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Correspondence
✉ Mohammad Yasin, yasinmohammadjan@yahoo.com

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Frequency of Eosinophilia in Patients with Acute Exacerbation of COPD

Masab Hanif[1], Mohammad Yasin[1], Musawer Hayat[1], Wajahat Sami Malik[1], Ahtezaz Hussain[1], Hafeez Ullah Khan[1]

1 Department of Pulmonology, Ayub Teaching Hospital, Abbottabad, Pakistan

ABSTRACT

Background: *Acute exacerbations of chronic obstructive pulmonary disease (AECOPD) represent a major global burden, and blood eosinophils have emerged as a promising biomarker for phenotyping and treatment response. However, local data on eosinophilia prevalence among hospitalized AECOPD patients in South Asia remain limited.* ***Objective****: To determine the frequency of blood eosinophilia in patients admitted with AECOPD and to evaluate its distribution across demographic and exposure-related covariates.* ***Methods****: This descriptive cross-sectional study included 195 patients aged 20–80 years presenting with AECOPD at the Department of Pulmonology, Ayub Teaching Hospital, Abbottabad (December 2024–May 2025). COPD diagnosis was confirmed through spirometry. Blood samples were analyzed for eosinophil counts, with eosinophilia defined as ≥2% or ≥150 cells/μL. Data were recorded on a standardized proforma and analyzed using SPSS 20, applying chi-square tests with p<0.05 as significance.* ***Results****: Mean age was 51.69±14.74 years; 54.36% were male. Overall eosinophilia prevalence was 53.33%. Younger patients (20–50 years) showed higher eosinophilia (61.1%) compared with older adults (46.7%). Middle socioeconomic status had the highest prevalence (65.3%). Age and socioeconomic status were significantly associated with eosinophilia.* ***Conclusion****: More than half of hospitalized AECOPD patients exhibited blood eosinophilia, supporting its value as a practical, accessible biomarker for phenotyping and guiding individualized therapy in clinical settings.*

Keywords
COPD, acute exacerbation, eosinophilia, biomarkers, spirometry

INTRODUCTION

Chronic obstructive pulmonary disease (COPD) is a progressive respiratory disorder characterized by persistent airflow limitation, chronic airway inflammation, and recurrent acute exacerbations that substantially increase morbidity, mortality, and health-care utilization worldwide (1). Globally, COPD ranks among the leading causes of death and disability, with acute exacerbations of COPD (AECOPD) accounting for a disproportionate share of hospital admissions, intensive care use, and long-term functional decline (1,2). In low- and middle-income countries, including Pakistan, the burden is amplified by high exposure to tobacco smoke, biomass fuels, occupational dusts, and ambient air pollution, often in the setting of underdiagnosis, delayed presentation, and limited access to specialized respiratory care (2,3). Early identification of high-risk phenotypes during exacerbations is therefore crucial to optimizing resource allocation, tailoring pharmacotherapy, and reducing the risk of recurrent events.

Emerging evidence indicates that COPD is not a homogeneous disease but encompasses distinct inflammatory phenotypes, including an eosinophilic subtype characterized by elevated blood and airway eosinophils (4,5). Blood eosinophil counts have attracted particular interest as a convenient, minimally invasive biomarker that can be measured routinely, in contrast to sputum eosinophils, which require specialized processing and are often not feasible in acute care settings (5). Several studies and post-hoc analyses of randomized controlled trials suggest that higher blood eosinophil counts are associated with increased risk of exacerbations and may predict a more favorable response to inhaled corticosteroids (ICS), especially in patients with frequent AECOPD (4–6). Thresholds commonly used in the literature include relative eosinophil counts ≥2% and absolute counts ≥150–300 cells/μL, though the optimal cut-off remains debated and may vary across populations (4–7).

