Original Article

Prognostic Significance of Absolute Neutrophil and Eosinophil Counts in AECOPD in Kolonel Abundjani Bangko General Hospital: A Retrospective Study

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Submitted: March 6th, 2026
Accepted: May 13th, 2026
Published: June 30th, 2026

Respir Sci. 2026; 6(3): 152-60
https://doi.org/10.36497/respirsci.v6i3.230

Abstract

Background: Chronic obstructive pulmonary disease (COPD) is a chronic and progressive respiratory condition characterized by persistent airflow obstruction. The disease trajectory is often punctuated by episodes of acute exacerbation, which significantly aggravate clinical status and increase healthcare burden. The biological plausibility and differential inflammatory pathways of these cell populations suggest evaluating the relationship between neutrophil and eosinophil profiles and the one-year exacerbation frequency in patients with acute COPD.

Method: This study was conducted using a retrospective observational analytic design and included all patients with a confirmed diagnosis of acute exacerbation of chronic obstructive pulmonary disease (AECOPD) who fulfilled the established inclusion criteria at Kolonel Abundjani Bangko General Hospital during April to December 2024. Clinical and laboratory data were retrieved from hospital medical records and subsequently analyzed using appropriate statistical methods.

Results: Thirty-eight patients fulfilled the predefined inclusion criteria and were subsequently enrolled in the analysis. The respondents were predominantly male (94.7%), older than 55 years (89.5%), with a documented smoking history (92.1%), with relatively short admissions (1–4 days) in 68.4%, and survival at discharge was observed in 94.7% of patients. Inferential statistical testing demonstrated no significant relationship between neutrophil or eosinophil counts and clinical prognosis in patients experiencing AECOPD (P>0.05).

Conclusion: Neutrophil nor eosinophil counts demonstrated a statistically significant association with prognostic indicators among patients admitted with acute COPD. However, elevated neutrophil levels (>8) were consistently more prevalent across prognostic strata compared with increased eosinophil counts. This distribution pattern suggests a relative predominance of neutrophil-mediated inflammatory activity during acute exacerbations.

Keywords: absolute neutrophil and eosinophil counts, AECOPD

INTRODUCTION

Chronic obstructive pulmonary disease (COPD) remains one of the most significant noncommunicable diseases contributing to global morbidity and mortality. According to the World Health Organization (WHO) in its Global Status Report on Noncommunicable Diseases, COPD is classified among the four leading noncommunicable diseases that collectively account for approximately 74% of worldwide deaths, together with cardiovascular diseases, stroke, cancer, and diabetes mellitus.[1]

Pathophysiologically, COPD is characterized by persistent and largely irreversible airflow limitation resulting from chronic inflammatory processes within the airways and lung parenchyma. Acute exacerbations, defined as sustained worsening of respiratory symptoms necessitating additional therapeutic intervention, substantially contribute to disease progression, impaired health-related quality of life, repeated hospital admissions, and increased mortality risk.[2]

Survival outcomes in patients experiencing acute exacerbations are determined by a multifactorial interplay that includes exacerbation severity, recurrence rate, advancing age, nutritional status, baseline pulmonary function, and coexisting comorbidities such as cardiovascular disease and metabolic disorders. Progressive structural lung damage, coupled with frequent rehospitalizations and systemic complications, further compromises long-term prognosis.[3]

Nevertheless, evidence indicates that survival and functional outcomes may be optimized through timely and comprehensive management strategies, including appropriate pharmacologic therapy, structured pulmonary rehabilitation, smoking cessation programs, long-term oxygen therapy when indicated, and preventive vaccination.[3] One of the strategies is to determine the fundamental components of COPD.

Inflammation constitutes a fundamental and biologically heterogeneous component of COPD, with substantial interindividual variability in both cellular composition and clinical expression. Among the diverse inflammatory mediators involved, neutrophils and eosinophils have emerged as key cellular determinants of distinct inflammatory phenotypes. Neutrophilic inflammation predominates in a large proportion of patients, particularly in episodes associated with bacterial infection, and has been consistently linked to greater airflow obstruction and an increased propensity for recurrent exacerbations.[4]

Conversely, a subset of patients exhibits elevated eosinophil counts in peripheral blood or airway secretions, indicative of an eosinophil-driven inflammatory pattern. This phenotype has been associated with enhanced therapeutic responsiveness to inhaled corticosteroids and may demonstrate different exacerbation trajectories compared with neutrophil-dominant disease.[5]

