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Blood and sputum eosinophilia in chronic obstructive pulmonary disease patients and their association with exacerbations: a prospective observational study from a tertiary care center

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Abstract

Identification of the chronic obstructive pulmonary disease (COPD) phenotype allows selection of the most appropriate drug for each patient. Blood eosinophilia, as a surrogate marker for airway eosinophilia, has been associated with a phenotype of COPD exacerbators. Thus, blood eosinophilia and/or sputum eosinophilia enable healthcare providers to assess disease severity and guide treatment decisions in COPD patients. Understanding these factors can aid in effective COPD management and improve patient outcomes.

A prospective observational study was conducted at a tertiary care hospital. A total of 140 diagnosed COPD patients who met the eligibility criteria and attended the Department of Tuberculosis and Respiratory Diseases were included in the study. All recruited patients underwent a thorough clinical assessment, including detailed history taking (with emphasis on exacerbations), physical examination, complete blood count, including absolute eosinophil count (AEC), sputum cytology, and chest X-ray. Eosinophilia was defined as an AEC>150 cells/μL and/or sputum eosinophilia ≥2%. If sputum cytology showed <2% eosinophils and <60% neutrophils, the sample was considered paucicellular, and those patients were excluded from statistical analysis. Appropriate statistical tests were applied to derive inferences.

Of the 140 enrolled COPD patients, 83 (59.3%) had stable COPD and 57 (40.7%) were experiencing acute exacerbation. Blood eosinophilia (AEC>150 cells/μL) was present in 76 (54.3%) patients. After excluding sputum samples with paucicellularity (n=69), sputum eosinophilia (≥2%) was present in 41 (57.7%) of the remaining 71 patients. A statistically significant association was observed between blood eosinophilia and a history of exacerbations ($p<0.001$). Similarly, sputum eosinophilia was significantly associated with a history of COPD exacerbations ($p=0.008$). COPD severity (GOLD stage) was significantly associated with blood eosinophilia ($p=0.044$). However, no statistically significant correlation was found between sputum eosinophilia and blood eosinophilia ($p=0.5$).

Measurement of blood and sputum eosinophils facilitates phenotyping of COPD patients and enhances precision in treatment without delay. The use of inhaled corticosteroids targets eosinophilic inflammation in patients with COPD exacerbations. Therefore, we recommend measuring blood eosinophil counts in all patients at the time of COPD diagnosis.

Key words: COPD exacerbation, absolute eosinophil count, sputum eosinophilia, inhaled corticosteroids.

Introduction

Chronic obstructive pulmonary disease (COPD) is a heterogeneous respiratory disorder projected to affect approximately 600 million individuals globally by 2050 [1]. In 2016, an estimated 55.3 million cases of COPD were documented in India, where it represents the second most common cause of death among non-communicable diseases. COPD is a major cause of morbidity and mortality worldwide and is projected to become the third leading cause of death by 2030 [2]. The fundamental pathological feature of COPD is chronic airflow limitation. The complex interplay between inflammation and tissue destruction leads to structural abnormalities of the tracheobronchial tree and parenchymal hyperinflation [3].

Despite well-established evidence of heightened innate immune activation in COPD, airway eosinophilic inflammation is present in 20–40% of patients [4]. Eosinophil quantification from lung tissue, bronchoalveolar lavage, and induced sputum has demonstrated that sputum eosinophilia (>2%) and blood eosinophilia (>150 cells/μL) occur in both stable COPD and during acute exacerbations [5-7]. However, sputum eosinophil assessment is labour-intensive and frequently limited by inadequate sample quality [8].

Exacerbations are associated with an increased risk of recurrent events, accelerated lung function decline, and higher all-cause mortality, despite optimal bronchodilator therapy [9]. Patients with type 2 inflammatory signatures exhibit a favourable response to glucocorticoids, with reduced exacerbation risk. Accordingly, the Global Initiative for Chronic Obstructive Lung Disease (GOLD) report recommends using blood eosinophil counts to guide the addition of inhaled corticosteroids (ICS) to bronchodilator therapy [3].

Given the marked heterogeneity of COPD, phenotypic identification is critical to support individualised therapy and avoid uniform prescribing practices. Data from India regarding the prevalence of eosinophilia in COPD remains limited. Therefore, we aimed to determine the prevalence of blood and sputum eosinophilia and examine their association with exacerbations.

Materials and Methods

This prospective observational study was conducted at a tertiary care hospital. A total of 140 patients with a confirmed diagnosis of COPD who met the eligibility criteria and attended the Department of TB and Respiratory Diseases were enrolled. After obtaining written informed consent, patients aged 40–65 years, including both newly diagnosed individuals and those on follow-up, were recruited.