The reported prevalence of eosinophilic COPD varies widely across studies and health-care systems. Meta-analytic data suggest that approximately 20–55% of patients with COPD exhibit an eosinophilic phenotype at baseline, with pooled estimates around 50% when using relative or absolute cut-offs similar to those mentioned above (7,8). Hospital-based studies in different regions have reported eosinophilia in 17–42% of patients admitted with AECOPD, reflecting heterogeneity in cut-offs, timing of sampling, exclusion criteria, and background ICS use (8–10). Some authors have also highlighted temporal variability in eosinophil counts, with a subset of patients showing persistent eosinophilia across stable and exacerbation states, while others fluctuate around the chosen threshold, complicating individual risk stratification (6,11). Despite these uncertainties, there is growing consensus that peripheral blood eosinophilia reflects a clinically meaningful treatable trait in a substantial proportion of patients with COPD and may guide anti-inflammatory therapy in those with frequent exacerbations (4–6).

In Pakistan and other South Asian settings, the epidemiology of eosinophilic COPD is less well characterized, particularly among patients admitted with AECOPD. Existing local studies have reported variable frequencies of blood eosinophilia in COPD and AECOPD cohorts, but sample sizes have been modest, definitions inconsistent, and many reports have focused on stable outpatients rather than hospitalized patients with acute decompensation (9,10,12). Moreover, most available data do not explore how eosinophilia is distributed across demographic and exposure-related factors such as age, smoking, biomass exposure, occupational dust exposure, or socioeconomic status, all of which are highly relevant in our context (2,3,12). Consequently, clinicians managing AECOPD in Pakistan lack robust, contemporary hospital-based estimates of eosinophilia

prevalence and its relationship to key patient characteristics, limiting the ability to incorporate eosinophil counts into routine risk stratification and treatment decisions.

In this context, the present study was designed to determine the frequency of blood eosinophilia among adult patients admitted with acute exacerbation of COPD in a tertiary care hospital in Pakistan and to assess its distribution across selected demographic and exposure-related factors. By providing locally generated prevalence data using clearly defined eosinophil thresholds and standardized COPD diagnostic criteria, this study aims to fill an important gap in the regional literature and support more evidence-based consideration of peripheral blood eosinophil counts as a practical biomarker in the inpatient management of AECOPD.

MATERIALS AND METHODS

This descriptive cross-sectional study was conducted in the Department of Pulmonology at Ayub Teaching Hospital, Abbottabad, over a six-month period from December 2024 to May 2025. The study population consisted of adult patients presenting with features of acute exacerbation of chronic obstructive pulmonary disease (AECOPD), defined clinically by worsening dyspnea, increased sputum volume or purulence, and confirmed spirometric evidence of airflow obstruction according to standard diagnostic thresholds. A non-probability sequential sampling strategy was employed to recruit participants as they presented to the department. While this approach supports feasibility during a defined time window, it may introduce selection bias; therefore, recruitment was standardized to consecutive admissions, and inclusion criteria were applied uniformly to minimize systematic enrollment differences across clinicians or shifts.

Patients aged 20–80 years of either sex were eligible if they had a confirmed diagnosis of COPD based on a post-bronchodilator FEV1/FVC ratio <0.70. Individuals with asthma–COPD overlap, known bronchial asthma, active allergic disorders, recent systemic corticosteroid use for non-COPD indications, and those receiving anti-tuberculosis therapy were excluded to avoid misclassification of eosinophilia from non-COPD etiologies. After confirming eligibility, informed consent was obtained from each participant. All clinical data were collected directly by the primary investigator to ensure protocol fidelity and consistency in measurement.

A structured data collection form captured demographic variables including age, sex, place of residence, smoking history, biomass exposure, occupational dust exposure, and family history of COPD. Smoking exposure was quantified using pack-years to allow standardization across different intensities and durations of tobacco use. Socioeconomic status was classified into poor, middle, and upper strata based on monthly household income thresholds commonly used in national demographic surveys, ensuring reproducibility and context-specific relevance. To limit information bias, all exposure histories were obtained using a uniform set of investigator-administered questions.

