ORIGINAL ARTICLE

Prevalence of peripheral Eosinophilia in familial allergic rhinitis

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Aim: This study aims to assess the prevalence of peripheral eosinophilia in patients with allergic rhinitis (AR) and determine its association with a positive family history. **Background:** AR is a chronic, immunoglobulin E-mediated condition that significantly affects the quality of life. Eosinophils are key effector cells involved in the allergic inflammatory cascade. Although peripheral eosinophilia is commonly observed in AR, its prevalence in patients with familial AR has not been thoroughly investigated. Understanding this association could support early identification of at-risk individuals and guide more tailored therapeutic strategies. **Observation and Results:** A cross-sectional observational study was conducted in a tertiary care hospital over 1 year. Seventy-eight patients aged 18–60 years with clinically diagnosed AR (score for AR ≥7) were included in this study. Peripheral blood samples were collected, and absolute eosinophil count (AEC) was measured. Eosinophilia was defined as AEC >350 cells/mm^3. A positive family history of AR in first-degree relatives was recorded. Results showed that 64.1% of patients exhibited peripheral eosinophilia, with 25.6% showing marked eosinophilia (>500 cells/mm^3). Among patients with a familial history of AR, 79.2% demonstrated eosinophilia, significantly higher than the 33.3% observed in those without a family history (P = 0.002). **Conclusion:** Peripheral eosinophilia is significantly more prevalent among AR patients with a positive family history, indicating a strong hereditary component in eosinophilic response. Clinical Significance: Eosinophil count, when combined with family history, can serve as a valuable and accessible tool for early identification, risk stratification, and personalized management of AR.

KEY WORDS: Absolute eosinophil count, allergic rhinitis, atopy, family history, genetic predisposition, nasal allergy, peripheral eosinophilia, score for allergic rhinitis

INTRODUCTION

Allergic rhinitis (AR) is a prevalent, chronic, immunoglobulin E (IgE)-mediated inflammatory condition of the nasal mucosa characterized by sneezing, nasal congestion, rhinorrhea, and nasal itching. It significantly impairs the quality of life and poses a growing public health concern due to its high prevalence and frequent underdiagnosis. Among the immunological hallmarks of AR, eosinophilia – a condition marked by elevated eosinophil counts in the blood or nasal secretions – has emerged as a reliable biomarker of allergic inflammation, particularly in atopic individuals.[1,2]

Eosinophils play a pivotal role in the pathogenesis of AR by releasing cytotoxic granules, cytokines, and chemokines that sustain and amplify mucosal inflammation.[3] Peripheral blood eosinophilia, defined typically as an absolute eosinophil count (AEC) exceeding 350 cells/mm^3, has been associated with disease severity, symptom persistence, and comorbidities such

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as asthma and sinusitis.[4] In the clinical setting, assessing eosinophil levels provides additional insight into disease activity and therapeutic response.

Family history remains one of the strongest predictors for the development of AR. Genetic predisposition, especially in first-degree relatives, is thought to influence immune system reactivity, including eosinophil proliferation and IgE-mediated hypersensitivity.[5] However, few studies have systematically examined the direct correlation between familial AR and the presence of peripheral eosinophilia.

MATERIALS AND METHODS

This was a cross-sectional, hospital-based observational study conducted over 1 year (August 2023–July 2024) in the Department of Otorhinolaryngology at a tertiary care hospital in Bareilly, Uttar Pradesh, India. The study included patients aged 18–60 years presenting with symptoms suggestive of AR. Inclusion criteria comprised a positive score for AR (SFAR ≥7) and documented family history of AR in first-degree relatives. Patients with known autoimmune diseases, parasitic infections, recent corticosteroid or antihistamine use, or chronic systemic illness were excluded from the study.

After obtaining informed written consent and ethical clearance, participants underwent detailed clinical evaluation, including symptom scoring and personal and familial history documentation. Peripheral blood samples were collected to determine total leukocyte count and differential leukocyte count. AEC was calculated, with eosinophilia defined as AEC >350 cells/mm^3.

Data were entered and analyzed using Statistical Package for the Social Sciences version 23.0. Descriptive statistics (mean, standard deviation, frequency, percentage) were used for summarization. Chi-square test and independent t-test were used to assess associations between peripheral eosinophilia and familial history. A $P < 0.05$ was considered statistically significant, also approved by the ethical committee-IEC/RMCH/58/2023/APR.

RESULTS

Demographic and Clinical Profile

The mean age of participants was 33.64 years in Group A and 28.69 years in Group B. Males were predominant in Group A, whereas females were more common in Group B. The duration of illness in most patients was <6 months. Winter was the most frequently reported season for symptom exacerbation. Sneezing emerged as the most prevalent symptom, followed by runny nose and nasal congestion, as shown in Table 1.

