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Eosinophil-Based Hematological Indices Differentiate Allergic Rhinitis Phenotypes in Children

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ABSTRACT

Objective: Allergic rhinitis (AR) is a common chronic inflammatory disease of childhood that exhibits distinct clinical phenotypes. The aim of this study was to compare the distribution of hematological and inflammatory parameters among seasonal, perennial, and mixed phenotypes in pediatric patients with AR and to evaluate the role of hematological indices in phenotype discrimination.

Materials and Methods: This retrospective, cross-sectional study included 124 patients diagnosed with allergic rhinitis. Patients were classified as having perennial, seasonal, or mixed allergic rhinitis. Complete blood count parameters obtained during the non-pollen season (December–February) and derived inflammatory indices were analyzed in all patients.

Results: Of the study population, 65.3% were male. Asthma comorbidity was significantly more frequent in the mixed allergic rhinitis group ($p<0.001$). Absolute eosinophil count, eosinophil-to-lymphocyte ratio (ELR), and eosinophil-to-neutrophil ratio (ENR) were significantly higher in the mixed phenotype compared with the perennial and seasonal groups (all $p<0.01$). In multivariable logistic regression analysis, absolute eosinophil count (per 100 cells/μL increase; odds ratio [OR]=1.54, 95% confidence interval [CI]: 1.21–1.97; $p<0.001$) and asthma comorbidity (OR=2.19, 95% CI: 1.01–4.75; $p=0.047$) were identified as independent predictors of non-seasonal allergic rhinitis (NSAR). Receiver operating characteristic (ROC) analysis demonstrated that eosinophil count had moderate discriminatory performance for differentiating NSAR from seasonal allergic rhinitis (area under the curve [AUC]=0.692, 95% CI: 0.600–0.785). An optimal cutoff value of ≥195 cells/μL yielded a sensitivity of 77.8% and a specificity of 54.3%.

Conclusion: In pediatric allergic rhinitis, the mixed phenotype is associated with a more pronounced systemic eosinophilic inflammatory response. Absolute eosinophil count and asthma comorbidity emerge as independent predictors of non-seasonal allergic rhinitis. These readily available parameters from routine clinical practice may serve as complementary biomarkers for the evaluation of allergic rhinitis phenotypes.

Keywords: Allergic rhinitis, child, eosinophils, inflammation, phenotype, asthma

INTRODUCTION

Allergic rhinitis (AR) is a common chronic upper airway disease in childhood, characterized by immunoglobulin E (IgE)–mediated inflammation of the nasal mucosa. AR typically presents with symptoms such as nasal discharge, nasal congestion, sneezing, and nasal pruritus. Owing to its adverse effects on quality of life, school performance, and the presence of comorbid respiratory conditions, AR is considered a significant public health problem (1,2).

According to the ARIA (Allergic Rhinitis and its Impact on Asthma) guidelines, allergic rhinitis is classified into seasonal, perennial, and mixed phenotypes based on symptom duration and patterns of allergen exposure (3). These phenotypes exhibit heterogeneity not only in their clinical course but also in the underlying immunological mechanisms and inflammatory profiles. In particular, mixed allergic rhinitis, characterized by both seasonal and perennial allergen exposure, has been suggested to be associated with a more complex and intense inflammatory response (4,5).

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In recent years, the role of type 2 inflammation in the pathophysiology of allergic rhinitis has been increasingly elucidated, with eosinophils, T helper 2 (Th2) cells, and related cytokines shown to be closely associated with disease severity and the presence of comorbidities (6,7). In this context, routinely available complete blood count parameters and inflammatory indices derived from these parameters have attracted growing interest as potential biomarkers for distinguishing disease phenotypes in clinical practice (8).

Composite inflammatory indices, such as the eosinophil-to-lymphocyte ratio (ELR), platelet-to-lymphocyte ratio (PLR), and eosinophil-to-neutrophil ratio (ENR), have been reported to reflect the severity of systemic inflammation in allergic diseases and to be associated with clinical phenotypes (8-11). However, data regarding the discriminatory value of these indices among allergic rhinitis phenotypes remain limited, and evidence in the pediatric population is particularly scarce.

Asthma, the most common comorbidity associated with allergic rhinitis, increases the inflammatory burden of the disease and renders its clinical course more complex. Within the framework of the "united airway disease" concept, the upper and lower airways are considered to be affected by shared inflammatory mechanisms (12). Therefore, taking asthma comorbidity into account in the evaluation of allergic rhinitis phenotypes is of substantial clinical importance.