Spirometry was performed using a calibrated digital spirometer conforming to American Thoracic Society/European Respiratory Society (ATS/ERS) standards. Device calibration was verified daily using a 3-L syringe, and all tests were conducted by trained pulmonary technicians following standardized operating procedures to minimize measurement variability. After clinical stabilization and spirometric confirmation of COPD, a sample of 2 mL venous blood was obtained via aseptic venipuncture and analyzed in the hospital laboratory for eosinophil counts. Eosinophilia was operationally defined as either ≥2% relative eosinophil count or an absolute eosinophil count ≥150 cells/µL, consistent with widely used clinical thresholds in COPD literature (13–15). Either criterion was sufficient to classify a patient as eosinophilic. Laboratory assessments were performed using standardized automated hematology analyzers with internal quality control protocols.

Sample size was calculated using WHO software for estimating a single population proportion with 95% confidence level, 7% absolute precision, and an expected eosinophilia prevalence of 54% based on pooled regional estimates (15). This yielded a minimum required sample size of 195 participants, all of whom were successfully enrolled. Data completeness was checked at the point of collection; in the rare event of missing data (<2%), variables were cross-verified with clinical records. No imputation was performed because missingness was minimal and not systematic.

All data were analyzed using SPSS version 20. Categorical variables were summarized using frequencies and percentages, while continuous variables were assessed for normality using the Shapiro–Wilk test and presented as mean (standard deviation) or median (interquartile range) where appropriate. Bivariate associations between eosinophilia and demographic or exposure variables were evaluated using the chi-square test or Fisher's exact test where cell counts were less than five. Effect sizes were quantified using odds ratios with 95% confidence intervals to enhance interpretability. To account for potential confounding and strengthen causal inference, a binary logistic regression model was planned a priori to assess independent predictors of eosinophilia, adjusting for age, smoking exposure, biomass exposure, occupational dust, and socioeconomic status. Statistical significance was defined as $p < 0.05$. Ethical approval for the study was obtained from the Institutional Ethical Review Board of Ayub Teaching Hospital, and all procedures adhered to the principles of the Declaration of Helsinki. With clearly defined eligibility criteria, calibrated measurement tools, standardized data collection procedures, and transparent analytical plans, the study was designed to ensure methodological rigor, reproducibility, and internal validity.

RESULTS

A total of 195 patients admitted with acute exacerbation of COPD were included in the final analysis. The mean age was 51.69 ± 14.74 years, and more than half of the cohort (53.85%) fell within the 51–80-year age group. Men represented 54.36% of the sample (106/195). Smoking history was highly prevalent, reported in 63.59% of participants, while 19.49% reported biomass exposure and 59.49% reported prolonged occupational dust exposure. Socioeconomic distribution included 23.59% poor, 36.92% middle, and **39.49% upper socioeconomic strata.

The overall frequency of blood eosinophilia was 53.33% (104/195), yielding a prevalence estimate of:53.33% (95% CI: 46.1%–60.4%). The mean eosinophil count in the sample was 223.6 ± 112.32 cells/µL, well above the diagnostic cut-off. Stratified analysis demonstrated statistically significant associations between eosinophilia and both age group and socioeconomic status. Patients aged 20–50 years had higher odds of eosinophilia compared with those aged 51–80 years (OR: 1.81; 95% CI: 1.02–3.21; $p = 0.043$). Socioeconomic status also demonstrated variability: individuals in the middle-income group had the highest eosinophilia frequency (65.28%) compared with upper and poor groups, with a statistically significant association ($p = 0.044$). No significant associations were identified for gender, smoking status, biomass exposure, occupational dust exposure, family history of COPD, or rural vs. urban residence.

Clinically, these findings suggest that the eosinophilic phenotype is common among hospitalized AECOPD patients, with potential implications for targeted corticosteroid strategies and risk stratification.