Despite demographic variations, the SFAR scores were comparable across groups (9.21 vs. 9.18), confirming equivalent allergic burden at baseline.

Symptom Progression and Treatment Efficacy

The total nasal symptom score (TNSS) improved significantly over the 4 weeks in both groups, with a more pronounced reduction in Group A. At baseline, TNSS scores were comparable (11.38 vs. 10.62). By the 4th week, Group A showed a mean score of 1.46 versus 3.90 in Group B ($P < 0.001$), as shown in Table 2.

Peripheral Eosinophilia

Approximately 64.1% of patients showed eosinophilia (AEC >350 cells/mm^3), while 25.6% had marked eosinophilia (>500 cells/mm^3), suggesting a substantial eosinophilic response among patients with AR, as shown in Table 3.

Familial History of AR with Eosinophilia

Eosinophilia was significantly more prevalent in patients with a positive familial history of AR (P = 0.002), indicating a potential genetic or inherited immunologic predisposition [Table 4].

DISCUSSION

The present study highlights the significant prevalence of peripheral eosinophilia among individuals with AR, particularly

Table 1: Demographic and clinical profile

Variable	Group A (Fexofenadine+ Montelukast)	Group B (Levocetirizine+ Montelukast)
Mean age (years) (%)	33.64±13.94	28.69±10.12
Gender: Male (%)	61.5	41.0
Gender: Female (%)	38.5	59.0
Duration of illness <6 months (%)	48.7	53.8
Winter season presentation (%)	43.6	43.6
Most common symptom: Sneezing (%)	53.8	59.0

Table 2: Total nasal symptom score over time

Time point	Group A (Fexofenadine+ Montelukast)	Group B (Levocetirizine+ Montelukast)
Baseline	11.38±1.68	10.62±2.03
2nd Week	5.05±1.90	6.46±2.16
4th Week	1.46±1.14	3.90±1.92

Table 3: Distribution of peripheral eosinophilia in study participants (*n*=78)

Eosinophil count (cells/mm^3)	Frequency	Percentage
<350	28	35.9
350–500	30	38.5
>500	20	25.6
Total	78	100

Table 4: Correlation of familial history of AR with eosinophilia (*n*=78)

Family history of AR	Peripheral eosinophilia present (AEC >350)	Peripheral eosinophilia absent (AEC ≤350)	Total (*n*)	*P*-value
Present (*n*=48)	38 (79.2)	10 (20.8)	48	0.002*
Absent (*n*=30)	10 (33.3)	20 (66.7)	30	
Total	48	30	78	

AEC: Absolute eosinophil count, AR: Allergic rhinitis

those with a documented familial history. Approximately 64.1% of the study population exhibited eosinophilia (AEC >350 cells/mm^3), and 79.2% of patients with a positive family history also showed elevated eosinophil counts, indicating a strong association between hereditary predisposition and eosinophilic immune response.

These findings are in line with the known pathophysiological role of eosinophils in allergic diseases. Eosinophils, on activation, release cytotoxic granules and inflammatory mediators that contribute to tissue remodeling, epithelial damage, and sustained inflammation in the nasal mucosa, particularly during allergen exposure phases.[6] Mucosal eosinophil infiltration has previously been observed in various atopic conditions, suggesting a common immunologic axis driving allergic manifestations.[7] This eosinophilic dominance is thought to be regulated by cytokines such as interleukin-5 and transcriptional mediators like STAT6, both of which play a central role in the Th2-mediated immune response.[8,9]

Familial clustering of eosinophilic diseases may be explained by shared genetic and environmental factors, which contribute to immune dysregulation, epithelial barrier defects, and heightened sensitization to allergens.[10] Peripheral eosinophilia has also been implicated in the severity and chronicity of AR, correlating with more persistent and treatment-resistant symptoms. Seasonal exacerbations observed in the study, especially during winter, may reflect increased indoor allergen exposure and reduced mucociliary clearance, both of which intensify eosinophilic inflammation.[11-13]

Interestingly, recent updates in global guidelines on AR emphasize the relevance of inflammatory biomarkers such as eosinophil counts for sub-classification and individualized treatment strategies.[12,14] Blood eosinophil measurements, while nonspecific, may serve as surrogate indicators for disease burden and systemic allergic activation.[8] Moreover, research has shown distinct clinical traits in seasonal versus perennial AR patients based on their eosinophilic profiles, suggesting that peripheral eosinophilia might aid in phenotyping and prognostication.[15]

CONCLUSION

This study demonstrates a strong association between peripheral eosinophilia and familial AR. A significant proportion of patients with a positive family history exhibited elevated eosinophil counts, suggesting a genetic or immunologic predisposition to eosinophilic inflammation in AR. These findings emphasize the potential utility of eosinophil counts as a supplementary marker for identifying at-risk individuals, particularly those with a known familial tendency.

