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THE CORRELATION OF PERIPHERAL EOSINOPHILIA WITH THE SEVERITY AND EXACERBATION IN CHRONIC OBSTRUCTIVE PULMONARY DISEASE PATIENTS

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ABSTRACT

Background: Chronic Obstructive Pulmonary Disease (COPD) is a heterogeneous disorder characterized by persistent airflow limitation and diverse inflammatory phenotypes. The eosinophilic phenotype has gained attention because of its potential association with exacerbation risk, disease severity, and corticosteroid responsiveness. **Aim:** This study evaluated the relationship between peripheral blood eosinophil counts and markers of disease severity, including spirometric indices, GOLD stage, and recent exacerbation frequency, among patients with COPD. **Methods:** A cross-sectional analytical study was conducted on 100 patients aged ≥40 years with confirmed COPD (post-bronchodilator FEV1/FVC <0.70) attending outpatient clinics at Mirjan Medical City, Babylon, Iraq. Peripheral blood eosinophil counts were measured and patients were categorized into two groups: <300 cells/µL and ≥300 cells/µL. Pulmonary function was assessed using spirometry. Associations between eosinophil levels and FEV1% predicted, GOLD stage, and exacerbations in the preceding six months were analyzed using Spearman's rank correlation. **Results:** The median eosinophil count was 247.5 cells/µL. Patients with eosinophil counts ≥300 cells/µL demonstrated significantly lower median FEV1% predicted (48% vs. 75%; p<0.001) and post-bronchodilator FEV1 (55% vs. 79%; p<0.001). Eosinophil counts increased progressively across GOLD stages, from 62.5 cells/µL in stage 1 to 805 cells/µL in stage 4. Significant negative correlations were observed between eosinophil counts and lung function parameters, while positive correlations were identified with GOLD stage (ρ=0.623, p<0.001) and exacerbation frequency (ρ=0.490, p<0.001). **Conclusion:** Elevated peripheral eosinophil counts are strongly associated with greater airflow limitation, advanced disease severity, and increased exacerbation burden in COPD, supporting their role as an accessible biomarker for high-risk phenotyping.

KEYWORDS: Chronic Obstructive Pulmonary Disease, Eosinophils, Phenotype, Spirometry, Exacerbation.

INTRODUCTION

Global Initiative for Chronic Obstructive Lung Disease (GOLD) defines Chronic Obstructive Pulmonary Disease (COPD) as a heterogeneous lung condition characterized by chronic respiratory symptoms—such as dyspnea, cough, sputum production, and exacerbations—resulting from airway (bronchitis, bronchiolitis) and/or alveolar (emphysema) abnormalities that lead to persistent, usually progressive airflow limitation.[1] Chronic bronchitis is clinically defined as a productive cough lasting at least three months in each of two consecutive years, after exclusion of other causes of chronic cough. Emphysema, in contrast, refers to permanent enlargement of airspaces distal to the terminal bronchioles accompanied by destruction of alveolar walls, a key pathological hallmark frequently observed in COPD. Airflow limitation is physiologically assessed by spirometry, particularly through measurement of the forced expiratory volume in one second (FEV_1) and forced vital capacity (FVC). A post-bronchodilator

FEV_1/FVC ratio <0.70 confirms persistent airflow obstruction and is essential for diagnosis.[1] Although some patients demonstrate partial reversibility following bronchodilator administration, airflow limitation is considered persistent unless lung function returns to normal ranges. The severity of COPD is further stratified according to GOLD staging based on FEV_1% predicted values, which correlate with symptom burden and future risk of exacerbations. Traditionally, COPD has been associated with neutrophilic airway inflammation. However, growing evidence indicates that a substantial subgroup of patients—estimated at 20–40%—exhibits elevated eosinophil counts in peripheral blood, sputum, or bronchial tissue.[2] This eosinophilic phenotype has emerged as a clinically relevant inflammatory pattern with potential implications for disease progression and exacerbation susceptibility. Studies suggest that eosinophilic inflammation may contribute to airway remodeling, heightened inflammatory responses, and increased risk of acute exacerbations.[3] Peripheral blood eosinophil (PBE) count has gained attention as a practical and reproducible biomarker for identifying this phenotype. Higher eosinophil levels, particularly ≥300 cells/μL, have been associated with an increased likelihood of response to inhaled corticosteroids (ICS), especially in reducing exacerbation frequency. Conversely, patients with low eosinophil counts (<100 cells/μL) appear to derive minimal benefit from ICS therapy and may be exposed to unnecessary adverse effects, including pneumonia. Consequently, recent GOLD reports recommend incorporating blood eosinophil counts into therapeutic decision-making, particularly when initiating or withdrawing ICS treatment.[1] Given the clinical relevance of eosinophilic inflammation, further evaluation of its relationship with disease severity and exacerbation burden is warranted. Therefore, this study aims to investigate the correlation between peripheral eosinophilia and markers of severity, including spirometric indices and exacerbation frequency, among patients with COPD.