Patients with a history of asthma, hemoptysis, use of systemic (oral) corticosteroids within the preceding three months, or inability to perform spirometry were excluded. Patients presenting with acute exacerbation, as defined by the Global Initiative for Chronic Obstructive Lung Disease (GOLD) guidelines, were categorised as exacerbation cases. Those with no emergency department visits or hospital admissions in the preceding 12 months were classified as having stable COPD [3].

All enrolled patients underwent comprehensive clinical evaluation, including detailed history taking with documentation of prior exacerbations, physical examination, complete blood count including absolute eosinophil count (AEC), sputum cytology, and chest radiography. Tobacco exposure was quantified using the Smoking Index (SI) [10,11]. Dyspnea severity was assessed using the Modified Medical Research Council (mMRC) scale and the COPD Assessment Test (CAT). Pulmonary function was evaluated using standard spirometry in accordance with established guidelines [12].

Peripheral blood eosinophil count was measured from a 5 mL venous blood sample collected in an EDTA vacutainer and analysed using an automated haematology analyser. A single induced sputum sample was collected from each enrolled patient. An induced sputum sample was obtained following nebulization with 3% hypertonic saline (NaCl). The sample was homogenised with dithiothreitol (DTT), agitated, allowed to stand at room temperature for 30–60 minutes, and subsequently centrifuged. Smears were prepared and stained using haematoxylin–eosin and Giemsa stains. Differential cell counts were performed by examining 400 cells under light microscopy at 400× magnification.

Sputum samples demonstrating <2% eosinophils and <60% neutrophils were classified as paucicellular and excluded from statistical analysis. Blood eosinophilia was defined as an AEC >150 cells/μL, and sputum eosinophilia as ≥2%. The AEC cutoff of >150 cells/μL was selected based on previously demonstrated concordance with sputum eosinophilia ≥2% and its use in major clinical trials [13].

The study protocol was approved by the Institutional Research Committee (RC-3653) and the Institutional Ethics Committee (RC-5176).

Statistical analysis

The data obtained were analysed using SPSS (v.20). The Shapiro–Wilk test was used to assess the normality of data distribution. Odds ratios and their associated 95% confidence intervals (CIs) were calculated. Proportions were compared using the Chi-square test. Differences between the

means of two groups were compared using the independent samples *t*-test. Categorical variables were analysed using the Chi-square test or Fisher's exact test, as appropriate, for 2 × 2 tables. The Mann–Whitney rank-sum test was used for continuous variables that were not normally distributed, and Spearman's rank test was used to assess correlation. A P value < 0.05 was considered statistically significant.

Results

A total of 140 COPD patients provided blood and sputum samples during their hospital visit. The demographic characteristics of the study population are described in Table 1. The mean age of the patients was 58.34 ± 6.49 years. Of the 140 patients, 114 (81.4%) were males, and 26 (18.6%) were females. A majority of patients (n = 67, 47.9%) belonged to the lower socioeconomic class according to the Modified Kuppuswamy Scale [14]. The mean BMI was 19.14 ± 3.09 kg/m².

In our study, 115 (82.2%) patients were smokers. As these patients were bidi smokers, the Smoking Index (SI; number of bidis smoked per day × number of years smoked) was used to quantify smoke exposure [10,11]. The median SI was 400, with an interquartile range (IQR) of 240.0–800.0. A history of environmental smoke exposure was noted in 25 patients: 15 (10.7%) were exposed to biomass fuel, and 10 (7.14%) were exposed to occupational smoke.

At assessment, 83 (59.3%) patients were in a stable state, while 57 (40.7%) were experiencing an exacerbation. According to the GOLD 2025 classification, 75 (53.6%) patients were in stage E, 51 (36.4%) in stage B, and 14 (10%) in stage A [15]. A history of exacerbation was present in 133 (95%) patients. Among these, 60 (45.2%) had severe exacerbations, 33 (24.8%) had moderate exacerbations, and 40 (30%) had mild exacerbations.

Among the study population (n = 140), the most frequent background pharmacological regimen was long-acting muscarinic antagonist (LAMA) monotherapy (30.7%, n = 43), followed by long-acting beta-2 agonist plus LAMA (LABA + LAMA) therapy (25.7%, n = 36) and inhaled corticosteroid plus LABA (ICS + LABA) therapy (25.7%, n = 36). Triple therapy (ICS + LABA + LAMA) was used in 7.9% (n = 11) of patients.