The aim of this study was to compare the distribution of hematological and inflammatory parameters among perennial, seasonal, and mixed phenotypes in pediatric patients with allergic rhinitis; to evaluate the potential role of eosinophil-based indices in phenotype discrimination; and to identify independent predictors of non-seasonal allergic rhinitis. In addition, the discriminatory performance of eosinophil counts was assessed to explore their clinical applicability.

MATERIALS and METHODS

This study had a retrospective, cross-sectional design. A total of 124 patients diagnosed with allergic rhinitis who were followed at a tertiary pediatric allergy and immunology outpatient clinic during the December–February periods between January 2019 and November 2025 were included in the study. The diagnosis and classification of allergic rhinitis were established in accordance with the Allergic Rhinitis and its Impact on Asthma (ARIA) guidelines, based on clinical history, physical examination findings, and evidence of allergen sensitization (13). Patients were classified into three phenotypic groups according to both allergen sensitization patterns and symptom characteristics. Seasonal allergic rhinitis was defined as the presence of rhinitis symptoms occurring predominantly during pollen seasons in patients sensitized to seasonal aeroallergens (grass, tree, or weed pollens). Perennial allergic rhinitis was defined as persistent year-round symptoms associated with sensitization to perennial indoor allergens such as house dust mite, mold fungi, or animal dander. Mixed allergic rhinitis was defined as the coexistence of both seasonal and perennial symptom patterns in patients sensitized to both seasonal and perennial allergens. Asthma diagnosis and severity were determined according to the Global Initiative for Asthma (GINA) guidelines. Asthma comorbidity was recorded according to documented clinical diagnoses in patient medical records. Patients with incomplete clinical or laboratory data were excluded from the study.

Allergen sensitization was evaluated based on the results of skin prick testing. Skin prick tests were performed using a standardized technique, and a test result was considered positive when the wheal diameter was at least 3 mm greater than that of the negative control (13). The allergens tested included grass pollen, tree pollen, weed pollen, house dust mite, mold fungi, and animal epithelia (cat and dog). Given that patients could be sensitized to more than one allergen, sensitization profiles were evaluated separately for each group. Complete blood count (CBC) parameters for all patients were retrospectively obtained from medical records during the non-pollen season (December–February). Only CBC results obtained during clinically stable visits without fever, acute respiratory symptoms, or documented infectious disease were included in the analysis. The hematological parameters analyzed included absolute eosinophil, lymphocyte, neutrophil, and platelet counts, as well as mean platelet volume. Using these parameters, the platelet-to-lymphocyte ratio (PLR), eosinophil-to-lymphocyte ratio (ELR), monocyte-to-lymphocyte ratio (MLR), monocyte-to-eosinophil ratio (MER), eosinophil-to-neutrophil ratio (ENR), and the systemic inflammation response index (SIRI) were calculated. The systemic inflammation response index (SIRI) is a novel inflammatory marker derived from peripheral blood cell counts and is calculated as neutrophil count × monocyte count / lymphocyte count. Venous blood

samples were collected in ethylenediaminetetraacetic acid (EDTA)–anticoagulated tubes as part of routine clinical evaluation. Differential white blood cell counts were determined using an automated hematology analyzer (Beckman Coulter LH 780, Miami, FL, USA).

