AMERICAN JOURNAL OF BIOLOGY AND NATURAL SCIENCES
https://biojournals.us/index.php/AJBNS
ISSN: 2997-7185

Article

Blood Eosinophil Count as a Diagnostic and Severity Biomarker in Bronchial Asthma: Correlations with Pulmonary Function and Peripheral Oxygen Saturation

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Citation: Talib A. M., Shaheed O. M. Blood Eosinophil Count as a Diagnostic and Severity Biomarker in Bronchial Asthma: Correlations with Pulmonary Function and Peripheral Oxygen Saturation. American Journal of Biology and Natural Sciences 2026, 3(5), 47-59.

Received: 06th Feb 2026
Revised: 11th Mar 2026
Accepted: 16th Apr 2026
Published: 11th May 2026

Abstract: Airway eosinophilic inflammation characterizes the leading phenotype of adult asthma, and BEC is considered a convenient biomarker for representing the level of underlying airway inflammation and severity of asthma. The current case-control study was carried out among adults visiting Al-Diwaniyah Teaching Hospital from October 2024 to March 2025. Participants included patients of bronchial asthma with subgroups of different severity (mild, moderate, and severe asthma) and an equal number of control subjects without any underlying disease. Blood eosinophils were measured by automated five-part differential count test. Spirometric indices, including FEV_1%, FVC%, FEV_1/FVC, as well as peripheral oxygen saturation, were evaluated among all participants. BEC was found to be significantly elevated in asthmatic patients as compared to control participants (2.5 times greater). Furthermore, the increase was statistically significant and exhibited a stepwise trend along with increasing severity subgroups with the rising eosinophilic inflammatory process ($P < 0.001$, Kruskal-Wallis). Inverse significant correlation was detected between BEC and all evaluated indices – FEV_1%, FVC%, FEV_1/FVC, as well as SpO_2, while positive correlations were obtained with duration and exacerbations of asthma (all $P < 0.001$). ROC curve analysis revealed high accuracy of BEC for diagnosing asthma (area under the curve = 94.8%; 95% CI: 90.7–99.0%) and an appropriate trade-off between sensitivity (90.0%) and specificity (88.3%).

Keywords: Bronchial Asthma, Eosinophil, FEV_1, FVC, SpO_2.

Introduction

Bronchial asthma is a chronic inflammatory disease of the airways that is characterized by reversible airflow limitation, hyperresponsiveness of the airways, and structural remodeling of the airways. It affects approximately 262 million people worldwide, resulting in 21.6 million disability-adjusted life years annually. Epidemiological projections have indicated that the disease will remain at high incidence rates through the year 2050 [1]. In Iraq, the disease burden of asthma is considerable, affecting the adult population of all ages without any particular age predilection, a fact that is substantiated by the recently conducted epidemiological study from the Al-Diwaniyah region of Iraq [2]. The fundamental abnormality in the pathophysiology of asthma is type 2 eosinophilic airway inflammation. Eosinophils migrate into the bronchial mucosa in the presence of the guidance factor

interleukin (IL)-5, the major eosinophilopoietic and eosinophil-activating cytokine, produced by $CD4^+$ T helper 2 (Th2) lymphocytes, type 2 innate lymphoid cells (ILC2s), and mast cells in the context of allergen exposure [3]. Once in the airway wall, activated eosinophils release a variety of granule proteins, including major basic protein (MBP), eosinophil cationic protein (ECP), and eosinophil-derived neurotoxin (EDN), as well as reactive oxygen species and lipid mediators, which have the capacity to induce epithelial shedding, smooth muscle dysfunction, subepithelial fibrosis, and mucus hypersecretion, which have a direct impact on the lung function, lung volumes, and gas exchange [3, 4]. Blood eosinophil count is a commonly measured, reproducible, and cost-effective hematologic parameter that is indicative of the systemic component of eosinophilic inflammatory response and indirectly reflects the degree of airway eosinophilia [5]. Moreover, the 2023 edition of the Global Initiative for Asthma (GINA) guidelines includes the BEC as a clinically relevant endotype marker to be considered when determining the step-up treatment strategy and the use of biologics targeting the IL-5 and IL-4/IL-13 pathways [6]. A count of ≥300 cells/μL is considered the primary criterion to diagnose the eosinophilic phenotype of asthma, which is present in 50-60% of all asthma patients worldwide [5, 6]. Pulmonary function tests such as FEV1, FVC, and FEV1/FVC remain the objective gold standard to measure the degree of airflow limitation or the severity of asthma. Oxygen saturation measured as SpO2 is a non-invasive test to monitor the degree of respiratory compromise, which is particularly valuable in patients with moderate to severe disease. The mechanistic link between eosinophilic airway inflammation and the objective parameters of respiratory compromise is the biological rationale to expect a positive correlation between the degree of eosinophilic inflammation reflected as BEC and the objective parameters of respiratory compromise. The mechanistic link between eosinophilic airway inflammation and the objective parameters of respiratory compromise is the biological rationale to expect a positive correlation between the degree of eosinophilic inflammation reflected as BEC and the objective parameters of respiratory compromise [4, 7]. Despite the established clinical significance of blood eosinophils as a biomarker of the degree of eosinophilic inflammation, the complete characterization of the association between the blood eosinophil count and the entire range of spirometric parameters and SpO2 in the GINA severity subgroups of adult Iraqi patients with asthma was not previously reported. Accordingly, the present study was conducted to compare the blood eosinophil count between patients with asthma and healthy controls, to characterize the blood eosinophil count across the range of mild, moderate, and severe subgroups of patients with asthma, to quantify the correlation between the blood eosinophil count and FEV1%, FVC%, FEV1/FVC, SpO2, disease duration, and exacerbation frequency, and to determine the diagnostic accuracy of the blood eosinophil count using the receiver operating characteristic curve analysis.