Biomarker-based stratification has gained increasing attention in COPD research, particularly in efforts to delineate inflammatory phenotypes and refine risk prediction models for exacerbations. Peripheral blood eosinophil count has been incorporated into therapeutic decision-making algorithms, as recommended by the Global Initiative for Chronic Obstructive Lung Disease (GOLD), primarily to support individualized selection of inhaled corticosteroid therapy. In contrast, the prognostic significance of circulating neutrophil levels remains an area of ongoing investigation, especially regarding their potential association with long-term exacerbation burden and adverse clinical outcomes.[6]

Considering the biological plausibility and differential inflammatory pathways represented by these cell populations, evaluating the relationship between neutrophil and eosinophil profiles and the one-year exacerbation frequency among patients presenting with acute COPD may provide clinically meaningful insights into disease stratification and outcome prediction. This investigation will contribute to the evidence base supporting personalized therapeutic strategies and more precise risk stratification for exacerbations in COPD. By clarifying the clinical relevance of inflammatory cell profiles, this study aims to enhance understanding of prognostic assessment in acute COPD and to inform more individualized management approaches.

METHOD

This study employed a retrospective observational analytic design using secondary data obtained from hospital medical records. This study was approved by the Institutional Ethics Committee of Kolonel Abundjani Bangko General Hospital. Due to the retrospective nature of the study and the use of anonymized medical record data, the requirement for informed consent was waived.

The study was conducted at Kolonel Abundjani Bangko General Hospital, Jambi, Indonesia, and included patients admitted to the pulmonology ward between April and December 2024. The respondents of the study included all patients diagnosed with Acute exacerbation of COPD (AECOPD) who fulfilled the predefined inclusion criteria, such as patients with a confirmed diagnosis of AECOPD, complete documentation of neutrophil and eosinophil counts, and clearly recorded clinical outcomes, including length of hospitalization and discharge status. Respondents were excluded if there was no confirmed diagnosis of AECOPD, if significant comorbid conditions were documented, or if essential clinical or laboratory data were incomplete.

A total sampling technique was applied, whereby all eligible patients within the study period were included in the analysis. Therefore, no a priori sample size calculation was performed. This approach was chosen to reflect real-world clinical data available within the defined study timeframe.

A confirmed diagnosis of COPD was established based on documented clinical diagnosis in the medical records, supported, where available, by spirometric evidence demonstrating a post-bronchodilator forced expiratory volume in one second to forced vital capacity ratio (FEV_1/FVC) <0.7, in accordance with the Global Initiative for Chronic Obstructive Lung Disease (GOLD) criteria. AECOPD was defined as an acute worsening of respiratory symptoms that required additional therapy, as documented by the treating physician.

The variables collected included demographic characteristics (age and sex), smoking history, absolute neutrophil and eosinophil counts, and clinical outcomes, including length of hospital stay and discharge status (survived or deceased). Neutrophil and eosinophil levels were categorized based on clinically relevant thresholds.

Data were analyzed using IBM SPSS Statistics for Windows, version 25.0 (IBM Corp., Armonk, NY, USA). Descriptive statistics were used to summarize baseline characteristics, with categorical variables presented as frequencies and percentages. The distribution of continuous variables was assessed for normality. Normally distributed data were expressed as mean±standard deviation, whereas non-normally distributed data were presented as median values.

Inferential statistical analysis was performed to evaluate the association between inflammatory cell counts and clinical outcomes. The Chi-square test was used for categorical variables when assumptions were met. Otherwise, the Mann–Whitney U test was applied as a non-parametric alternative. A value of $P<0.05$ was considered statistically significant.

RESULT

Between April and December 2024, patients presenting with acute COPD and requiring hospitalization at the pulmonology ward of Kolonel Abundjani Bangko General Hospital were consecutively identified. During this period, 56 cases were recorded and underwent eligibility assessment based on the predefined inclusion and exclusion criteria specified in the study protocol. Following the screening process, 38 patients satisfied the eligibility criteria and were included in the final analytical cohort, whereas 18 patients were excluded due to the presence of exclusion factors.

Table 1 presents the baseline demographic and clinical profile of respondents. Of the 56 screened cases, 38 fulfilled the eligibility criteria and constituted the final study cohort. The respondents were overwhelmingly male (94.7%), and older than 55 years (89.5%), consistent with the age-related burden typically observed in acute COPD. A history of tobacco exposure was documented in 92.1% of patients, reinforcing the central etiological role of smoking within this study.