The history of exacerbations showed a statistically significant association with pharmacological therapy in COPD patients (p = 0.01). Patients with prior exacerbations were more likely to receive ICS-containing and triple therapy regimens, while those without exacerbations were mainly treated with other therapies.

Blood eosinophilia

Of the 140 enrolled COPD patients, 76 (54.3%) had blood eosinophilia (absolute eosinophil count [AEC] >150 cells/μL). There were no statistically significant differences between patients with and without blood eosinophilia with respect to age, gender, duration of symptoms, CAT scores, or spirometry parameters (Table 2). A statistically significant association was observed between GOLD stage and absolute blood eosinophil count ($p = 0.04$).

The distribution of AEC >150 cells/μL differed significantly across pharmacological therapy groups ($p = 0.001$). The proportion of patients with AEC >150 cells/μL was lowest among those receiving LABA monotherapy (21.4%) and progressively higher in patients treated with LAMA (41.9%), ICS + LABA (52.8%), LABA + LAMA (75.0%), and ICS + LABA + LAMA (81.8%). Overall, combination and triple inhaler therapies were associated with a higher prevalence of elevated AEC compared with single-agent bronchodilator therapy.

There was no statistically significant association between current exacerbation status and blood eosinophilia ($p > 0.05$). However, a statistically significant association was observed between a history of exacerbations and blood eosinophilia ($p = 0.001$), as shown in Table 3.

Sputum eosinophilia

Among the enrolled patients, 69 (49.3%) had pauci-cellular sputum, while 71 (50.7%) samples met the criteria for adequate cellularity. After excluding pauci-cellular samples, 41 (57.7%) of the remaining 71 patients were found to have sputum eosinophilia (>2% eosinophils on sputum cytology). Pauci-cellular sputum samples were excluded from the statistical analysis.

There were no statistically significant differences between patients with and without sputum eosinophilia with respect to age, gender, duration of symptoms, CAT scores, or spirometry parameters (Table 2). No statistically significant association was observed between GOLD stage and sputum eosinophilia ($p = 0.41$).

There was also no statistically significant association between pharmacological therapy category and sputum eosinophilic status (≥2% vs <2%) in this cohort. Although sputum eosinophilia (≥2%) was numerically more frequent among patients receiving LABA + LAMA therapy (41.7%), the overall distribution of eosinophilic and non-eosinophilic phenotypes did not differ significantly across treatment groups.

No statistically significant association was found between current exacerbation status and sputum eosinophilia ($p > 0.05$). However, a statistically significant association was observed between a history of exacerbations and sputum eosinophilia ($p = 0.008$), as shown in Table 3.

Figure 1 depicts the correlation between blood and sputum eosinophilia using Spearman's correlation coefficient. A weak, non-significant positive correlation was observed between sputum and blood eosinophilia ($\rho = 0.072$, $p = 0.5$).

Discussion

The conceptual framework of COPD phenotyping dates back to the observations of Dornhorst in the 1950s, who described two distinct clinical presentations— "pink puffers" and "blue bloaters" [16]. Contemporary studies, including the ECLIPSE study, have further emphasised substantial heterogeneity in clinical presentation, quality of life, and exacerbation risk among patients with comparable degrees of airflow limitation [17]. Although eosinophils have traditionally been associated with bronchial asthma, accumulating evidence demonstrates their presence in a subset of COPD patients, suggesting a distinct inflammatory endotype [18,19].

In the present study, 76 (54.3%) patients exhibited blood eosinophilia (AEC >150 cells/μL), and 41 (57.7%) demonstrated sputum eosinophilia (≥2%). Published literature reports wide variability in the prevalence of eosinophilic COPD (18.8%–66.9%), with pooled estimates approximating 55% [19]. Differences across studies reflect heterogeneity in design, population characteristics, disease status (stable versus exacerbation), and eosinophil thresholds. Nevertheless, an AEC threshold >150 cells/μL remains a clinically meaningful and evidence-based cutoff that balances sensitivity and specificity for predicting corticosteroid responsiveness and guiding therapy. A threshold of ≥150 identifies patients more likely to have eosinophil-driven exacerbations, better steroid responsiveness and less bacterial colonisation [13].

We did not observe significant differences between eosinophilic and non-eosinophilic groups with respect to age, sex, symptom duration, CAT score, or spirometric parameters. Furthermore, the current disease state (stable versus exacerbation) was not significantly associated with blood or sputum eosinophilia. However, a history of prior exacerbations demonstrated a significant association with both blood eosinophilia ($p=0.001$) and sputum eosinophilia ($p=0.008$). These findings align with observations from the ECLIPSE study, where eosinophil levels were not associated with baseline disease characteristics [20].

