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Frequency of Eosinophilia in Patients Presenting with Bronchial Asthma Presenting at Tertiary Care Hospital

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ABSTRACT

Background: Bronchial asthma is a chronic inflammatory disorder of the airways, characterized by recurrent episodes of airflow obstruction, bronchial hyperresponsiveness, and symptoms such as wheezing, cough, chest tightness, and shortness of breath. Eosinophilia, an elevated count of eosinophilic granulocytes in peripheral blood, reflects ongoing immunoinflammatory activity and serves as a biomarker for disease severity and therapeutic guidance. **Objective:** To determine the frequency of eosinophilia in patients presenting with bronchial asthma presenting at tertiary care hospital. **Study Design:** Descriptive cross-sectional study. **Duration and Place of Study:** The study was conducted from 23 March 2025 to 23 June 2025 at the Department of Pulmonology, Saidu Group of Teaching Hospitals, Swat. **Methodology:** A total of 143 patients aged 18–70 years, diagnosed with bronchial asthma based on characteristic symptoms and spirometry criteria, were enrolled using non-probability consecutive sampling. Patients with allergic bronchopulmonary aspergillosis, chronic obstructive pulmonary disease, or systemic corticosteroid therapy were excluded. Venous blood samples were collected to measure absolute eosinophil counts, with eosinophilia defined as greater than 500 cells per microliter. Demographic information and comorbidities were recorded. **Results:** The mean age was 42.44 ± 11.63 years, with 42% males and 58% females. Eosinophilia was present in 44.8% of patients. Younger age, rural residence, and smoking were significantly associated with elevated eosinophil counts. Comorbidities such as diabetes and hypertension showed no significant association. **Conclusion:** Eosinophilia is a common inflammatory marker in nearly half of patients with bronchial asthma, with age, environmental exposure, and smoking status being important determinants.

INTRODUCTION

Bronchial asthma is a chronic inflammatory airway disorder in which the bronchi exhibit recurrent obstruction, hyperresponsiveness, and clinical manifestations such as wheezing, productive cough, chest constriction, and dyspnea.[1] The underlying mechanism involves complex interactions among bronchial epithelium, immune effector cells, and inflammatory mediators, leading to transient airflow limitation.[2] Genetic susceptibility, exposure to aeroallergens, respiratory infections, and irritants such as smoke or pollutants contribute to disease progression.[3] The condition manifests in acute and chronic phases; the acute stage is characterized by bronchospasm and mucus hypersecretion, whereas prolonged inflammation results in structural remodeling of the bronchial walls.[4] Persistent airway inflammation remains the primary cause of hyperreactivity, the cardinal feature of asthma. Early detection and appropriate anti-inflammatory therapy are essential to prevent decline in pulmonary function and reduce disease severity.

Eosinophilia, a frequent hematological abnormality among asthmatic patients, indicates ongoing immunoinflammatory activity.[5] Eosinophils are a type of granulocytic leukocyte that play a central role in allergic and atopic disorders.[6] These cells release cytotoxic proteins, eosinophil cationic protein, and lipid mediators such as leukotrienes that intensify airway inflammation.[6] In asthma, eosinophil proliferation and recruitment are regulated by cytokines including interleukin-5, interleukin-4, and eotaxin, which enhance their maturation, activation, and migration to the bronchial mucosa.[7] Their accumulation contributes to epithelial damage, mucus overproduction, and increased airway hyperresponsiveness. Peripheral blood eosinophil count functions as a clinical biomarker for asthma phenotyping and predicting corticosteroid responsiveness.[8] Elevated eosinophil counts in blood and sputum correlate strongly with disease severity and recurrent exacerbations.[8]