METHOD

This observational cross-sectional analytical study was conducted at Mirjan Medical City, Babylon, Iraq. The design incorporated both cross-sectional and retrospective components. Peripheral blood eosinophil counts and spirometric parameters were measured at the time of recruitment, whereas data regarding exacerbation frequency during the preceding six months were obtained retrospectively from medical records and patient interviews. A total of 100 patients diagnosed with Chronic Obstructive Pulmonary Disease were consecutively enrolled from outpatient clinics. The sample size was guided by previously reported prevalence rates indicating that approximately 20–40% of COPD patients exhibit an eosinophilic phenotype. Inclusion criteria were: age ≥40 years; spirometric confirmation of persistent airflow limitation defined as a post-bronchodilator FEV_1/FVC ratio <0.70; and a history of significant smoking exposure (≥10 pack-years), including current and former smokers. Exclusion criteria included physician-diagnosed asthma or asthma–COPD overlap, autoimmune or systemic inflammatory diseases, drug-induced eosinophilia, and known solid or hematologic malignancies. For peripheral blood eosinophil (PBE) assessment, 3–5 mL of venous blood was collected from the antecubital vein into potassium-EDTA tubes. Samples were analyzed using an automated 5-part differential hematology analyzer (Siemens ADVIA® 2120i or equivalent Diagon D-Cell 60), which differentiates leukocyte populations based on peroxidase activity and nuclear density. Participants were stratified into two groups: Group A (<300 cells/μL) and Group B (≥300 cells/μL). Demographic and clinical data collected included age, sex, residence (urban/rural), smoking status, body mass index (BMI), and comorbidities (hypertension and diabetes mellitus). Pulmonary function tests were performed before and after bronchodilator administration, and disease severity was classified according to GOLD grading based on post-bronchodilator FEV_1% predicted. The primary outcome was the frequency of COPD exacerbations within the previous six months, categorized by need for hospitalization or home-based management. Ethical approval was obtained from the Iraqi Council of Medical Specializations, and the study adhered to the Declaration of Helsinki principles. Verbal informed consent was secured from all participants. Statistical analysis was performed using JAMOVI (version 2.2.8). Continuous variables were assessed for normality using the Shapiro–Wilk test and expressed as median and interquartile range. Categorical variables were presented as frequencies and percentages. Group comparisons were conducted using the Mann–Whitney U test, and correlations were evaluated using Spearman's rank correlation coefficient. A p-value <0.05 was considered statistically significant.

RESULTS

A total of 100 patients with confirmed Chronic Obstructive Pulmonary Disease were included in the present analysis. Comprehensive demographic, clinical, and laboratory characteristics were evaluated for all participants. The median age was 64 years (IQR 58.75–68; range 47–75), reflecting a predominantly older adult population. The median body mass index (BMI) was 22 kg/m² (IQR 21–24; range 19–32), indicating that most patients were within the normal to lower-normal weight range. Pulmonary function testing demonstrated overall moderate airflow limitation. The median forced expiratory volume in one second (FEV_1) was 64% of the predicted value (IQR 48–82%; range 26–89%). Similarly, the median post-bronchodilator FEV_1 was 65.5% of predicted (IQR 51.75–84%; range 27–91%), confirming persistent but variably severe airflow obstruction across the cohort. Regarding inflammatory markers, the median peripheral blood eosinophil count was 247.5 cells/μL (IQR 87.5–547.5; range 0–1395). The corresponding median eosinophil percentage was 2% (IQR 1–6%; range 0–9%). These findings

demonstrate considerable interindividual variability in eosinophil levels among patients with COPD. A detailed summary of baseline demographic, spirometric, and laboratory parameters is presented in Table 1.

