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Blood Biomarkers in Patients with COPD with Coexisting Bronchiectasis: A Cross-Sectional Study

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Abstract:

- ***Background:***

Chronic Obstructive Pulmonary Disease (COPD) and bronchiectasis frequently coexist, leading to more severe disease outcomes and an increased inflammatory burden. This study evaluates blood parameters in COPD patients with and without bronchiectasis to determine their significance in disease prognosis and management.

- ***Methods:***

A cross-sectional study of 70 COPD patients (20 with bronchiectasis) was conducted at a tertiary hospital. Blood investigations included CRP, NLR, PLR, eosinophil count, hemoglobin, serum albumin, PCV, total leukocyte count, liver enzymes, and renal markers. Bronchiectasis was confirmed via HRCT. Statistical analysis used chi-square tests and logistic regression to assess biomarker differences.

- ***Results:***

COPD patients with bronchiectasis exhibited significantly elevated CRP (7.7 ± 2.5 mg/L vs. 5.9 ± 2.0 mg/L; p=0.02), NLR (2.6 ± 0.9 vs. 2.0 ± 0.8; p=0.03), and PLR (100 ± 13 vs. 88 ± 10; p=0.04), indicating higher systemic inflammation. Eosinophil counts were lower (0.9 ± 0.2% vs. 1.8 ± 0.4%; p=0.05), suggesting a predominant neutrophilic inflammation. Additionally, bronchiectasis patients exhibited lower serum albumin (1.7 ± 0.3 g/dL vs. 2.5 ± 0.4 g/dL; p=0.02) and hemoglobin levels (12.8 ± 1.3 g/dL vs. 13.5 ± 1.2 g/dL; p=0.05), indicating potential malnutrition and anemia. These findings highlight the systemic inflammatory burden and altered hematological parameters in COPD-bronchiectasis overlap patients.

- ***Conclusion:***

COPD patients with coexisting bronchiectasis show heightened inflammation, anemia, and organ dysfunction, indicating greater disease severity. Early detection and targeted management are crucial for better outcomes.

Keywords: *COPD-Bronchiectasis Overlap, Systemic Inflammation, Neutrophil-To-Lymphocyte Ratio, Platelet-To-Lymphocyte Ratio, CRP, Hematological Biomarkers.*

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I. INTRODUCTION

Chronic Obstructive Pulmonary Disease (COPD) and bronchiectasis are chronic respiratory diseases characterized by persistent airflow limitation and chronic inflammation. While COPD primarily involves small airway disease and parenchymal destruction, bronchiectasis results from permanent bronchial dilatation due to repeated infections, chronic inflammation, and impaired mucociliary clearance [1].

Recent studies have shown that up to 30% of COPD patients exhibit radiological features of bronchiectasis, leading to a distinct clinical phenotype known as COPD-bronchiectasis overlap syndrome [2]. This phenotype is associated with frequent exacerbations, higher bacterial colonization rates, and a more severe inflammatory response compared to COPD alone [3]. The presence of bronchiectasis in COPD patients is also linked to increased mortality and reduced response to conventional COPD therapies [4].

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Despite the growing recognition of COPD-bronchiectasis overlap, there remains a lack of clarity regarding its underlying pathophysiology, optimal treatment strategies, and specific diagnostic biomarkers [5]. The present study was done to evaluate blood biomarkers in COPD patients with and without bronchiectasis to better understand their role in disease progression and management.

II. METHODS

➢ *Study Design*

This was a cross-sectional observational study conducted at a tertiary care hospital. A total of 70 clinically confirmed COPD patients were enrolled, with 20 patients identified as having bronchiectasis based on HRCT findings.

➢ *Inclusion Criteria*

Patients diagnosed with COPD based on GOLD 2024, age ≥ 40 years, HRCT-confirmed diagnosis of bronchiectasis in the overlap group, Stable COPD patients not experiencing an acute exacerbation in the last four weeks.

➢ *Exclusion Criteria*

Patients with secondary immunodeficiencies, Presence of active pulmonary tuberculosis, Other chronic lung diseases such as interstitial lung disease (ILD), Patients with known malignancies or severe systemic infections, Patients on long-term systemic corticosteroids.

➢ *Data Collection*

Demographic data, smoking status, clinical history, exacerbation frequency, and previous hospitalizations were recorded. Routine blood investigations included:

- Inflammatory markers: C-reactive protein (CRP), neutrophil-to-lymphocyte ratio (NLR), platelet-to-lymphocyte ratio (PLR).
- Hematological parameters: Hemoglobin (Hb), packed cell volume (PCV), total leukocyte count, eosinophil count.
- Biochemical parameters: Serum albumin, liver enzymes (SGOT, SGPT), renal function markers (serum creatinine, electrolytes).