With respect to hospitalization outcomes, most patients experienced a relatively short length of stay, with 68.4% of patients discharged within 1–4 days. In-

hospital survival was high, as 94.7% of patients were discharged alive. Laboratory evaluation revealed a mean neutrophil count of 9.20±3.765 and a mean eosinophil count of 0.85±0.722. These hematological indices were subsequently examined to explore their potential association with duration of hospitalization and overall clinical outcomes.

Table 1. Characteristics of Patients with Acute Chronic Obstructive Pulmonary Disease

Characteristics	n	%
Gender		
Male	36	94.7
Female	2	5.3
Age		
<40 Years	1	2.6
40-55 Years	3	7.9
>55 Years	34	89.5
Smoker		
Yes	35	92.1
No	3	7.9
Length of Hospitalization		
1-4 Days	26	68.4
5-8 Days	12	31.6
Outcome		
Lived	36	94.7
Died	2	5.3
Absolute Neutrophil Count	9.20±3.765*	
Absolute Eosinophil Count	0.85±0.722*	

Note: *Mean±SD

Table 2 presents the distribution of hospitalization duration according to categorized neutrophil levels. Elevated neutrophil counts (>8) were observed predominantly in both length-of-stay groups, comprising 76.9% of patients hospitalized for 1–4 days and 58.3% of those hospitalized for 5–8 days. Although higher neutrophil values were common across both categories, inferential analysis demonstrated no statistically significant relationship between neutrophil count and duration of hospitalization (P=0.240), suggesting that neutrophil elevation alone did not predict length of stay in patients with acute COPD.

A comparable pattern was observed in the analysis of eosinophil levels. The majority of patients in both the 1–4 day and 5–8 day hospitalization groups exhibited eosinophil counts within the 0.1–1.5 range, accounting for 76.9% and 91.7%, respectively. Despite this consistent distribution across groups, statistical testing revealed no significant association between eosinophil count and hospital stay duration (P=0.241). These findings indicate that, within this cohort, neither neutrophil nor eosinophil levels were independently associated with the length of inpatient management during acute COPD episodes.

Table 2. Relationship between Neutrophil and Eosinophil Levels and Length of Hospitalization

Variables	Length of Hospitalization		P
	1-4 Days	5-8 Days	
Neutrophil Cell			
<1.2	0 (0%)	0 (0%)	
1.2-8	6 (23.1%)	5 (41.7%)	0.240[a]
>8	20 (76.9%)	7 (58.3%)	
Eosinophil Cell			
<0.1	1 (3.8%)	1 (8.3%)	
0.1-1.5	20 (76.9%)	11 (91.7%)	0.241[a]
>1.5	5 (19.2%)	0 (0%)	

Note: [a]Mann–Whitney U test

Table 3 summarizes the distribution of clinical outcomes according to categorized neutrophil levels in patients with AECOPD. Elevated neutrophil counts (>8) were observed in the majority of patients across both outcome groups,

representing 69.4% of survivors and 100% of non-survivors. Despite the apparent predominance of higher neutrophil values in both categories, inferential analysis did not demonstrate a statistically significant association between neutrophil level and in-hospital outcome (P=0.354). These findings suggest that neutrophil elevation alone was not an independent predictor of mortality within this study.

A similar trend was identified in the evaluation of eosinophil levels. Most patients, irrespective of survival status, exhibited eosinophil counts within the 0.1–1.5 range, accounting for 80.6% of survivors and 100% of non-survivors. Although this distribution indicates that eosinophil levels were largely within the normal range across outcome groups, statistical testing revealed no significant relationship between eosinophil count and clinical outcome (P=0.788). Collectively, these results indicate that neither neutrophil nor eosinophil levels were significantly associated with mortality among patients hospitalized for acute COPD in this cohort.

Table 3. Relationship between Neutrophil and Eosinophil Levels and Outcome

Variables	Outcome		P
	Lived	Died	
Neutrophil Cell			
< 1.2	0 (0%)	0 (0%)	
1.2-8	11 (30.6%)	0 (0%)	0.354[a]
>8	25 (69.4%)	2 (100%)	
Eosinophil Cell			
< 0.1	2 (5.6%)	0 (0%)	
0.1-1.5	29 (80.6%)	2 (100%)	0.788[a]
>1.5	5 (13.9%)	0 (0%)	