Materials and Methods

2.1. Study Design and Ethical Approval

A case-control study was carried out in Al-Diwaniyah Teaching Hospital, Department of Chest and Respiratory Diseases, and the Allergy and Asthma Centre from October 2025 to March 2026. The study was approved by the Ethics Committee of the College of Medicine, University of Al-Qadisiyah. The study was carried out in full compliance with the ethical principles for medical research involving human subjects set forth in the Declaration of Helsinki. Informed consent was obtained from all subjects prior to the study.

2.2. Study Participants

A total of 120 adult subjects were included in the study, of whom 60 were patients with established bronchial asthma and 60 were apparently healthy subjects who acted as the control group. In the study group, the patients with bronchial asthma were divided into mild persistent (25 patients), moderate persistent (20 patients), and severe persistent (15 patients) groups according to the severity of the disease as per the GINA criteria 2023. The control subjects were matched individually with the study subjects for age and sex. In addition, the control subjects had no history of asthma, chronic

respiratory disease, atopic disease, autoimmune disease, or any systemic disease. Also, the control subjects were non-smokers and were not on any regular medication.

2.2.1. Inclusion Criteria

Eligibility criteria for participants to be enrolled in the study included the following conditions being met: (1) the participant should be between 18 years and 70 years of age; (2) the participant should have a confirmed diagnosis of bronchial asthma as certified by a consultant pulmonologist with spirometric evidence of reversible airflow obstruction as indicated by the FEV1/FVC ratio less than 0.70 and/or reversibility of ≥ 12% and ≥ 200 mL in FEV1, as per GINA 2023 criteria; (3) the participant should be willing to be enrolled in the study and should give informed consent to be part of the study; and (4) the participant should not have acute infection as indicated by normal C-reactive protein levels.

2.2.2. Exclusion Criteria

Exclusion criteria included the presence of any of the following: (1) co-existing chronic respiratory diseases (chronic obstructive pulmonary disease, bronchiectasis, interstitial lung disease); (2) systemic comorbid diseases (heart failure, hepatic or renal disease); (3) autoimmune or primary immunodeficiency diseases; (4) morbid obesity (BMI >40 kg/m²); (5) substantial smoking history; (6) systemic immunosuppressive or corticosteroid therapy; or (7) malignancy history. All participants had blood CRP levels assessed, and those with abnormal results were excluded as it could be a confounding factor if they had any underlying inflammatory condition.

2.3. Blood Sample Collection

Five milliliters of venous blood were obtained from each of the participants under aseptic precautions using the standard venipuncture method. Two milliliters of blood were taken in K2-EDTA anti-coagulation tubes (Al-Malak, China), and complete blood count (CBC) analysis was performed. Three milliliters of blood were taken in plain gel tubes, left at room temperature for 10-20 minutes, followed by centrifugation at 3,200 rpm for 10 minutes to obtain serum, which was stored at -80°C until required for the analysis of C-reactive protein (CRP).

2.4. Determination of Blood Eosinophil Count

The percentage of eosinophils (Eos%) and the absolute number of eosinophils (cells/μL) were obtained as part of a complete blood count with a five-part differential leukocyte analysis by using the Zybio Z50 automated hematology analyzer (Zybio Inc., Chongqing, China), as per the manufacturer's instructions. All samples were processed within two hours of sample collection to assure reliable analysis.

2.5. Spirometric Assessment

Spirometric parameters, including forced expiratory volume in 1 second (FEV_1) % predicted, forced vital capacity (FVC) % predicted, and FEV_1/FVC ratio, were measured using a calibrated spirometer according to American Thoracic Society/European Respiratory Society (ATS/ERS) standardization guidelines. For each individual, a minimum of three acceptable and reproducible spirometric efforts were obtained. Post-bronchodilator spirometry was carried out after inhalation of 200 μg salbutamol using a metered-dose inhaler with a spacer. Reversibility was defined as an increase in FEV_1 ≥ 12% and ≥ 200 mL. Oxygen saturation was measured using pulse oximetry at rest before spirometry.

2.6. Statistical Analysis

Statistical analyses were carried out using the Statistical Package for the Social Sciences (SPSS) software version 26 (IBM Corporation, Armonk, NY, USA) and Microsoft Office Excel 2010. Normality of the data was tested using the Kolmogorov-Smirnov test. Normally distributed continuous data were presented as the mean ± SD, whereas non-normally distributed data were presented as the median along with the interquartile range. Categorical data were shown as the frequency and percentage. Independent samples t-tests were used for analyzing normally distributed continuous data, whereas the Mann-Whitney U test was used for analyzing non-normally distributed data. The Kruskal Wallis H test was used to test the hypothesis that the data were from four different populations. Pairwise

comparisons were done using the Mann-Whitney U test. Since there were six pairwise comparisons, the significance level was corrected using the Bonferroni method. Therefore, the significance level was set at $P < 0.0083$. The chi-square test was used for analyzing categorical data. Spearman correlation coefficients were calculated to study the correlation between BEC and the functional parameters in the asthma group. The ROC curve was used to study the efficacy of BEC as a diagnostic tool. The cutoff values were determined using the Youden Index. The area under the curve, 95% CI, sensitivity, specificity, positive predictive values, and negative predictive values were calculated. Statistical significance was set at $P \leq 0.05$, which was considered high if $P \leq 0.001$.