Given the role of eosinophils in perpetuating allergic inflammation and symptom severity, early recognition of peripheral eosinophilia may aid in optimizing treatment strategies, especially in resource-limited settings where access to advanced immunologic testing is constrained. Incorporating eosinophil count evaluation into routine clinical assessment may also facilitate stratified care and better disease control.

Clinical Significance

Peripheral eosinophil count serves as a simple and cost-effective biomarker for identifying AR patients with a strong familial predisposition. Early detection of eosinophilia in such individuals can guide more targeted anti-inflammatory therapy and improve clinical outcomes. Incorporating eosinophil assessment alongside family history enhances diagnostic precision. This approach supports stratified management, enabling better symptom control, and personalized care. The study underscores the clinical utility of eosinophil monitoring in routine allergy evaluations.

REFERENCES

1. Durrani SR, Mukkada VA, Guilbert TW. Eosinophilic esophagitis: An important comorbid condition of asthma? Clin Rev Allergy Immunol 2018;55:56-64.
2. El-Shazly AE, Henket M, Lefebvre PP, Louis R. 2B4 (CD244) is involved in eosinophil adhesion and chemotaxis, and its surface expression is increased in allergic rhinitis after challenge. Int J Immunopathol Pharmacol 2011;24:949-60.
3. Borres MP, Björkstén B. Peripheral blood eosinophils and IL-4 in infancy in relation to the appearance of allergic disease during the first 6 years of life. Pediatr Allergy Immunol 2004;15:216-20.
4. Jensen SK, Melgaard ME, Pedersen CT, Yang L, Vahman N, Thyssen JP, *et al*. Limited clinical role of blood eosinophil levels in early life atopic disease: A mother-child cohort study. Pediatr Allergy Immunol 2023;34:e14050.
5. Letner D, Farris A, Khalili H, Garber J. Pollen-food allergy syndrome is a common allergic comorbidity in adults with eosinophilic esophagitis. Dis Esophagus 2018;31:1-8
6. D'Arienzo A, Manguso F, Astarita C, D'Armiento FP, Scarpa R, Gargano D, *et al*. Allergy and mucosal eosinophil infiltrate in ulcerative colitis. Scand J Gastroenterol 2000;35:624-31.
7. Zou YX, Zhang J, Ma C, Li J, Zai J, Guo YS. Clinical efficacy

of montelukast sodium in treating infantile wheezing. Eur Rev Med Pharmacol Sci 2014;18:775-80.
8. El-Gamal Y. Churg-strauss syndrome in the pediatric age group. World Allergy Organ J 2008;1:34-40.
9. Doherty TA, Khorram N, Chang JE, Kim HK, Rosenthal P, Croft M, *et al*. STAT6 regulates natural helper cell proliferation during lung inflammation initiated by alternaria. Am J Physiol Lung Cell Mol Physiol 2012;303:L577-88.
10. Rane SR, Kadgi NV, Agrawal SA, Kavatkar AN. Nasal discharge cytology an important diagnostic method for allergic fungal sinusitis: Report of three cases. J Clin Diagn Res 2014;8:121-2.
11. Guillevin L, Guittard T, Bletry O, Godeau P, Rosenthal P. Systemic necrotizing angiitis with asthma: Causes and precipitating factors in 43 cases. Lung 1987;165:165-72.
12. Bousquet J, Anto JM, Bachert C, Scadding GK, Togias A, Baiardini I, *et al*. Allergic rhinitis. Nat Rev Dis Primers 2020;6:95.
13. De Ru JA, Bayoumy AB. Intramuscular corticosteroid injections should be an option under the policy level recommendation in the international consensus statement on allergic rhinitis. Int Forum Allergy Rhinol 2023;13:18348-40.
14. Xu X, Li J, Zhang X, Xi L, Gao Y, Li X, *et al*. Blood and local eosinophil levels in chronic rhinitis: Observations during seasonal allergen exposure and non-exposure. World Allergy Organ J 2024;17:100930.
15. Wang K, Zhang Y, Wan L, Li J, Wang C, Zhang L. Comparison of clinical traits for seasonal and perennial allergic rhinitis during allergen exposure. Allergy Asthma Proc 2024;45: 173-9.