Clinically, the association between elevated blood eosinophil counts and a history of exacerbation suggests that AEC may serve as a biomarker to identify patients at increased risk of severe exacerbations [21]. Importantly, eosinophilia may represent a dynamic inflammatory trait rather than a fixed phenotype. Stable eosinophilia likely reflects a persistent biological endotype, whereas eosinophilia during exacerbation may represent an acute inflammatory response

influenced by heterogeneous triggers [8,21,22]. Variability in sampling timing, cutoff definitions, and cohort enrichment explains the broad range of reported prevalence and limits direct inter-study comparisons.

Consistent with prior reports, we did not observe a significant correlation between sputum and blood eosinophil counts [22,23]. Although peripheral blood eosinophils are considered surrogate markers of airway eosinophilia and are recommended for therapeutic guidance by the Global Initiative for Chronic Obstructive Lung Disease (GOLD) strategy, sputum eosinophil measurement may be influenced by sample quality and local airway factors [15].

The combination and triple inhaler therapies were associated with a higher prevalence of elevated AEC compared with single-agent bronchodilator therapy, and the patients on triple therapy experienced fewer exacerbations in the past. This again highlighted the importance of endotyping COPD based on AEC and choosing personalised therapy for the patients [24-26].

Blood and sputum eosinophil measurements contribute to the identification of an eosinophilic COPD endotype associated with differential response to inhaled corticosteroids (ICS). Higher eosinophil counts have been associated with greater benefit from ICS in reducing exacerbation frequency, supporting biomarker-driven treatment strategies. However, in regions with high parasitic prevalence, such as India, eosinophilia should be interpreted cautiously, as parasitic infections may confound peripheral eosinophil levels.

This study has a few limitations. First, only a single sputum sample was analysed, and a substantial proportion of samples were excluded due to paucicellularity. Second, there was limited representation of patients receiving triple therapy (ICS + LABA + LAMA). Third, routine evaluation for parasitic infections and allergic bronchopulmonary aspergillosis (ABPA) was not performed in patients with eosinophilia.

Conclusions

COPD is a heterogeneous obstructive airway disease characterized by complex and incompletely understood inflammatory mechanisms. This heterogeneity contributes to variability in clinical presentation, disease progression, and therapeutic response. Stratifying patients into biologically relevant subgroups represents an essential step toward precision medicine in COPD.

Blood eosinophil count is a practical and clinically applicable biomarker for phenotypic stratification and risk assessment. Routine measurement of blood eosinophil counts in patients with COPD may facilitate individualized therapeutic decision-making and optimize the use of inhaled corticosteroids.

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Table 1. Demographic profile of the COPD patients enrolled

Variables	
Age (years), mean (S.D.)	58.34 (6.5)
Smoking Index*, median (IQR)	400.0 (240-800)
CAT Score, mean (S.D.)	18.40 (6.2)
BMI (kg/m^2), mean (S.D.)	19.14 (3.09)
Absolute eosinophil count, median (IQR)	170.0 (100 – 350)
Sputum eosinophil count (%), median (IQR)	1 (1-3)
FEV 1 % predicted, mean (S.D.)	44.11 (15.3)
FEV1 /FVC, mean (S.D.)	50.30 (9.5)

*Median smoking index was calculated after excluding 25 non-smoker COPD patients.

Table 2. Distribution of variables based on blood eosinophil count and sputum eosinophil.

Variables	Absolute eosinophil count, n=140		p	Sputum eosinophil (pauci-cellular smears excluded), n=71		p
	≤150 cells/mm^3 (n=64)	>150 cells/mm^3 (n=76)		<2% (n=30)	≥2% (n=41)	
Age (years), mean (S.D.)	58.14(6.39)	58.51(6.61)	0.737	59.26(6.58)	56.90(7.54)	0.173
CAT Score, mean (S.D.)	18.87(5.73)	18.00(6.65)	0.411	19.20(5.80)	19.14(7.13)	0.973
Duration of COPD (years), median (IQR)	5.0(3.0-10.0)	6.0(3.0-10.0)	0.611	5.0(2.0-10.0)	6.0(3.0-10.0)	0.521
BMI (kg/m^2), mean (S.D.)	19.17(3.30)	19.12(2.94)	0.932	19.22(3.17)	19.24(2.98)	0.981
Hb (gm%), mean (S.D.)	13.39(1.93)	13.90(1.48)	0.077	13.66(1.75)	13.65(1.64)	0.980
FEV1 (litres), mean (S.D.)	1.02(0.34)	1.15(0.47)	0.060	1.12(0.39)	1.03(0.42)	0.380
FVC (litres), mean (S.D.)	2.09(0.55)	2.25(0.67)	0.117	2.16(0.55)	2.11(0.69)	0.731
FEV1/FVC, mean (S.D.)	49.30(8.91)	51.14(9.95)	0.255	52.03(8.45)	49.24(9.51)	0.206

Table 3. Association between sputum/blood eosinophils and COPD exacerbation.