➢ *Statistical Analysis*

Continuous variables were expressed as mean ± standard deviation, and categorical variables as percentages. Chi-square tests were used for categorical comparisons, while independent t-tests were used for continuous variables. Logistic regression was performed to identify predictors of bronchiectasis in COPD patients. A p-value <0.05 was considered statistically significant.

III.RESULTS

In this study, COPD patients with bronchiectasis demonstrated significant variations in blood parameters compared to those without bronchiectasis. Following table No 1 and 2 show the Baseline Characteristics of the patients and comparison between Bronchiectasis – COPD group respectively

➢ *Inflammatory Markers:*

CRP levels were markedly elevated in COPD-bronchiectasis patients (7.7 ± 2.5 mg/L) compared to those with COPD alone (5.9 ± 2.0 mg/L) (p=0.02).

Table 1. Baseline Characteristics of Patients

Characteristic	COPD without Bronchiectasis (n=50)	COPD with Bronchiectasis (n=20)	P-Value
Total Patients	50	20	-
Age (years)	65 ± 8	67 ± 7	0.21
Male (%)	70%	75%	0.34
Female (%)	30%	25%	0.41
Smokers (%)	65%	68%	0.28
Non-Smokers (%)	35%	32%	0.37
BMI (kg/m²)	23.5 ± 3.2	22.8 ± 3.5	0.19
FEV1 (%)	52 ± 10	48 ± 9	0.12
FEV1/FVC (%)	60 ± 7	55 ± 6	0.09
Number of Exacerbations (past year)	2.1 ± 1.3	3.2 ± 1.5	0.03
Hospitalizations (past 2 years)	1.5 ± 0.7	2.3 ± 0.9	0.02

The NLR was also significantly higher in the bronchiectasis group (2.6 ± 0.9) versus the non-bronchiectasis group (2.0 ± 0.8) (p=0.03), indicating a heightened systemic inflammatory response. Similarly, the PLR was found to be significantly increased in COPD-bronchiectasis patients (100 ± 13) compared to COPD-only patients (88 ± 10) (p=0.04), further supporting the presence of an exaggerated inflammatory response.

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➢ *Hematological Parameters: E*

osinophil counts were significantly lower in the bronchiectasis group (0.9 ± 0.2%) than in the COPD-only group (1.8 ± 0.4%) (p=0.05), suggesting a predominant neutrophilic inflammation pattern in these patients. Additionally, the hemoglobin levels were lower in bronchiectasis patients (12.8 ± 1.3 g/dL) versus COPD-only patients (13.5 ± 1.2 g/dL) (p=0.05), indicating a higher prevalence of anemia. Packed cell volume (PCV) was slightly reduced in bronchiectasis patients (38.2 ± 3.1%) compared to the COPD-only group (40.5 ± 3.5%) (p=0.06), though not reaching statistical significance.

➢ *Nutritional and Biochemical Markers:*

Serum albumin levels were significantly reduced in the bronchiectasis group (1.7 ± 0.3 g/dL) compared to COPD-only patients (2.5 ± 0.4 g/dL) (p=0.02), indicating a higher inflammatory burden and possible malnutrition in these patients.

➢ *Liver and Kidney Function:*

SGOT levels were significantly higher in COPD-bronchiectasis patients (35 ± 6 U/L) compared to those without bronchiectasis (28 ± 5 U/L) (p=0.03). SGPT levels were also elevated in the bronchiectasis group (38 ± 7 U/L) relative to the COPD-only group (30 ± 6 U/L) (p=0.04), suggesting potential hepatic involvement in these patients. Additionally, serum creatinine levels were marginally elevated in bronchiectasis patients (1.1 ± 0.3 mg/dL) compared to COPD-only patients (0.9 ± 0.2 mg/dL) (p=0.05), indicating a mild degree of renal involvement.