Table 1: Baseline Characteristics of Continuous Variables in COPD Patients.

Variable	Median (IQR)	Minimum - Maximum
Ages(years)	64 (58.75 - 68)	47 - 75
BMI (kg/m2)	22 (21 - 24)	19 - 32
FEV1(%)	64 (48 - 82)	26 - 89
FEV1/post BD(%)	65.5 (51.75 - 84)	27 - 91
Eosinophils (cells/µL)	247.5 (87.5 - 547.5)	0 - 1395
eosinophils (%)	2 (1 - 6)	0 - 9

Among the 100 patients included in the study, 82% were male and 18% were female, indicating a marked male predominance. The majority of participants (78%) resided in urban areas, while 22% were from rural regions. Regarding smoking status, 86% were current smokers and 14% were ex-smokers, reflecting the strong association between smoking exposure and Chronic Obstructive Pulmonary Disease. Common comorbidities included hypertension in 27% of patients and diabetes mellitus in 22%. Based on GOLD severity classification using post-bronchodilator FEV_1% predicted, 28% of patients were categorized as GOLD stage 1, 41% as GOLD stage 2, 23% as GOLD stage 3, and 8% as GOLD stage 4. With respect to exacerbation history, most patients (82%) experienced fewer than two exacerbations in the preceding year, whereas 18% reported two or more exacerbations. When comparing baseline characteristics according to peripheral blood eosinophil levels (<300 cells/µL vs. ≥300 cells/µL), significant differences were observed. Patients in the high-eosinophil group were older, with a median age of 65 years compared to 62 years in the normal-eosinophil group ($p = 0.007$). They also had a lower median body mass index (21 vs. 23 kg/m², $p = 0.026$). Pulmonary function parameters were significantly worse among patients with elevated eosinophil counts. The median FEV_1% predicted was 48% in the high-eosinophil group versus 75% in the normal-eosinophil group ($p < 0.001$). Similarly, the median post-bronchodilator FEV_1% predicted was 55% compared to 79%, respectively ($p < 0.001$). These comparative findings are detailed in Table 2.

Table 2: Comparison of baseline Clinical and spirometry Parameters Between Normal and High Blood Eosinophil Groups.

Variable	Normal (<300)	High (≥300)	p-value
Age (year)	62 (56.5 - 67.5)	65 (62 - 69)	0.007
BMI	23 (22 - 25)	21 (21 - 23)	0.026
FEV1(%)	75 (58 - 85)	48 (39 - 60)	<0.001
FEV1/post-BD(%)	79 (60 - 87)	55 (45 - 65)	<0.001

As presented in Table 3, a progressive increase in peripheral blood eosinophil counts was observed with advancing disease severity according to GOLD classification in patients with Chronic Obstructive Pulmonary Disease. Patients classified as GOLD stage 1 had the lowest median eosinophil count at 62.5 cells/µL (IQR 0–153). In GOLD stage 2, the median count increased to 240 cells/µL (IQR 100–390). This upward trend became more pronounced in the advanced stages, where GOLD stage 3 patients demonstrated a median eosinophil count of 630 cells/µL (IQR 290–788). The highest levels were recorded in GOLD stage 4, with a median of 805 cells/µL (IQR 473–990). Overall, these findings indicate a clear positive gradient between GOLD stage and peripheral eosinophil levels, suggesting that more severe airflow limitation is associated with higher systemic eosinophilic inflammation.

Table 3: Distribution of Blood Eosinophil Counts Across GOLD Severity Stages.

