry findings indicated more severe airflow limitation among patients with PE compared to those without, though these differences were not statistically significant. Similar patterns have been reported by Ilievska et al., who suggested that systemic inflammation, hypoxemia, and reduced mobility in severe COPD may predispose to venous thromboembolism independent of spirometric severity (11). The GOLD 2025 report also emphasizes the systemic nature of COPD, noting that comorbidities, including cardiovascular and thromboembolic disease, significantly impact prognosis (1).

Arterial blood gas (ABG) analysis further highlighted that patients with PE had significantly lower PaO_2 and $PaCO_2$ levels, which could serve as an additional clinical clue to suspect PE in AECOPD. Hypoxemia disproportionate to the degree of airway obstruction should prompt clinicians to consider vascular etiologies (3,7,8). This finding supports earlier reports that hypoxemia, particularly when refractory to standard therapy, is a red flag for underlying embolic disease.

Another critical aspect is the potential role of glucocorticoids in increasing VTE risk. A population-based study demonstrated that systemic glucocorticoid use was associated with a significantly elevated risk of venous thromboembolism, including PE (14). Given that glucocorticoids remain central to AECOPD management, this association warrants careful clinical consideration, particularly in patients with additional thromboembolic risk factors.

In summary, this study demonstrates that PE is not exceedingly common but remains a clinically significant complication in AECOPD. Early recognition and timely management are essential to improving patient outcomes. Our findings reinforce the importance of a systematic diagnostic approach—incorporating clinical suspicion, biomarkers, echocardiography, and CTPA when indicated—in patients with unexplained hypoxemia or evidence of right heart strain.

Limitations & Future Scope

This was a single-center study with a relatively small sample size (n=80), which may limit the generalizability of findings.Exclusion of patients with renal dysfunction, malignancy, or recent surgery may have led to underestimation of the true prevalence of pulmonary embolism (PE) in AECOPD.The use of CTPA as the sole diagnostic modality and the cross-sectional design prevent establishing causal relationships. Larger multicenter and longitudinal studies are warranted to validate these findings and provide broader generalizability. Exploration of novel biomarkers and clinical prediction tools could aid in early identification of high-risk patients.

CONCLUSION

Pulmonary embolism is a clinically significant comorbidity among patients admitted with acute exacerbation of COPD. In this study, the prevalence was found to be 8.75%, with higher occurrence in patients presenting with pulmonary hypertension and cor pulmonale. These findings emphasize the importance of maintaining high clinical suspicion for PE in AECOPD cases, particularly in elderly, sedentary patients or those with severe COPD and evidence of right heart strain. Timely diagnosis and management can help reduce morbidity and mortality in this population.

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