Parameter	Type of exacerbation history			Chi-squared p value
	Mild	Moderate	Severe	
Sputum eosinophil <2%	9(60.0)	9(69.2)	11(27.5)	9.355,0.008
Sputum eosinophil ≥2%	6(40.0)	4(30.8)	29(72.5)	
Absolute eosinophil (AEC) ≤150 cells/µl	14(35.0)	24(72.7)	11(27.5)	13.544, 0.001
Absolute eosinophil (AEC) >150 cells/µl	26(65.0)	9(27.3)	38(63.3)	

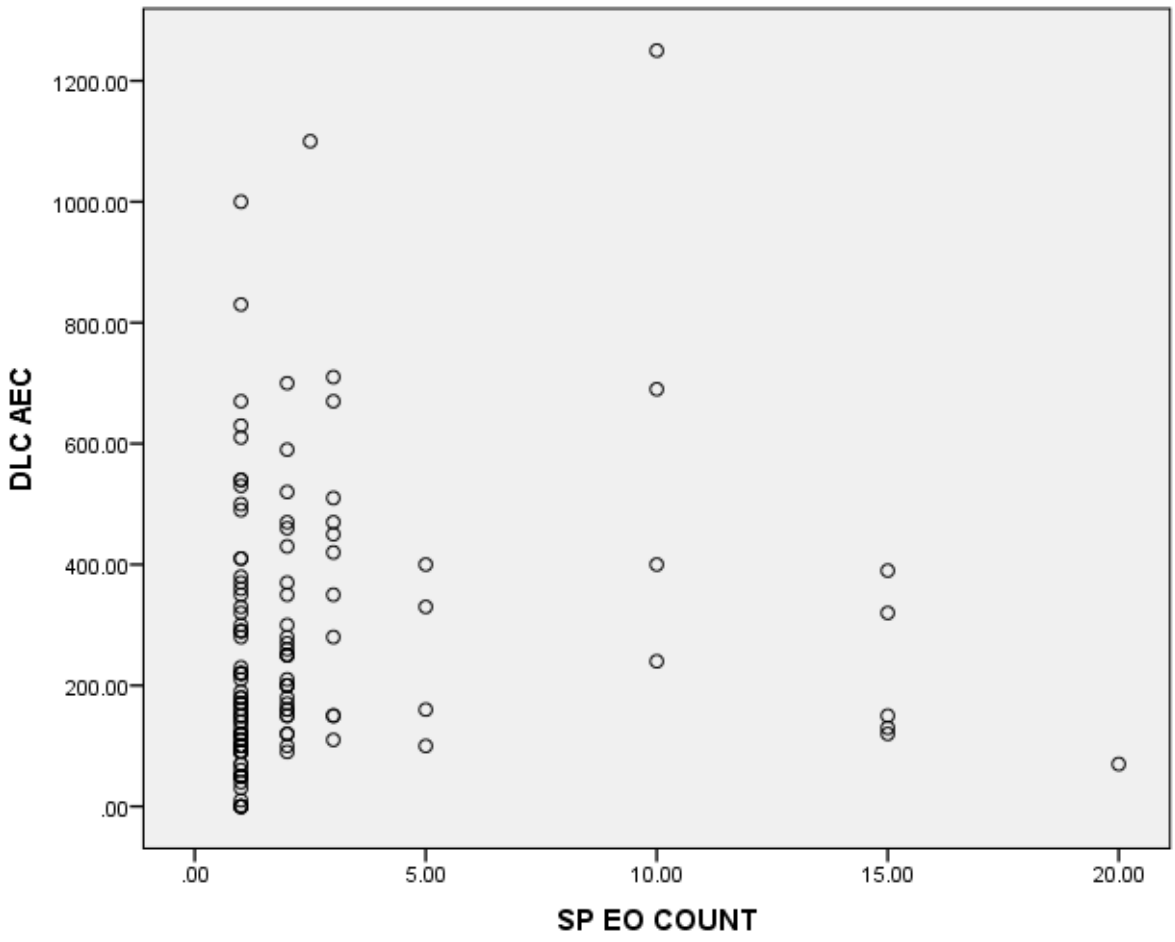

Figure 1. Spearman's Correlation coefficient between Sputum eosinophil count and Absolute eosinophil count.