GOLD stage	Median(IQR)	Minimum	Maximum	Number
GOLD 1	62.5 (0-153)	0	465	28
GOLD 2	240 (100-390)	0	1200	41
GOLD 3	630 (290-788)	80	1200	23
GOLD 4	805 (473-990)	260	1395	8

Spearman's rank correlation analysis (Table 4) revealed significant associations between peripheral blood eosinophil counts and established indices of disease severity in patients with Chronic Obstructive Pulmonary Disease. Peripheral eosinophil levels demonstrated strong negative correlations with lung function parameters. Specifically, eosinophil count was inversely correlated with FEV_1% predicted ($\rho = -0.645$, $p < 0.001$) and with post-bronchodilator FEV_1% predicted ($\rho = -0.568$, $p < 0.001$), indicating that higher eosinophil

counts were associated with more pronounced airflow limitation. In contrast, eosinophil counts showed significant positive correlations with markers of disease severity. A strong positive relationship was observed between eosinophil count and GOLD stage ($\rho = +0.623$, $p < 0.001$), reflecting increasing eosinophilic inflammation with advancing airflow obstruction. Additionally, eosinophil levels were positively correlated with the number of COPD exacerbations reported in the previous year ($\rho = +0.490$, $p < 0.001$), suggesting a link between eosinophilia and heightened exacerbation risk. All correlations were statistically significant at the $p < 0.001$ level, underscoring a consistent pattern whereby elevated peripheral blood eosinophil counts were associated with worse lung function, higher GOLD severity stages, and increased exacerbation frequency within this cohort.

Table 4: Spearman's correlation between eosinophil count and indices of COPD severity.

Variable	spearman's rho	p-value
FEV1(%)	- 0.645	<0.001
FEV1 post-BD(%)	- 0.568	<0.001
GOLD stage	+ 0.623	<0.001
Number of exacerbation in the last 6 months	+ 0.49	<0.001

DISCUSSION

In this cohort of 100 patients with Chronic Obstructive Pulmonary Disease, elevated peripheral blood eosinophil counts (≥300 cells/μL) were significantly associated with older age, lower BMI, greater airflow limitation, higher GOLD stage, and increased exacerbation frequency. These findings suggest that eosinophilia identifies a subgroup of patients with more advanced disease and greater clinical instability. The concept of eosinophilic COPD as a distinct inflammatory phenotype is increasingly recognized. Approximately 20–40% of patients demonstrate elevated blood or sputum eosinophils, reflecting a "type 2" inflammatory pattern traditionally linked to asthma but now acknowledged in COPD.[4,5] This phenotype has been associated with heightened exacerbation risk and differential therapeutic responsiveness. Our results align with previous evidence indicating that patients with higher eosinophil counts experience more frequent exacerbations and worse lung function indices. Large longitudinal studies have produced mixed findings regarding the predictive value of blood eosinophils. The ECLIPSE and COPD Gene cohorts demonstrated that blood eosinophil counts, particularly ≥300 cells/μL, were associated with increased exacerbation risk.[3] Furthermore, the SPIROMICS study suggested that sputum eosinophils may better predict exacerbation risk than blood eosinophils, highlighting ongoing controversy regarding the optimal biomarker. Beyond exacerbations, emerging imaging studies have linked higher eosinophil counts with greater emphysema burden on computed tomography and lower diffusing capacity (DLCO), suggesting potential relationships between systemic eosinophilia and structural lung damage.[6] Conversely, other observational analyses, including the CHAIN and BODE cohorts and subsequent meta-analyses, reported weaker or non-significant associations in stable patients.[7,8] Our observation of progressively rising eosinophil counts across GOLD stages supports the hypothesis that eosinophilic inflammation may contribute to disease progression or reflect a more severe inflammatory milieu.

CONCLUSION

The present study demonstrates that elevated peripheral blood eosinophil levels are significantly associated with worse airflow limitation, advanced GOLD staging, and higher exacerbation frequency. These findings reinforce the clinical relevance of eosinophils as a readily accessible biomarker for identifying a high-risk COPD phenotype. Stratifying patients according to inflammatory profile may help guide individualized management, particularly in decisions regarding corticosteroid therapy, while acknowledging that the predictive role of blood eosinophils remains an area of active investigation.

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