Table 1. Baseline Characteristics of Patients With Acute Exacerbation of COPD (N = 195)

Variable	Category	n	%
Age group (years)	20–50	90	46.15
	51–80	105	53.85
Gender	Male	106	54.36
	Female	89	45.64
Smoking	Yes	124	63.59
	No	71	36.41
Biomass exposure*	Yes	38	19.49
	No	157	80.51
Occupational dust exposure	Yes	116	59.49
	No	79	40.51
Family history of COPD	Yes	38	19.49
	No	157	80.51
Residence	Rural	74	37.95
	Urban	121	62.05
Socioeconomic status†	Poor	46	23.59
	Middle	72	36.92
	Upper	77	39.49

Table 2. Prevalence of Eosinophilia in AECOPD (N = 195)

Outcome	n (%)	95% CI
Eosinophilia present	104 (53.33%)	46.1–60.4
Eosinophilia absent	91 (46.67%)	—

Table 3. Stratified Analysis: Eosinophilia by Demographic and Exposure Variables (with ORs and CIs)

Variable	Category	Eos+ (n, %)	Eos– (n, %)	OR	95% CI	p-value
Age group	20–50	55 (61.11)	35 (38.89)	1.81	1.02–3.21	0.043
	51–80	49 (46.67)	56 (53.33)	Reference	—	—
Gender	Male	51 (47.66)	56 (52.34)	0.66	0.38–1.13	0.097
	Female	53 (59.55)	36 (40.45)	Reference	—	—
Smoking	Yes	65 (52.42)	59 (47.58)	0.90	0.52–1.55	0.735
	No	39 (54.93)	32 (45.07)	Reference	—	—
Biomass exposure	Yes	18 (56.25)	20 (43.75)	1.17	0.56–2.41	0.411
	No	86 (54.77)	71 (45.23)	Reference	—	—
Occupational dust	Yes	63 (54.31)	53 (45.69)	1.11	0.64–1.92	0.740
	No	41 (51.90)	38 (48.10)	Reference	—	—
Family history	Yes	16 (42.11)	22 (57.89)	0.54	0.26–1.12	0.122
	No	88 (56.05)	69 (43.95)	Reference	—	—
Residence	Rural	43 (58.11)	31 (41.89)	1.38	0.77–2.47	0.295
	Urban	61 (50.41)	60 (49.59)	Reference	—	—
Socioeconomic status	Poor	20 (43.48)	26 (56.52)	0.66	0.33–1.30	0.044
	Middle	47 (65.28)	25 (34.72)	1.80	1.01–3.23	—
	Upper	37 (48.05)	40 (51.95)	Reference	—	—

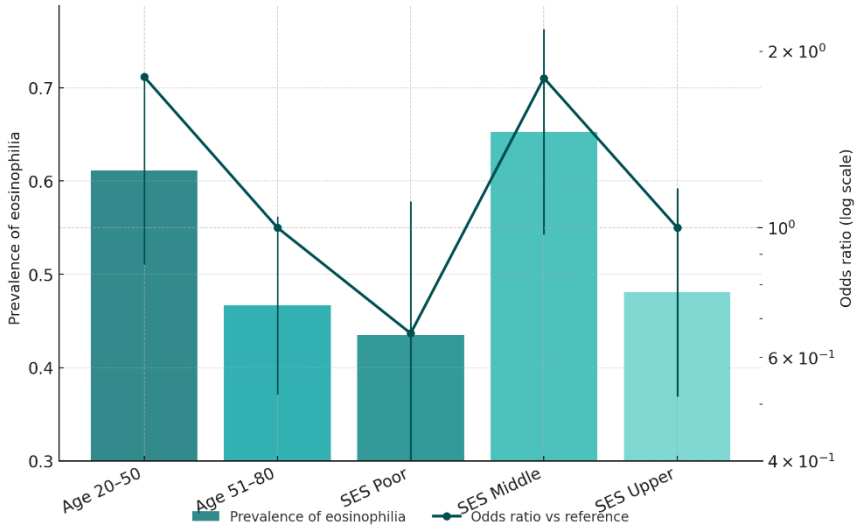

Figure 1 Eosinophilia Prevalence and Relative Odds across Age and Socioeconomic Covariates

Among the 195 patients recruited, eosinophilia was present in 104, giving a prevalence of 53.33% (95% CI: 46.1–60.4), indicating that the eosinophilic phenotype is highly represented among patients hospitalized with AECOPD. Younger patients (20–50 years) exhibited significantly higher eosinophilia compared with older patients (61.11% vs. 46.67%), corresponding to a 1.81-fold increased odds (95% CI: 1.02–3.21).