Statistical Analysis

Statistical analysis were performed using the R statistical software within the RStudio integrated development environment (RStudio version 2026.01.0; Posit Software, PBC, Boston, MA, USA). Continuous variables were assessed for normality using visual inspection of histograms and the Shapiro–Wilk test. As most laboratory parameters did not follow a normal distribution, continuous variables are presented as median and interquartile range (IQR), while categorical variables are reported as counts and percentages. All statistical tests were two-tailed, and a p value <0.05 was considered statistically significant. Comparisons of continuous laboratory parameters among the three allergic rhinitis phenotypes (perennial, seasonal, and mixed) were performed using the Kruskal–Wallis test. For variables demonstrating statistically significant differences, post hoc pairwise comparisons were conducted using Dunn's test with Bonferroni correction to adjust for multiple testing. Although composite inflammatory indices such as the eosinophil-to-lymphocyte ratio (ELR) and platelet-to-lymphocyte ratio (PLR) showed statistically significant differences among allergic rhinitis phenotypes in univariate analyses, these indices were not included in the multivariable logistic regression model. This decision was based on concerns regarding substantial collinearity with their constituent components—particularly absolute eosinophil and lymphocyte counts—as well as the limited number of events in certain subgroups, which could compromise model stability. To minimize multicollinearity and reduce the risk of overfitting, the final multivariable model included only absolute eosinophil count and clinically relevant covariates. Although the Hosmer–Lemeshow test indicated some lack of calibration, the model was retained due to its clinical interpretability and statistically significant global fit. For logistic regression and ROC analyses, the perennial and mixed allergic rhinitis phenotypes were combined and defined as non-seasonal allergic rhinitis (NSAR), as both phenotypes are characterized by year-round allergen exposure and persistent inflammatory burden. The discriminatory performance of absolute eosinophil count in differentiating NSAR from seasonal allergic rhinitis was evaluated using ROC curve analysis. The area under the curve (AUC) and corresponding 95% confidence intervals were calculated, and sensitivity and specificity were assessed across a range of cutoff values.

RESULTS

A total of 124 patients were included in the study, of whom 81 (65.3%) were male. Patients were categorized into three groups: perennial (n=19), seasonal (n=70), and mixed (n=35) allergic rhinitis. When age distribution was evaluated across groups, the median age was 10.0 years in the perennial group, 12.75 years in the seasonal group, and 12.0 years in the mixed group (Table I). No statistically significant difference in age was observed among the three groups (Kruskal–Wallis test, p=0.541). Sex distribution was also comparable among the groups (p=0.914).

Although no significant differences were observed among the groups with respect to demographic characteristics, marked differences were identified in clinical features. The distribution of asthma comorbidity differed significantly among the groups (p<0.001). Asthma was most frequently observed in the mixed allergic rhinitis group

Table I: Demographic characteristics of the perennial, seasonal, and mixed allergic rhinitis groups

Variable	Perennial (n=19)	Seasonal (n=70)	Mixed (n=35)	p-value
Age (years)	10.0 (6.5–16.0)	12.75 (8.88–16.0)	12.0 (9.0–15.8)	0.541
Sex, n (%)				0.914
Female	6 (31.6)	24 (34.3)	13 (37.1)	
Male	13 (68.4)	46 (65.7)	22 (62.9)	
Asthma comorbidity, n (%)				**<0.001**
Absent	14 (73.7)	38 (54.3)	6 (17.1)	
Present	5 (26.3)	32 (45.7)	29 (82.9)	

Data are presented as median (Q1–Q3) or n (%), as appropriate. Comparisons between groups were performed using the Kruskal–Wallis test for continuous variables and the chi-square test for categorical variables.

(82.9%), followed by the seasonal (45.7%) and perennial (26.3%) groups. Asthma frequency was significantly higher in the mixed allergic rhinitis group than in both the perennial and seasonal groups, whereas no significant difference was observed between the perennial and seasonal phenotypes.

The distribution of allergen sensitization profiles across the groups is presented in Table II. In the seasonal allergic rhinitis group, the vast majority of patients exhibited sensitization to grass pollen (95.7%). In contrast, the most frequently identified sensitizations in the perennial group were house dust mite (36.8%), mold fungi (42.1%), and animal epithelia (cat and dog, each 31.6%). In the mixed allergic rhinitis group, concomitant sensitization to both pollen and indoor allergens was observed. Grass pollen sensitization was present in all patients (100%), while sensitization to house dust mite (28.6%), mold fungi (42.9%), and animal epithelia (62.9% cat and 42.9% dog) was also notable. Sensitization to tree and weed pollens was observed at comparable rates in the seasonal and mixed groups (34.3%–41.4% and 34.3%–40.0%, respectively).

Considering the observed differences in asthma comorbidity, hematological and inflammatory parameters were compared among allergic rhinitis phenotypes (Table III).