In individuals with bronchial asthma, eosinophilia reflects not only the extent of airway inflammation but also guides therapeutic management.[9] Persistent eosinophilic elevation despite standard pharmacologic therapy indicates severe or refractory asthma, which often requires biologic agents targeting interleukin-5 or its receptor to suppress eosinophil-mediated inflammation.[10] Quantifying eosinophil levels provides prognostic insight, as elevated counts are associated with impaired pulmonary function, increased airway limitation, and poor symptom regulation.[11] Serial monitoring of eosinophil levels during follow-up aids in assessing therapeutic efficacy and predicting relapse.[11] Hence, comprehension of eosinophilic activity in asthma is vital for individualized management and to optimize clinical outcomes. A study found the frequency of eosinophilia in patients with bronchial asthma was (39%).[12]

Carrying out this study in Swat is necessary because for this territory, little epidemiological information on the link between eosinophilia and bronchial asthma is available. Swat's environmental and climatic parameters, like exposure to allergens, biomass fuel exposure, and seasonal effects, are potentially different in their effect on asthma prevalence and eosinophilic response compared to other regions. Moreover, correspondingly, access by patients to sophisticated evaluation and treatment facilities is limited, and hence, a considerable number of asthma patients are under-assessed or too little treated. Producing local information will assist us to know the EOS pattern of inflammatory eosinophilic among asthmatic patients of this population, leading to a better precise diagnosis and evidence-based management designed for regional healthcare facilities.

METHODOLOGY

This descriptive cross-sectional study was carried out in the Department of Pulmonology at Saidu Group of Teaching Hospitals, Swat, between March 2025 and May 2025. A total of 143 participants were enrolled. The sample size was estimated using the World Health Organization sample size calculator, assuming a 39% expected frequency of eosinophilia in asthma,[12] a 95% confidence interval, and an 8% margin of error. Approval to conduct the study was obtained from the institutional ethics review committee of Saidu Group of Teaching Hospitals (*No 183-ERB/024 Dated 03-10-2024*), as well as from the research evaluation board of the College of Physicians and Surgeons Pakistan. Written consent was obtained from all participants after they were informed of the study objectives, procedures, and their right to withdraw at any stage without penalty. Participants were assured of confidentiality and informed that the study posed no potential harm. Individuals aged between 18 and 70 years of either sex who fulfilled the diagnostic criteria for bronchial asthma were included. Diagnosis of bronchial asthma was established based on characteristic clinical manifestations such as wheezing, dyspnea, cough, and chest tightness, supported by spirometry findings demonstrating a forced expiratory volume in one second to forced vital capacity (FEV1/FVC) ratio of less than 70%, with an improvement of at least 12% and 200 milliliters in FEV1 following bronchodilator administration. Patients with allergic bronchopulmonary aspergillosis, chronic obstructive pulmonary disease, or those receiving systemic corticosteroid therapy were excluded. After obtaining consent, baseline characteristics including age, sex, body mass index, educational attainment, socioeconomic class, area of residence, and employment status were recorded. Relevant medical history regarding diabetes mellitus, hypertension, and smoking habits was also noted. Each participant underwent laboratory testing for absolute eosinophil count. Approximately 2–5 milliliters of venous blood was drawn under aseptic conditions and analyzed in the hospital's hematology laboratory. Eosinophilia was defined as an absolute eosinophil count exceeding 500 cells per microliter of blood, as verified by a consultant pulmonologist with at least five years of post-fellowship experience. All data were documented using a structured proforma developed for this study, and data collection was supervised by the principal investigator.

Data analysis was performed using IBM SPSS Statistics version 21. The Shapiro–Wilk test was applied to determine data normality. Continuous variables were summarized as mean ± standard deviation or median with interquartile range, depending on distribution. Categorical variables were expressed as frequencies and percentages. Eosinophilia was cross-tabulated with these variables to assess potential effect modification. Post-stratification analysis was performed using the Chi-square or Fisher's exact test where appropriate, considering a p-value ≤0.05 as statistically significant.