Results

3.1. Demographic and Clinical Characteristics

Table 1 and Figure 1 show the demographic and baseline clinical characteristics of the four study groups. The total number of participants was 120, comprising 60 asthmatic patients and 60 healthy controls. The asthmatic patients had mild, moderate, or severe asthma, comprising 25, 20, and 15 patients, respectively. The mean age of the asthmatic patients was 50.63 ± 15.50 years, significantly higher than the age of the control group, at 43.42 ± 15.35 years ($P = 0.012$). The mean ages of the mild, moderate, and severe asthmatic patients were 54.48 ± 14.93 years, 50.60 ± 16.26 years, and 44.27 ± 14.18 years, respectively. The females outnumbered the males in all the groups, but the ratio of males to females was not significantly different between the asthmatic patients and the control group (43.3% vs 45.0% male, $P = 0.860$). The mean body mass index (BMI) of the asthmatic patients was significantly higher than that of the control group (27.77 ± 5.15 kg/m² vs 22.37 ± 2.59 kg/m², $P < 0.001$), especially in the severe asthma subgroup (34.46 ± 4.55 kg/m²). The disease duration and number of exacerbations increased with the severity of asthma ($P < 0.001$).

Table 1. Demographic and Clinical Characteristics of Study Participants.

Variable	Control (n=60)	Mild (n=25)	Moderate (n=20)	Severe (n=15)	P-value
Age (years) Mean ± SD	43.42 ± 15.35	54.48 ± 14.93	50.60 ± 16.26	44.27 ± 14.18	0.012†
Sex: Male Female	27 (45.0%) 33 (55.0%)	9 (36.0%) 16 (64.0%)	10 (50.0%) 10 (50.0%)	7 (46.7%) 8 (53.3%)	0.860¥
BMI (kg/m²) Mean ± SD	22.37 ± 2.59	25.09 ± 2.93	26.10 ± 2.94	34.46 ± 4.55	<0.001‡
Disease Duration (years) Mean ± SD	—	5.16 ± 2.42	11.39 ± 3.38	22.05 ± 6.04	<0.001‡
Exacerbations/Year Mean ± SD	—	0.97 ± 0.71	3.13 ± 0.63	6.14 ± 1.28	<0.001‡

† Independent samples t-test; ¥ Chi-square test; ‡ Kruskal–Wallis H test. All asthma subgroups combined vs control.