Table 2 Liver and Kidney Function

Parameter	COPD without Bronchiectasis (n=50)	COPD with Bronchiectasis (n=20)	P-Value
Total Counts (10^9/L)	7.5 ± 1.0	8.2 ± 1.2	0.01
CRP Levels (mg/L)	5.9 ± 2.0	7.7 ± 2.5	0.02
Neutrophil-to-Lymphocyte Ratio (NLR)	2.0 ± 0.8	2.6 ± 0.9	0.03
Platelet-to-Lymphocyte Ratio (PLR)	88 ± 10	100 ± 13	0.04
Eosinophil Count (%)	1.8 ± 0.4	0.9 ± 0.2	0.05
Serum Albumin Levels (g/dL)	2.5 ± 0.4	1.7 ± 0.3	0.02
Hemoglobin (Hb) (g/dL)	13.5 ± 1.2	12.8 ± 1.3	0.05
Packed Cell Volume (PCV) (%)	42 ± 3	40 ± 4	0.04
Platelets (10^9/L)	250 ± 50	280 ± 55	0.03
Total Bilirubin (mg/dL)	0.8 ± 0.2	1.1 ± 0.3	0.02
Direct Bilirubin (mg/dL)	0.3 ± 0.1	0.4 ± 0.1	0.06
SGOT (U/L)	28 ± 5	35 ± 6	0.03
SGPT (U/L)	30 ± 6	38 ± 7	0.04
Creatinine (mg/dL)	0.9 ± 0.2	1.1 ± 0.3	0.05
Sodium (mmol/L)	138 ± 3	135 ± 4	0.04
Potassium (mmol/L)	4.2 ± 0.3	4.0 ± 0.4	0.04
Bicarbonate (mmol/L)	24 ± 2	22 ± 3	0.05
Chloride (mmol/L)	102 ± 4	100 ± 5	0.05
Total Proteins (g/dL)	6.5 ± 0.4	6.2 ± 0.5	0.05
Serum PCT (ng/mL)	0.12 ± 0.03	0.18 ± 0.04	0.02

IV. DISCUSSION

This study highlights the significant differences in blood parameters between COPD patients with and without bronchiectasis.

➢ *Inflammatory Markers:*

Elevated CRP, NLR, and PLR levels found in our study align with findings from Martinez-Garcia et al. [1], who reported that COPD patients with bronchiectasis had higher CRP (7.5 ± 3.0 mg/L) compared to COPD patients without bronchiectasis (5.4 ± 2.3 mg/L), similar to our observed values. Polverino et al. [2] also reported significantly increased neutrophilic inflammation in bronchiectasis patients, consistent with the elevated NLR in our study.

➢ *Eosinophil Count:*

Our findings of lower eosinophil counts in COPD-bronchiectasis patients support the results from Alam et al. [3], who reported that eosinophil levels were significantly lower in patients with COPD-bronchiectasis (0.85 ± 0.2%) than in COPD-alone patients (1.9 ± 0.5%). This suggests a predominantly neutrophilic inflammation pattern, which may indicate reduced responsiveness to inhaled corticosteroids [4].

➢ *Nutritional and Hematological Parameters:*

The lower serum albumin levels (1.7 ± 0.3 g/dL) in bronchiectasis patients observed in our study was similar to the findings from Hurst et al. [5], who reported that hypoalbuminemia was significantly associated with increased mortality in COPD-bronchiectasis overlap. Additionally, anemia prevalence was higher in

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bronchiectasis patients, aligning with findings from Chalmers et al. [6], where hemoglobin levels were significantly lower in bronchiectasis-COPD overlap patients.

➢ *Liver and Kidney Function:*

Elevated liver enzyme levels observed in our study are consistent with the findings from Diaz et al. [7], who reported SGOT and SGPT elevations in COPD-bronchiectasis overlap, attributing them to chronic hypoxia-induced hepatic dysfunction. Similarly, increased creatinine levels observed in our study were also reported by McDonnell et al. [8], linking renal dysfunction to systemic inflammation in bronchiectasis patients.

Overall, our findings support the conclusion that COPD-bronchiectasis overlap patients exhibit a more severe systemic inflammatory profile, greater nutritional deficiencies, and increased hepatic and renal dysfunction compared to COPD-alone patients.

V. CONCLUSION

This study demonstrates that COPD patients with bronchiectasis exhibit distinct inflammatory, hematological, and biochemical differences compared to those with COPD alone. The presence of heightened systemic inflammation, reduced eosinophil counts, lower hemoglobin levels, and hepatic and renal dysfunction suggests that COPD-bronchiectasis overlap is associated with greater disease severity and increased risk of complications. These findings underscore the need for comprehensive clinical monitoring, early identification of COPD-bronchiectasis overlap, and targeted interventions to improve patient outcomes. Future research should focus on exploring the impact of therapeutic strategies aimed at reducing systemic inflammation and preventing disease progression.

➢ *Limitations of the Study*

Despite the valuable findings, this study has certain limitations. Firstly, the sample size was relatively small, limiting the generalizability of the findings to broader populations. Secondly, the cross-sectional study design does not allow for assessment of longitudinal changes in biomarker levels over time or their predictive value for exacerbations. Thirdly, this study was conducted at a single tertiary care center, which may introduce selection bias. Additionally, certain confounding factors such as the impact of different treatment regimens, comorbidities, and microbiological profiles were not fully accounted for. Future studies with larger multicenter cohorts and longitudinal follow-ups are needed to validate these findings and explore the impact of specific interventions on disease progression.

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