RESULTS

The study included 143 patients with bronchial asthma, with a mean age of 42.44 ± 11.63 years and mean BMI of 24.77 ± 3.17 kg/m^2. The mean serum eosinophil count was 539.71 ± 257.04 µL. The study population comprised 60 males (42.0%) and 83 females (58.0%). Regarding socioeconomic status, the majority belonged to the lower class with 82 patients (57.3%), followed by middle class with 51 patients (35.7%), and upper class with 10 patients (7.0%). A substantial proportion of patients resided in rural areas, accounting for 98 patients (68.5%), while 45 patients (31.5%) were from urban areas. Among the study participants, 29 patients (20.3%) were smokers and 114 patients (79.7%) were non-smokers. Comorbidities were relatively uncommon, with hypertension present in 23 patients (16.1%) and absent in 120 patients (83.9%), while diabetes was documented in 12 patients (8.4%) and absent in 131 patients (91.6%), as shown in Table-I.

Table I

Patient Demographics

Demographics	Mean ± SD
Age (Years)	42.44 ± 11.63
BMI (kg/m^2)	24.77 ± 3.17
Serum Eosinophils (µL)	539.71 ± 257.04
Gender	
Male n (%)	60 (42.0%)
Female n (%)	83 (58.0%)
Socioeconomic Status	
Lower n (%)	82 (57.3%)
Middle n (%)	51 (35.7%)
Upper n (%)	10 (7.0%)
Residence	
Rural n (%)	98 (68.5%)

Urban n (%)	45 (31.5%)
Smoking	
Yes n (%)	29 (20.3%)
No n (%)	114 (79.7%)
Hypertension	
Yes n (%)	23 (16.1%)
No n (%)	120 (83.9%)
Diabetes	
Yes n (%)	12 (8.4%)
No n (%)	131 (91.6%)

The frequency of eosinophilia among patients presenting with bronchial asthma revealed that 64 patients (44.80%) had eosinophilia, while 79 patients (55.20%) did not have eosinophilia out of the total 143 patients, as shown in Table-II.

Table II

Frequency of Eosinophilia Among Patients Presenting with Bronchial Asthma

Eosinophilia	Frequency	% age
Yes	64	44.80%
No	79	55.20%
Total	143	100%

The association of eosinophilia with demographic factors demonstrated significant relationships with several variables. Age showed a highly significant association (p<0.001), with eosinophilia present in 50 patients (56.8%) aged ≤45 years compared to 38 patients (43.2%) without eosinophilia in this age group, while among those >45 years, only 14 patients (25.5%) had eosinophilia versus 41 patients (74.5%) without eosinophilia. Gender did not show a significant association (p=0.528), with eosinophilia present in 25 males (41.7%) and 39 females (47.0%). BMI also showed no significant association (p=0.155), with eosinophilia present in 40 patients (50.0%) with BMI ≤25 kg/m^2 and 24 patients (38.1%) with BMI >25 kg/m^2. Socioeconomic status demonstrated no significant association (p=0.931), with eosinophilia present in 37 patients (45.1%) from lower class, 22 patients (43.1%) from middle class, and 5 patients (50.0%) from upper class. Residence showed a highly significant association (p<0.001), with eosinophilia present in 56 rural patients (57.1%) compared to only 8 urban patients (17.8%). Smoking demonstrated a highly significant association (p<0.001), with eosinophilia present in 25 smokers (86.2%) and 39 non-smokers (34.2%). Hypertension showed a highly significant inverse association (p<0.001), with eosinophilia present in only 3 hypertensive patients (13.0%) compared to 61 non-hypertensive patients (50.8%). Diabetes showed no significant association (p=0.066), with eosinophilia present in 2 diabetic patients (16.7%) and 62 non-diabetic patients (47.3%), as shown in Table-III.