Table II: Distribution of allergen sensitization within perennial, seasonal, and mixed allergic rhinitis groups

Allergens (+)	Perennial rhinitis group (n=19)	Seasonal rhinitis group (n=70)	Mixed rhinitis group (n=35)
Grass pollen	0 (0.0)	67 (95.7)	35 (100)
House dust mite	7 (36.8)	0 (0.0)	10 (28.6)
Mold	8 (42.1)	0 (0.0)	15 (42.9)
Cat dander	6 (31.6)	0 (0.0)	22 (62.9)
Dog dander	6 (31.6)	0 (0.0)	15 (42.9)
Weed pollen	0 (0.0)	29 (41.4)	12 (34.3)
Tree pollen	0 (0.0)	24 (34.3)	14 (40.0)

Data are presented as n (%). Percentages indicate the proportion within each group. Patients could be sensitized to more than one allergen; therefore, column totals do not sum to 100%.

Table III: Comparison of hematological inflammatory indices among perennial, seasonal, and mixed allergic rhinitis phenotypes

Laboratory parameters	Perennial rhinitis (n=19) Median (Q1–Q3)	Seasonal rhinitis (n=70) Median (Q1–Q3)	Mixed rhinitis (n=35) Median (Q1–Q3)	p value†
Basophil count	20.0 (8.5-20.0)	20.0 (10.0-30.0)	40.0 (15.0-50.0)	**0.008**
Absolute eosinophil count	280 (185-310)	190 (90-330)	380 (200-460)	**<0.001**
Absolute monocyte count	610 (520-740)	540 (460-740)	590 (480-700)	0.479
Absolute lymphocyte count	3390 (2940-3660)	2760 (2030-3500)	2720 (2420-3140)	**0.014**
Platelet count	277000 (258000-348000)	305000 (270000-344000)	325000 (268000-345000)	0.655
Absolute neutrophil count	4060 (2680-4930)	3930 (2920-4870)	3490 (2520-4490)	0.643
Mean platelet volume (MPV, fL)	9.50 (9.00-9.80)	9.80 (9.10-10.50)	9.70 (9.35-10.60)	0.285
Neutrophil-to-lymphocyte ratio (NLR)	1.115 (0.989-1.490)	1.156 (0.996-1.961)	1.261 (0.973-1.950)	0.725
Platelet-to-lymphocyte ratio (PLR)	95.08 (66.11-115.27)	110.93 (91.48-130.07)	114.14 (93.06-127.69)	**0.020**
Eosinophil-to-lymphocyte ratio (ELR)	0.085 (0.048-0.108)	0.064 (0.028-0.112)	0.141 (0.093-0.152)	**<0.001**
Eosinophil-to-neutrophil ratio (ENR)	0.062 (0.040-0.117)	0.057 (0.025-0.099)	0.100 (0.072-0.154)	**0.002**
Systemic Inflammation Response Index (SIRI)	338235 (257122-593558)	403561 (272615-597031)	351750 (250081-651119)	0.431
Eosinophil-to-monocyte ratio (EMR)	0.378 (0.270-0.596)	0.347 (0.235-0.549)	0.719 (0.500-0.790)	**< 0.001**
Monocyte-to-lymphocyte ratio (MLR)	0.187 (0.143-0.208)	0.198 (0.150-0.245)	0.219 (0.185-0.247)	0.290

† Continuous variables are presented as median (interquartile range). Comparisons among groups were performed using the Kruskal–Wallis test. For variables with statistically significant differences, post-hoc pairwise comparisons were conducted using Dunn's test with Bonferroni correction. Cell counts are expressed as cells/μL, MPV as fL, and ratio-based indices are unitless.

Table IV: Multivariable logistic regression analysis for predictors of non-seasonal allergic rhinitis

Variable	Adjusted OR	95% CI	p-value
Comorbidity (asthma)	2.19	1.01–4.75	0.047
Eosinophil count (per 100 cells/μL increase)	1.54	1.21–1.97	<0.001

OR: odds ratio; **CI:** confidence interval.

Model χ^2=17.604, p <0.001; Nagelkerke R^2=0.177; Hosmer–Lemeshow p=0.007.

Significant differences were observed among allergic rhinitis phenotypes with respect to lymphocyte count, platelet-to-lymphocyte ratio (PLR), eosinophil-related indices, and basophil count (Table III). Lymphocyte count was higher in the perennial phenotype, whereas eosinophil-based inflammatory markers—including absolute eosinophil count, eosinophil-to-lymphocyte ratio (ELR), and eosinophil-to-neutrophil ratio (ENR)—were significantly elevated in the mixed phenotype. Basophil count was also higher in the mixed group. No significant differences were identified for the remaining hematological parameters.