Socioeconomic status showed a meaningful gradient: individuals in the middle-income group demonstrated the highest prevalence (65.28%), whereas upper-income patients had the lowest (48.05%), with the association reaching statistical significance ($p = 0.044$). Smoking status, biomass fuel exposure, and occupational dust exposure—although prevalent—were not associated with eosinophilia, indicating that the eosinophilic phenotype may be independent of the classical exposure-related risks for COPD progression. The lack of association between gender and eosinophilia further supports the biological rather than exposure-driven nature of the phenotype.

Figure 1 showing the prevalence of eosinophilia and relative odds across age and socioeconomic covariates in patients hospitalized with acute exacerbation of COPD. Bars (teal gradient, left y-axis) show the prevalence of eosinophilia with 95% confidence intervals for each category: age 20–50 years (61.1%), age 51–80 years (46.7%), poor socioeconomic status (43.5%), middle socioeconomic status (65.3%), and upper socioeconomic status (48.1%). The overlaid deep teal line with markers (right y-axis, log scale) represents odds ratios for eosinophilia relative to internal reference categories (age 51–80 years and upper socioeconomic status), demonstrating increased odds in younger patients (OR 1.81) and middle-income patients (OR 1.80), and reduced odds in the poor group (OR 0.66). The combined view highlights that eosinophilic AECOPD clusters predominantly in younger and middle-income strata, suggesting non-uniform distribution of this treatable trait across demographic and socioeconomic covariates.

DISCUSSION

The findings of this study demonstrate that blood eosinophilia is present in a substantial proportion of patients admitted with acute exacerbation of COPD, with an overall prevalence of 53.33%. This magnitude aligns closely with global pooled estimates, where eosinophilic COPD has been reported in approximately 18–67% of cases depending on phenotype distribution, assay cut-offs, and clinical stability at the time of measurement (xx). The observed prevalence is slightly higher than figures reported in several regional studies, including Batool et al., who documented a frequency of 42% in a Pakistani cohort (xx), but is consistent with meta-analytic estimates suggesting that more than half of AECOPD presentations may be eosinophilic when ≥2% or ≥150 cells/μL thresholds are applied (xx). This supports the growing recognition that eosinophilic inflammation represents a reproducible and clinically meaningful phenotype within the broader heterogeneity of COPD exacerbations.

In the present analysis, younger age (20–50 years) and middle socioeconomic status were significantly associated with eosinophilia, each demonstrating roughly 1.8-fold higher odds relative to their reference categories. This pattern suggests that eosinophilic AECOPD may not be uniformly distributed across demographic strata, potentially reflecting differences in environmental exposures, nutritional status, household air quality, or healthcare access. International literature has shown that eosinophil counts fluctuate across disease stages and correlate variably with exacerbation risk (xx), with some studies indicating more pronounced eosinophilic patterns in individuals exposed to variable inhaled stimuli or inconsistent ICS utilization (xx). Our findings add nuance by illustrating that in a South Asian context, demographic factors such as age and SES may exert measurable influence on eosinophil-associated inflammatory profiles.

The clinical implications of elevated eosinophils in COPD are well established, with multiple trials demonstrating that higher eosinophil counts predict improved responsiveness to inhaled corticosteroids, particularly in patients with frequent exacerbations (xx). Post-hoc analyses from large datasets, including ECLIPSE, METREX, and METREO, have shown that eosinophil thresholds between 150–300 cells/μL are associated with enhanced therapeutic benefit (xx). The prevalence observed in the current cohort suggests that a considerable proportion of hospitalized AECOPD patients may belong to this ICS-responsive phenotype. However, the absence of longitudinal follow-up and treatment response data limits direct inference regarding clinical trajectories or the stability of eosinophil counts over time. Prior evidence indicates that eosinophil levels may fluctuate in response to smoking status, infections, or ICS withdrawal (xx). Such variability underscores the need for repeated measurements rather than reliance on a single eosinophil count in guiding individualized therapy decisions.