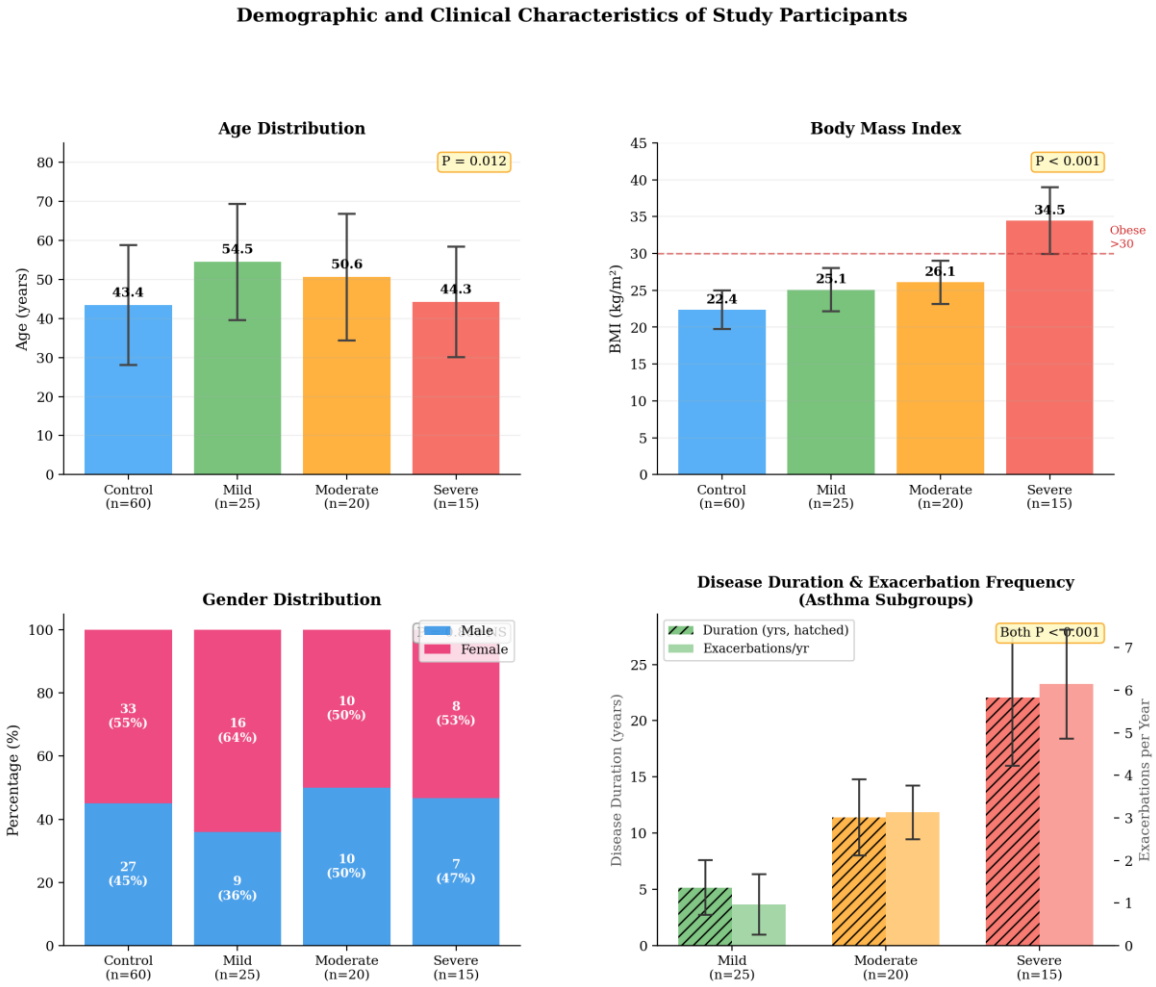

Figure 1. Demographic and clinical characteristics of study participants. (A) Age distribution (Mean ± SD). (B) Body mass index; dashed line = obesity threshold (BMI > 30 kg/m²). (C) Sex distribution (stacked bars). (D) Disease duration (hatched bars, left axis) and exacerbation frequency (solid bars, right axis) for asthma subgroups.

3.2. Blood Eosinophil Count Across Study Groups

The hematologic values of eosinophils in all the four study populations are presented in Table 2, while Figures 2 and 3 show the results graphically. The absolute count of eosinophils was found to be significantly higher in patients suffering from asthma (529.36 ± 240.40 cells/μL; median: 535, IQR: 335-700) compared to the control population (214.19 ± 47.48 cells/μL; median: 210, IQR: 181-243; $P < 0.001$). An increasing trend was evident in the eosinophil count as the severity of asthma increased, as confirmed by the Kruskal-Wallis H test ($H = 82.74$; $P < 0.001$). The count was found to be 323.35 ± 68.18 cells/μL in mild asthma, while it was 598.52 ± 107.64 cells/μL in moderate asthma, and it was 780.51 ± 263.93 cells/μL in patients suffering from severe asthma. Pairwise comparisons between all the groups revealed statistical significance ($P < 0.001$), while the comparison between moderate and severe asthma also showed high statistical significance ($P = 0.005$), thereby reiterating the effectiveness of blood eosinophils as a diagnostic tool even in the more advanced stages of the disease.

Table 2. Blood Eosinophil Count by Study Group (Mean ± SD, Median, and IQR).

Group	n	Mean ± SD (cells/μL)	Median (IQR)	P-value vs Control
Healthy Control	60	214.19 ± 47.48	210 (181–243)	—

Mild Asthma	25	323.35 ± 68.18	309 (286–364)	<0.001 ***
Moderate Asthma	20	598.52 ± 107.64	585 (544–654)	<0.001 ***
Severe Asthma	15	780.51 ± 263.93	810 (761–966)	<0.001 ***
All Asthma Combined	60	529.36 ± 240.40	535 (335–700)	<0.001 ***

**** P < 0.001 (Mann–Whitney U test). Moderate vs Severe: P = 0.005 (**). Kruskal–Wallis H across four groups: H = 82.74, P < 0.001. IQR: interquartile range.*

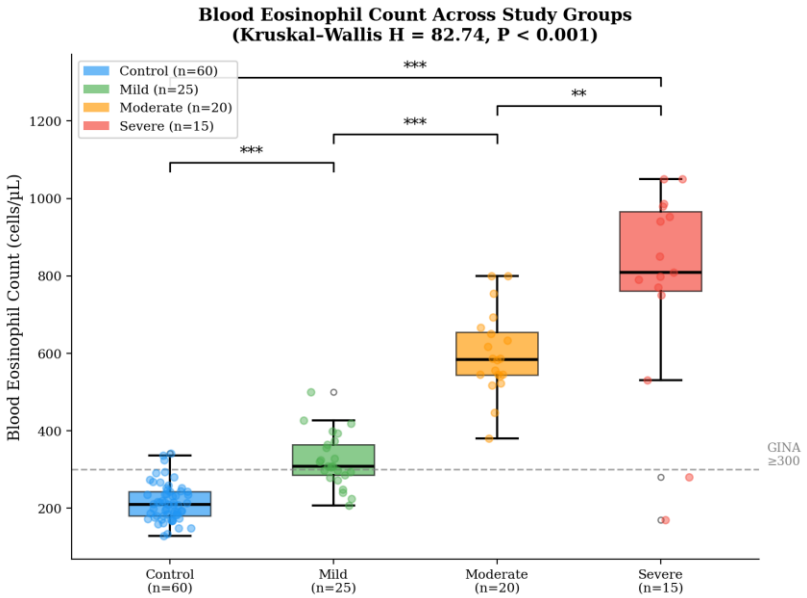

Figure 2. Blood eosinophil count across study groups. Box plots with overlaid individual data points (jitter). Dashed line: GINA threshold (≥300 cells/μL). *** P < 0.001 (Mann–Whitney U); ** P < 0.01 for moderate vs severe. Kruskal–Wallis H = 82.74, P < 0.001.