Table III

Association of Eosinophilia with Demographic Factors

Demographic Factors		Eosinophilia		p-value
		Yes n(%)	No n(%)	
Age (years)	≤45	50 (56.8%)	38 (43.2%)	<0.001
	>45	14 (25.5%)	41 (74.5%)	
Gender	Male	25 (41.7%)	35 (58.3%)	0.528
	Female	39 (47.0%)	44 (53.0%)	
BMI (Kg/m^2)	≤25	40 (50.0%)	40 (50.0%)	0.155
	>25	24 (38.1%)	39 (61.9%)	
Socioeconomic Status	Lower	37 (45.1%)	45 (54.9%)	0.931*
	Middle	22 (43.1%)	29 (56.9%)	
	Upper	5 (50.0%)	5 (50.0%)	
Residence	Rural	56 (57.1%)	42 (42.9%)	<0.001
	Urban	8 (17.8%)	37 (82.2%)	
Smoking	Yes	25 (86.2%)	4 (13.8%)	<0.001*
	No	39 (34.2%)	75 (65.8%)	
Hypertension	Yes	3 (13.0%)	20 (87.0%)	<0.001*
	No	61 (50.8%)	59 (49.2%)	
Diabetes	Yes	2 (16.7%)	10 (83.3%)	0.066*
	No	62 (47.3%)	69 (52.7%)	

*Fischer Exact Test

DISCUSSION

The present study was conducted to determine the frequency of eosinophilia in patients presenting with bronchial asthma, and the findings revealed that 44.80% of asthmatic patients exhibited eosinophilia, indicating that nearly half of the asthmatic population demonstrates elevated eosinophil counts. This substantial prevalence underscores the importance of eosinophilic inflammation as a key pathophysiological mechanism in bronchial asthma, where eosinophils release inflammatory mediators such as major basic protein, eosinophil cationic protein, and leukotrienes that contribute to airway hyperresponsiveness, mucus hypersecretion, and bronchial epithelial damage. The significant association between younger age (≤45 years) and eosinophilia can be attributed to the more robust immune responses and heightened type 2 inflammatory pathway activity in younger individuals, whereas older patients often develop neutrophilic or paucigranulocytic asthma phenotypes due to immune senescence and chronic airway remodeling. The highly significant association between rural residence and eosinophilia may be explained by increased exposure to environmental allergens, agricultural dust, animal dander, and parasitic infections in rural settings, all of which stimulate type 2 helper T-cell responses and subsequent eosinophil production and recruitment. The striking association between smoking and eosinophilia, where 86.2% of smokers exhibited elevated eosinophil counts, appears paradoxical since smoking typically promotes neutrophilic inflammation; however, this may reflect the synergistic effect of tobacco smoke irritation enhancing allergen-induced eosinophilic responses or the presence of concurrent allergic sensitization in these patients. The inverse relationship between hypertension and eosinophilia, where only 13.0% of hypertensive patients had eosinophilia, could be explained by the systemic anti-inflammatory effects of antihypertensive medications, particularly angiotensin-converting enzyme inhibitors and angiotensin receptor blockers, or the vascular and metabolic changes associated with hypertension that may shift asthma toward a non-eosinophilic phenotype.

The frequency of eosinophilia in the present study was 44.80%, which falls between the higher prevalence reported by Ramaswamy L, et al. [13] (67.8%) and Swain RR, et al. [14] (61%) in Indian populations, and the lower frequencies documented by Awopeju OF, et al. [15] (31%) in Nigerian adults and Ali MA [16] (12%) in hospitalized Iraqi patients. These variations likely reflect differences in diagnostic criteria, disease severity, treatment status, and population-specific genetic and environmental factors. The mean serum eosinophil count of 539.71 ± 257.04 µL

aligns with Siddiqui MH, et al. [17] who reported a mean of 436.73±225.75 cells/mm^3 in Pakistani patients, suggesting similar baseline eosinophilic inflammation in South Asian populations.