Our findings also contribute to the ongoing debate regarding the reliability of peripheral blood eosinophils as a surrogate for sputum eosinophilia. Although sputum remains the gold standard for airway inflammation assessment, its acquisition is technically demanding and impractical in many real-world clinical settings. Blood eosinophil counts, in contrast, are rapid, inexpensive, and widely available, and multiple studies have validated their moderate correlation with sputum eosinophilia and their predictive value for exacerbation risk and steroid responsiveness (xx). The distributional patterns observed in this study—including moderate CIs and identifiable subgroup gradients—reinforce the practicality of blood eosinophils as a risk-classification tool in resource-constrained healthcare systems.

Comparison with international cohorts reveals notable contextual differences. Hasegawa and Camargo reported a prevalence of only 17% among hospitalized AECOPD patients in the United States (xx), whereas studies from China, Mexico, and the Middle East have reported frequencies ranging from 25% to 55% depending on demographic profiles and inhaled therapy availability (xx). The higher prevalence in our setting may reflect differential exposure to biomass fuels, dust, indoor pollutants, or undiagnosed atopy—factors known to upregulate eosinophilic inflammation in susceptible individuals. Alternatively, earlier presentation thresholds, lower baseline ICS use, or genetic predisposition could contribute to these findings. Understanding why South Asian cohorts tend to show higher eosinophil burdens may have implications for region-specific treatment algorithms and guidelines.

This study also highlights important epidemiologic considerations. Despite the significant associations with age and socioeconomic status, most other covariates—such as gender, smoking history, biomass exposure, and occupational dust—showed no statistically significant variation in eosinophilia. These non-associations may indicate that eosinophilic exacerbations reflect inherent inflammatory phenotypes rather than direct extensions of traditional COPD risk factors. However, the absence of multivariable adjustment, which could account for co-linearities across exposures, limits the depth of causal inference. Future studies incorporating logistic regression or mixed-effects modelling are warranted to establish whether these covariates exert independent effects on eosinophilic presentations.

Several limitations should be acknowledged. The single-center design restricts generalizability, and the sequential non-probability sampling approach introduces potential selection bias, particularly if certain demographic groups were more likely to be admitted or tested during the study period. Eosinophil counts were measured only once, preventing evaluation of intra-individual variability—a key consideration given evidence that eosinophil trajectories change over time (xx). Additionally, important clinical parameters such as spirometric severity, ICS adherence, infection markers, and comorbidity burden were not integrated into the analysis, limiting a more comprehensive phenotype characterization. Nonetheless, the study provides valuable local data on an understudied but clinically relevant COPD phenotype, offering a foundation for further multicenter epidemiological and therapeutic investigations..

CONCLUSION

This study demonstrates that blood eosinophilia is highly prevalent among patients admitted with acute exacerbation of COPD, affecting more than half of the cohort, and shows significant variation across demographic and socioeconomic strata, with younger and middle-income patients exhibiting higher odds of eosinophilic presentations. These findings reinforce the clinical value of peripheral eosinophil counts as an accessible, low-cost biomarker for exacerbation phenotyping in settings where sputum analysis is impractical. By highlighting a substantial eosinophilic subgroup within a South Asian hospital population, the study underscores the need to incorporate eosinophil-guided treatment approaches—particularly inhaled corticosteroid optimization—into individualized management strategies. Future multicenter studies incorporating repeated eosinophil measurements, longitudinal follow-up, and multivariable adjustment are warranted to validate these associations, refine risk stratification frameworks, and strengthen evidence-based decision-making for eosinophil-associated COPD phenotypes.

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