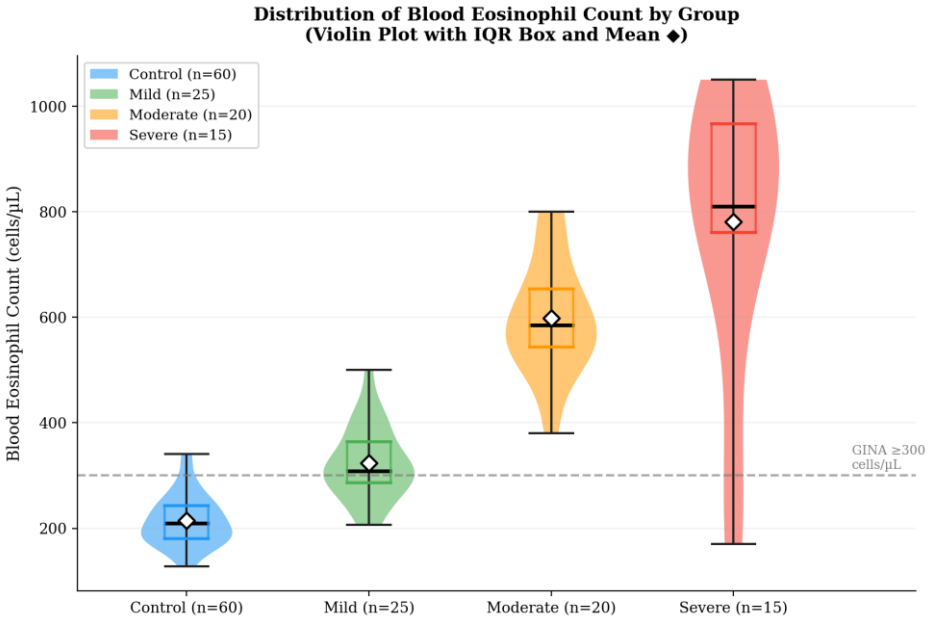

Figure 3. Violin plot showing the distribution of blood eosinophil count by study group. Width reflects data density; inner box marks the IQR; white diamond (◆) denotes the group mean; horizontal bar marks the median. Dashed line: GINA threshold (≥300 cells/μL).

3.3. Spirometric Parameters and Peripheral Oxygen Saturation

Table 3 and Figure 4 show all values were significantly different between the asthmatic patients and controls, and there was a progressive decline with increased severity of asthma. FEV1% predicted was significantly reduced in the asthmatic patients compared with the controls and decreased progressively with increased severity of the disease. FEV1% predicted in the controls was 90.54 ± 5.52%, whereas it was 65.91 ± 4.70% in mild, 47.60 ± 5.17% in moderate, and 29.49 ± 4.85% in severe asthmatic patients. FVC% also decreased progressively with increased severity of the disease. FVC% in the controls was 93.30 ± 7.55%. FVC% in mild, moderate, and severe asthmatic patients was 76.59 ± 3.18%, 61.88 ± 3.90%, and 47.92 ± 4.17%, respectively. FEV1/FVC ratio, which is an index of the obstructive ventilatory pattern seen in asthma, progressively decreased with increased severity of the disease. FEV1/FVC ratio in the controls was 0.79 ± 0.03, whereas it was 0.70 ± 0.02 in mild, 0.59 ± 0.03 in moderate, and 0.48 ± 0.04 in severe asthmatic patients. SpO2 levels progressively decreased with increased severity of the disease. SpO2 levels in the controls were 98.03 ± 0.56%. SpO2 levels in mild, moderate, and severe asthmatic patients were 96.65 ± 0.93%, 94.63 ± 1.04%, and 88.57 ± 1.95%, respectively. SpO2 levels in the severe asthmatic patients were compatible with clinically significant hypoxemia.

Table 3. Spirometric Parameters and SpO_2 by Study Group (Mean ± SD).

Parameter	Control (n=60)	Mild (n=25)	Moderate (n=20)	Severe (n=15)	P-value*
FEV_1% predicted	90.54 ± 5.52	65.91 ± 4.70	47.60 ± 5.17	29.49 ± 4.85	<0.001
FVC% predicted	93.30 ± 7.55	76.59 ± 3.18	61.88 ± 3.90	47.92 ± 4.17	<0.001
FEV_1/FVC ratio	0.79 ± 0.03	0.70 ± 0.02	0.59 ± 0.03	0.48 ± 0.04	<0.001
SpO_2 (%)	98.03 ± 0.56	96.65 ± 0.93	94.63 ± 1.04	88.57 ± 1.95	<0.001

** Kruskal–Wallis H test across four groups (FEV_1%: H=102.28; SpO_2: H=90.96). All pairwise Mann–Whitney U comparisons were statistically significant at P < 0.001.*

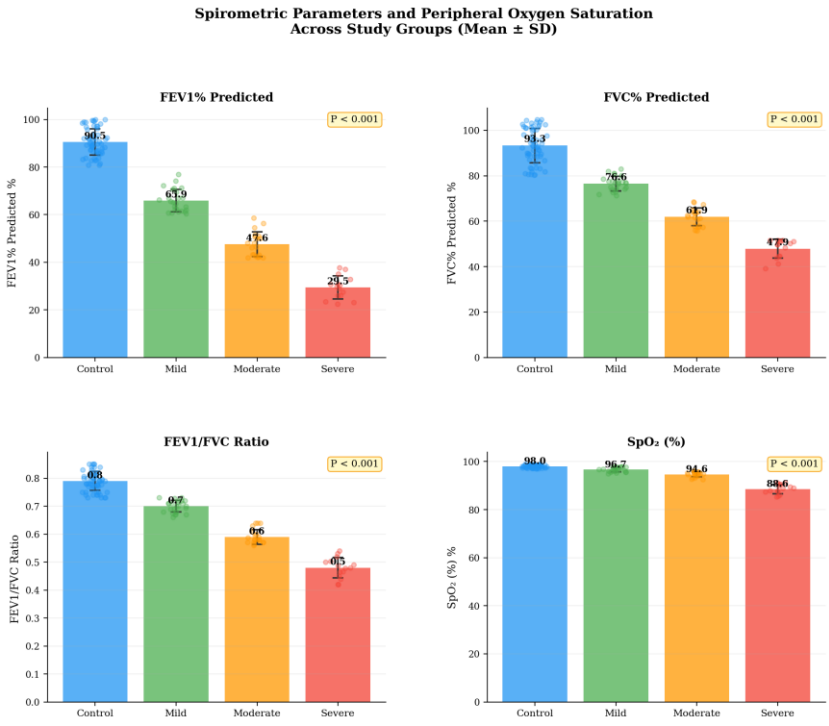

Figure 4. Spirometric parameters and peripheral oxygen saturation (SpO_2) across study groups. Bars represent group means ± SD with individual data points overlaid. (A) FEV_1% predicted; (B) FVC% predicted; (C) FEV_1/FVC ratio; (D) SpO_2 (%). All intergroup comparisons: P < 0.001.