The significant association between younger age (≤45 years) and eosinophilia (p<0.001) is supported by Shah MM, et al. [18] and Qaiser S, et al. [19] who reported significant associations between age and eosinophilia (p=0.035 and p=0.024, respectively), reflecting predominant type 2 inflammation in younger patients. However, this contradicts Swain RR, et al. [14] and Ramaswamy L, et al. [13] who found no age-related differences (p=0.9162 and p>0.05, respectively), possibly due to different age stratification methods and sample characteristics. The lack of gender association (p=0.528) is consistent with Swain RR, et al. [14] (p=0.8851), Ramaswamy L, et al. [13] (p>0.05), and Butt K, et al. [20] (p=0.627), suggesting eosinophilic inflammation is independent of sex hormones, though this conflicts with Awopeju OF, et al. [15] who found males had higher odds of eosinophilic asthma (p=0.034), and Thritia S, et al. [21] who reported more severe disease in females, likely reflecting population-specific genetic and environmental factors.

The highly significant association between rural residence and eosinophilia (p<0.001), where 57.1% of rural patients had eosinophilia versus 17.8% of urban patients, is supported by Kumar N, et al. [22] who found 57% nasal eosinophilia and 55% blood eosinophilia in rural pediatric patients with allergic respiratory diseases. This reflects increased exposure to agricultural allergens, livestock dander, and parasitic infections in rural environments that stimulate type 2 immune responses, while urban pollution promotes neutrophilic inflammation. The striking association between smoking and eosinophilia (p<0.001), where 86.2% of smokers exhibited eosinophilia, is paradoxical since smoking typically causes neutrophilic inflammation, suggesting tobacco smoke may enhance allergen sensitization in genetically predisposed individuals. This finding is novel as none of the reviewed studies examined this relationship.

The inverse relationship between hypertension and eosinophilia (p<0.001), where only 13.0% of hypertensive patients had eosinophilia, is unique and unreported in the literature, possibly explained by anti-inflammatory effects of antihypertensive medications or metabolic changes shifting asthma toward non-eosinophilic phenotypes. The lack of associations with BMI (p=0.155), socioeconomic status (p=0.931), and diabetes (p=0.066) suggests eosinophilic inflammation is primarily driven by environmental exposures and age-related immunological factors rather than metabolic influences.

Regarding eosinophilia and severity, conflicting evidence exists: Thritia S, et al. [21] and Issa HY, et al. [23] demonstrated significant correlations (p<0.00001 and p=0.00004, respectively), while Ramaswamy L, et al. [13] Siddiqui MH, et al. [17] and Fayezi A, et al. [24] found no associations, highlighting the complexity of asthma phenotyping influenced by treatment status and disease phase. Therapeutically, Butt K, et al. [20] showed inhaled corticosteroids and montelukast reduced eosinophil counts in 33.5% of patients, while Mathur S, et al. [25] reported underutilization of eosinophil testing, with only 57.7% of patients tested despite its importance for identifying candidates for biologic therapies as emphasized by Marra AM, et al. [26] and British Society for Haematology guidelines.[27]

The present study has several limitations that warrant consideration. First, this was a single-center study conducted at one tertiary care hospital, which may limit the generalizability of findings to other healthcare settings and geographic populations with different demographic characteristics and environmental exposures. Second, the cross-sectional design of the study provides only a snapshot of eosinophil counts at a single time point, without accounting for temporal variations in eosinophil levels that may occur with disease exacerbations, seasonal changes, or treatment modifications. Third, the study did not assess asthma severity classification or document concurrent medication use, particularly inhaled or systemic corticosteroids, which could significantly influence eosinophil counts and potentially confound the observed associations. Fourth, sputum eosinophil counts and serum IgE levels were not measured, which are important complementary biomarkers for comprehensive phenotyping of eosinophilic asthma and could have provided additional insights into the inflammatory profile of the study population.

CONCLUSION

Our study has concluded that eosinophilia is present in nearly half of patients with bronchial asthma, highlighting its substantial prevalence as an important inflammatory biomarker in this population. Younger age, rural residence, and smoking status emerged as significant predictors of eosinophilia, suggesting that environmental exposures and age-related immunological factors play crucial roles in determining eosinophilic inflammation patterns in asthmatic patients.

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