Note: [a]Mann–Whitney U test

DISCUSSION

In the present cohort of patients admitted with acute COPD, males constituted an overwhelming majority (94.7%), indicating a pronounced sex-related disparity in disease presentation. This pattern mirrors epidemiological data reported by Zhang et al, in which male patients represented nearly two-thirds of the COPD population, highlighting the sustained predominance of COPD among men.[7]

The age profile of this study was similarly skewed toward older individuals, with 89.5% of participants aged above 55 years. Such findings are in line with observations by MacNee, who demonstrated a markedly higher prevalence of COPD among individuals older than 60 years.[8] Otherwise, a strikingly high proportion of participants (92.1%) reported a history of smoking, emphasizing the pivotal contribution of tobacco exposure to COPD development and clinical progression.[7]

Beyond these epidemiological observations, several biological and pathophysiological mechanisms may further explain the predominance of males in acute exacerbations of COPD. Long-term exposure to cigarette smoke induces persistent airway inflammation, oxidative stress, and structural remodeling of the lung parenchyma.[4]

In addition, sex-related differences in airway structure, inflammatory responses, and hormonal regulation have been shown to influence susceptibility to COPD and its clinical manifestations.[9] Estrogen and other

sex hormones play a role in modulating immune and inflammatory pathways in the lung, potentially contributing to differences in disease expression between males and females.[10]

Furthermore, genetic and chromosomal factors, as well as differences in occupational and environmental exposures, have also been implicated in sex-related variability in chronic respiratory diseases.[11] Historically, COPD has been more prevalent in men due to higher smoking exposure, although recent evidence suggests an increasing burden among women as well.[12] Taken together, these behavioral, biological, and environmental factors may explain the higher proportion of male patients observed in this study.

In this study, no statistically significant association was identified between circulating neutrophil levels and the duration of hospitalization among patients admitted with acute COPD (P=0.240). Although neutrophilia has been linked to heightened inflammatory activity and greater exacerbation burden, its influence on hospital stay appears to diminish when demographic and clinical confounders are taken into account.

Comparable observations were reported by Lonergan et al.[13] From a mechanistic perspective, neutrophils play a central role in COPD-related inflammation through the release of proteases, reactive oxygen species, and neutrophil extracellular traps, contributing to airway damage and disease progression.[14]

However, their levels alone may not adequately reflect disease severity or predict short-term outcomes without integration with other clinical variables. A similar pattern emerged in the evaluation of eosinophil counts. The present analysis did not demonstrate a significant correlation between peripheral eosinophil levels and length of stay (P=0.241).

This is in agreement with findings from David et.[15] Eosinophils are known to represent a distinct inflammatory phenotype in COPD, particularly associated with corticosteroid responsiveness, yet their role as independent predictors of acute clinical outcomes remains inconsistent across studies.

In the present cohort of patients hospitalized for acute COPD, no statistically significant association was observed between circulating neutrophil levels and in-hospital survival (P=0.354). Similar conclusions were drawn by Jiang et al.[16] An analogous pattern was identified for eosinophil counts, consistent with Vanetti et al.[17]

These findings suggest that while inflammatory biomarkers reflect underlying pathophysiology, their isolated measurement may not provide sufficient prognostic discrimination. Instead, multidimensional models incorporating clinical, functional, and biological parameters may offer more robust prognostic value.

Several limitations of this study should be acknowledged. First, the relatively small sample size (n=38) may have limited the statistical power and

increased the likelihood of type II error. Second, the retrospective design introduces inherent limitations, including potential information bias and incomplete data recording. Important confounding variables, such as disease severity (e.g., GOLD stage), pulmonary function parameters, comorbid conditions, and treatment variations, were not consistently available. These unmeasured factors may have influenced both inflammatory biomarker levels and clinical outcomes. Despite these limitations, this study provides valuable insight into inflammatory cell profiles in acute COPD within a real-world clinical setting and may serve as a basis for future prospective investigations.

CONCLUSION

In this study, neither neutrophil nor eosinophil counts demonstrated a statistically significant association with prognostic indicators, including duration of hospitalization and survival outcomes, among patients admitted with acute COPD. Despite the absence of statistical significance, elevated neutrophil levels (>8) were consistently more prevalent across prognostic strata compared with increased eosinophil counts. This distribution pattern suggests a relative predominance of neutrophil-mediated inflammatory activity during acute exacerbations. Although this tendency did not translate into measurable prognostic discrimination, it may reflect the underlying inflammatory milieu characteristic of acute COPD episodes, where neutrophilic responses are typically more pronounced than eosinophilic involvement.

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