3.4. Correlations Between Blood Eosinophil Count and Clinical Parameters

Spearman's rank correlation analysis was performed among the asthma population (n = 60) to investigate the relationships between BEC and various spirometric parameters, SpO2, disease duration, and frequency of exacerbation (Table 4; Figures 5 and 6).

Significant negative correlations of BEC with all pulmonary function parameters, including FEV1% (r = -0.637, P < 0.001), FVC% (r = -0.735, P < 0.001), and FEV1/FVC ratio (r = -0.740, P < 0.001), were observed. These correlations indicate that as the eosinophil count increases, the severity of airflow limitation and decreased lung volumes also increase. The strongest negative correlation was seen between BEC and FEV1/FVC ratio (r = -0.740).

A negative correlation between BEC and SpO2 (r = -0.664, P < 0.001) was also seen, indicating progressive desaturation as the eosinophil count rises. As per the severity gradient, BEC correlated positively with disease duration (r = 0.635, P < 0.001) and frequency of exacerbation (r = 0.711, P < 0.001), indicating the strongest positive correlations seen in the study population. A very strong positive correlation was seen between SpO2 and FEV1% (r = 0.906, P < 0.001), indicating the close physiological correlation between airflow limitation and desaturation as per the severity gradient.

Table 4. Spearman Rank Correlations Between Blood Eosinophil Count and Clinical/Functional Parameters in Asthmatic Patients (n = 60).

Parameter	Spearman r	P-value
FEV_1% predicted	−0.637	<0.001
FVC% predicted	−0.735	<0.001
FEV_1/FVC ratio	−0.740	<0.001
SpO_2 (%)	−0.664	<0.001
Disease Duration (years)	+0.635	<0.001
Exacerbations per Year	+0.711	<0.001
SpO_2 vs FEV_1% †	+0.906	<0.001

† SpO_2 vs FEV_1% correlation computed within the asthma group. Negative r: inverse relationship; Positive r: direct relationship.

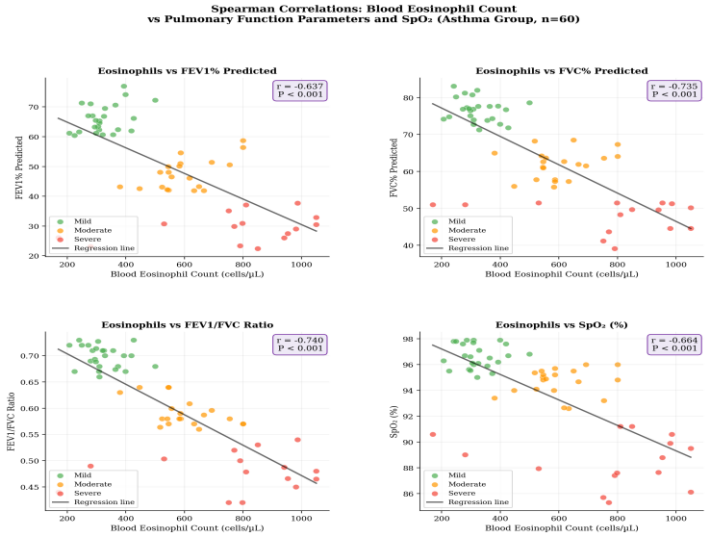

Figure 5. Spearman correlations between blood eosinophil count and respiratory parameters (asthma group, n = 60). Points colour-coded by severity. (A) Eosinophils vs FEV_1% (r = −0.637); (B) Eosinophils vs FVC% (r = −0.735); (C) Eosinophils vs FEV_1/FVC (r = −0.740); (D) Eosinophils vs SpO_2 (r = −0.664). All P < 0.001.

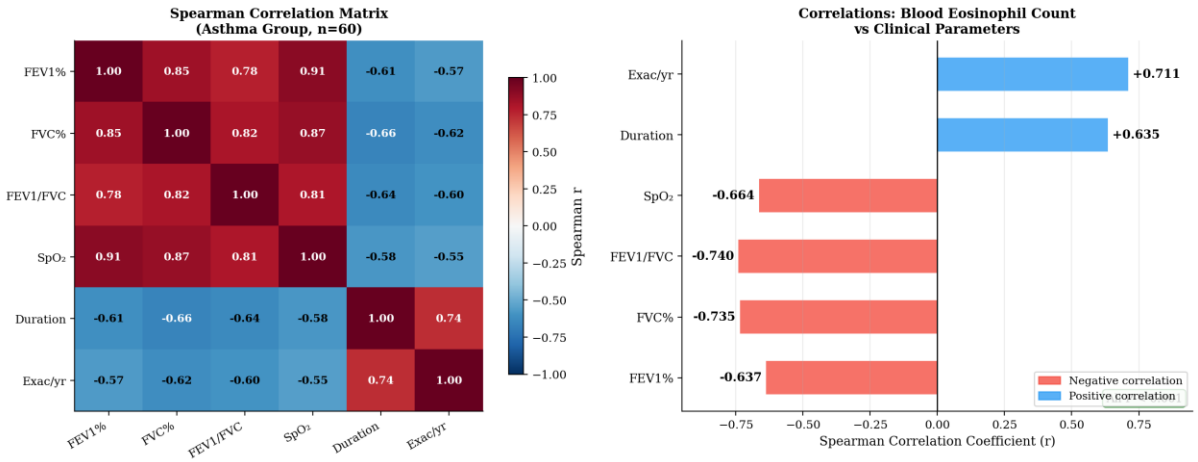

Figure 6. Spearman correlation analysis in the asthma group (n = 60). Left panel: correlation matrix heat-map; colour scale ranges from red (−1.0) to blue (+1.0). Right panel: bar chart of blood eosinophil count correlations with each clinical parameter (red bars: inverse; blue bars: positive). All correlations significant at P < 0.001.

3.5. Diagnostic Accuracy of Blood Eosinophil Count: ROC Curve Analysis

To determine the accuracy of the blood eosinophil count as a diagnostic tool to differentiate between healthy controls and asthmatic patients, the receiver operating characteristic (ROC) curve was analyzed (Table 5 & Figure 7). The results showed that the blood eosinophil count was highly accurate as a diagnostic tool to differentiate between healthy controls and asthmatic patients, with the area under the curve (AUC) being 0.948 (95% CI = 0.907-0.990; P < 0.001). Using the Youden index to determine the appropriate cutoff value of the blood eosinophil count to differentiate between healthy controls and asthmatic patients, the results showed that the appropriate value was >272 cells/μL with 90.0% sensitivity (54/60 asthmatic patients correctly identified) and 88.3% specificity (53/60 controls correctly identified). The positive predictive value was 88.5%, while the negative predictive value was 89.8%.

Table 5. ROC Curve Analysis of Blood Eosinophil Count for Diagnosis of Bronchial Asthma.

Diagnostic Parameter	Value
AUC (95% CI)	0.948 (0.907–0.990)
P-value	<0.001
Optimal Cutoff (Youden Index)	>272 cells/μL
Sensitivity	90.0% (54/60 asthmatic patients)
Specificity	88.3% (53/60 healthy controls)
Positive Predictive Value (PPV)	88.5%
Negative Predictive Value (NPV)	89.8%
True Positives / False Negatives	54 / 6
True Negatives / False Positives	53 / 7

AUC: area under the curve; CI: confidence interval; PPV: positive predictive value; NPV: negative predictive value. Optimal cutoff determined by Youden index (sensitivity + specificity − 1). Youden index at this cutoff: 0.783.

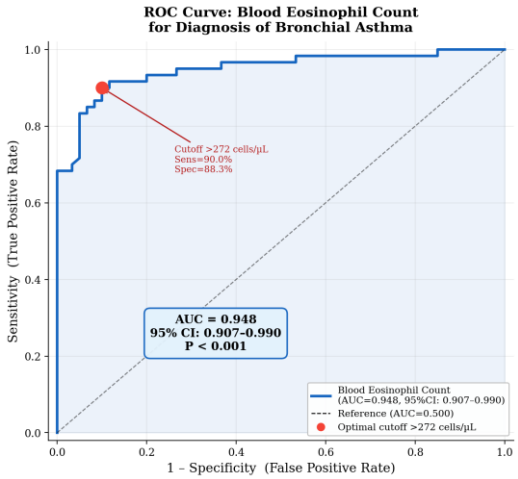

Figure 7. ROC curve for blood eosinophil count as a diagnostic biomarker for bronchial asthma. The red circle marks the Youden-optimal cutoff (>272 cells/μL). AUC = 0.948 (95% CI: 0.907–0.990; P < 0.001); sensitivity = 90.0%; specificity = 88.3%; PPV = 88.5%; NPV = 89.8%.

Discussion

The 2.5-fold increase in mean BEC levels in asthmatic patients compared to controls (529.36 vs 214.19 cells/μL; P < 0.001) may be explained by the continuous effect of IL-5 on eosinophilopoiesis in type 2 immune responses, which extends beyond exacerbation episodes [3, 4]. This level was also significantly higher in mild cases of asthma compared to controls (323.35 vs 214.19 cells/μL; P < 0.001). This observation is in line with Guida et al. [5], who confirmed the value of BEC in T2-high phenotypes. In addition to this comparison, BEC levels were found to increase with increasing severity of asthma according to the GINA classification: mild (323.35 ± 68.18), moderate (598.52 ± 107.64), and severe (780.51 ± 263.93 cells/μL; P = 0.005 for moderate-to-severe cases). This finding may be explained by an increase in IL-5 production and a decrease in apoptosis with disease severity [4]. In addition, this observation may also explain the utility of BEC levels in deciding on escalation to biologics according to the GINA guidelines [6]. This observation is in line with Moore et al. [8], where cluster analysis according to the Severe Asthma Research Programme (SARP) showed that BEC levels were a discriminating feature in eosinophilic phenotypes. In a study by Toledo-Pons et al. [9], it was reported that BEC levels measured once may not be of prognostic value in real-world cases.

The strong inverse correlations between BEC and FEV_1% (r = -0.637), FVC% (r = -0.735), and FEV_1/FVC (r = -0.740) may be explained by the eosinophil granule proteins MBP, ECP, and EDN, which cause damage to the airway epithelium, smooth muscle hyperresponsiveness, and subepithelial fibrosis, resulting in obstructive and restrictive lung function impairments [3, 4]. The strong correlations between BEC and FVC% and FEV_1/FVC may be explained by the post-hoc analyses by Papi et al. [7] on the TRIMARAN and TRIGGER trials, where the investigators demonstrated the association between persistent eosinophilic inflammation and airflow limitation, and Backman et al. [10], where the investigators demonstrated the independent association between blood eosinophilia and accelerated decline in FEV_1 over 18 years. The absence of correlations by Parambil et al. [11] may be explained by the immunosuppressive effect of steroids on BEC and airway inflammation in their patient cohort.

BEC correlated inversely with SpO_2 (r = -0.664; P < 0.001), and SpO_2 decreased from 98.03% in the control group to 88.57% in the severe asthma group. The pathogenesis may be explained by the ECP-induced mucus hypersecretion, epithelium shedding, and small airway constriction, resulting in impaired gas exchange [4, 7]. The very strong correlation between SpO_2 and FEV_1% (r = 0.906; P < 0.001) may be explained by the close physiological correlation between airflow limitation and hypoxemia, and the findings by Moore et al. [8] and Wagener et al. [12]. The BEC and SpO_2 correlations have not been demonstrated previously in the Iraqi population, and the effect of the inflammatory process in the severe subgroup, mediated by the obese state, should be further evaluated.

Complementary to the cross-sectional data, BEC correlated positively with disease duration (r = 0.635) and exacerbation frequency (r = 0.711; P < 0.001). Prolonged eosinophilic airway inflammation causes the accumulation of airway remodeling changes, including smooth muscle hypertrophy, subepithelial fibrosis, and goblet cell hyperplasia. This maintains the total number of eosinophils in the circulation [3, 4]. The correlation with exacerbation frequency is strongest, which is consistent with the dose-response relationship shown by Price et al. [13] in a UK population study of 130,248 patients and the meta-analysis by Mallah et al. [14], who calculated an odds ratio of 1.31; 95% CI: 1.16–1.49 for BEC ≥200 cells/μL. This supports the GINA-endorsed indication for the use of BEC as a predictor of exacerbation risk [6]. Toledo-Pons et al. [9] highlighted the importance of eosinophil variability rather than the actual cell count in predicting hospitalization.

In terms of diagnostic accuracy, BEC was found to be very accurate, with AUC = 0.948, 95% CI = 0.907–0.990. The Youden optimal cutoff for BEC was >272 cells/μL, with 90.0% sensitivity, 88.3% specificity, 88.5% positive predictive value, and 89.8% negative predictive value. The increased level of IL-5-driven eosinophil production and decreased eosinophil apoptosis in asthma ensure that BEC remains well separated from healthy controls [3, 4]. This cutoff value is very close to the GINA cutoff value of ≥300 cells/μL. The AUC for BEC compares well to the pooled AUC value of 0.80 reported by Korevaar et al. [15] in their systematic review, likely because of the very strict exclusion criteria used. In their study, Li et al. [16] reported a lower AUC value for BEC, 0.768, using bronchoprovocation as the reference standard, stating that BEC is not sufficient on its own for patients in the intermediate BEC group. This statement supports the use of BEC in conjunction with spirometry, not instead of spirometry. The higher mean age in the asthmatic patients (50.63 vs. 43.42 years; P = 0.012) is likely because the study design focused on adult-onset asthma in the Iraqi tertiary healthcare patient population. The higher percentage of women (56.7% vs. 55.0%) did not reach significance and would not be expected to have biased the study. The very high BMI in the severe subgroup (mean ± SD, 34.46 ± 4.55 kg/m²) should be considered, as leptin, tumour necrosis factor-α (TNF-α), and IL-6 produced by adipose tissue may contribute to eosinophilic inflammation and decreased bronchodilator response [17].

The limitations of the study should be noted. Firstly, the cross-sectional nature of the study does not allow the evaluation of the dynamics of changes in BEC over time or in response to treatment. Secondly, the spirometric parameters of FEV1% and FVC% share a physiological interdependence; the concurrent correlation of these two parameters with BEC should be considered with the potential for multicollinearity. Future work would benefit from the inclusion of multivariate regression modeling. Thirdly, the exclusion of sputum eosinophil cytology and fractional exhaled nitric oxide (FeNO) analysis would have provided direct evidence of eosinophilic airway inflammation. Fourthly, the study population was derived from a single center in Al-Diwaniyah Governorate; it would be important to validate the study's observations in a broader population, especially in the context of a primary care population. Future work should also include the evaluation of the dynamics of changes in BEC over time, the inclusion of sputum cytology, FeNO, and treatment outcomes in the context of the Iraqi asthma population.

Conclusion

Blood eosinophil count is a readily available, non-invasive, and cost-effective biomarker that demonstrates excellent diagnostic accuracy for bronchial asthma in the studied adult Iraqi population, with well-balanced sensitivity and specificity at the Youden-optimal cutoff. BEC exhibited a consistent progressive elevation across GINA severity subgroups and strong inverse correlations with FEV_1%, FVC%, FEV_1/FVC ratio, and SpO_2, as well as positive correlations with disease duration and exacerbation frequency. These findings collectively support the integration of blood eosinophil count into routine clinical assessment for asthma diagnosis, severity stratification, exacerbation risk prediction, and monitoring of therapeutic response, particularly in resource-limited healthcare settings where advanced biomarker platforms are not available.

Acknowledgements

The authors gratefully acknowledge the medical staff of the Department of Chest and Respiratory Diseases and the Allergy and Asthma Centre at Al-Diwaniyah Teaching Hospital for their assistance in patient recruitment and clinical data collection. Sincere appreciation is extended to all study participants for their voluntary contribution.

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