Variables that were found to be statistically significant in univariate analyses were considered for inclusion in the multivariable logistic regression model. Due to the presence of collinearity among eosinophil-based indices, absolute eosinophil count was selected as a representative parameter and included in the model. In the multivariable logistic regression analysis, both eosinophil count and asthma comorbidity were independently associated with non-seasonal allergic rhinitis (Table IV). Each increase of 100 cells/μL in eosinophil count was associated with a significant increase in the odds of NSAR (odds ratio [OR]=1.54; 95% confidence interval [CI]: 1.21–1.97; p<0.001). In addition, the presence of asthma in patients with allergic rhinitis was significantly associated with NSAR, nearly doubling the likelihood of this phenotype (OR=2.19; 95% CI: 1.01–4.75; p=0.047). Overall, the model was statistically significant (χ^2=17.604; p<0.001) and explained 17.7% of the variance according to the Nagelkerke R^2 value.

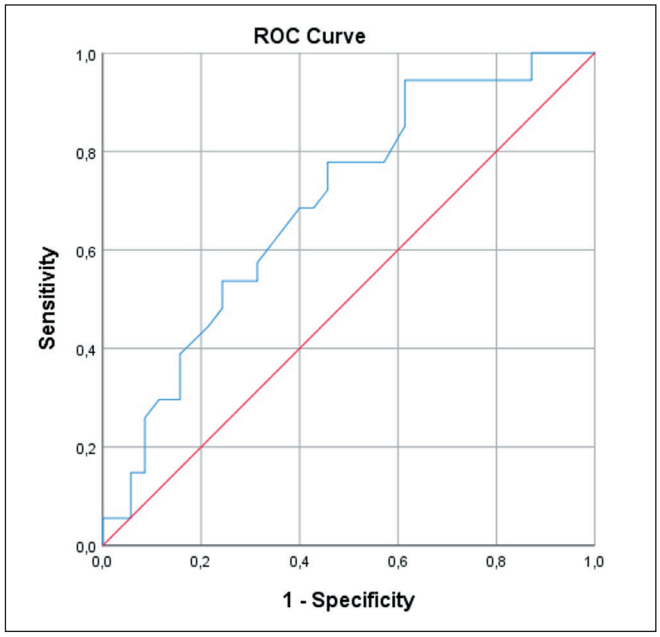

Figure 1: ROC curve of eosinophil count for distinguishing seasonal allergic rhinitis from nonseasonal allergic rhinitis. The area under the curve (AUC) was 0.692 (95% CI: 0.600–0.785), indicating a moderate discriminatory performance.

ROC curve analysis was performed to evaluate the discriminatory performance of eosinophil count. Eosinophil count demonstrated statistically significant discrimination between non-seasonal allergic rhinitis and seasonal allergic rhinitis (area under the curve [AUC]=0.692; 95% confidence interval [CI]: 0.600–0.785; p<0.001). Using the Youden index, the optimal cutoff value was identified as ≥195 cells/μL, yielding a sensitivity of 77.8% and a specificity of 54.3% (Figure 1).

Kruskal–Wallis analysis demonstrated significant differences among perennial, seasonal, and mixed allergic rhinitis groups in basophil count, absolute eosinophil count, eosinophil-derived ratios (ELR, ENR, EMR), lymphocyte count, and PLR (all p<0.05). Post hoc Dunn–Bonferroni analysis further indicated that eosinophil-related indices were generally highest in the mixed phenotype, with significant pairwise differences mainly involving this group.

DISCUSSION

Although allergic rhinitis phenotypes are traditionally defined according to symptom duration and patterns of allergen exposure, increasing attention has been directed toward the underlying inflammatory heterogeneity of this classification (14). In particular, mixed allergic rhinitis has been proposed to be associated with a more complex and

intense inflammatory response as a result of combined seasonal and perennial allergen exposure. In the present study, the marked elevation of the eosinophil-to-lymphocyte ratio (ELR) and eosinophil-to-neutrophil ratio (ENR) in the mixed phenotype may be associated with a more prominent eosinophil-predominant inflammatory profile in this group. These findings may suggest that peripheral eosinophil-based indices are associated with inflammatory activity beyond localized nasal symptoms; however, direct systemic or airway inflammatory mechanisms were not evaluated in the present study (15).

Eosinophils are key effector cells in allergic inflammation and are activated primarily through T helper 2 (Th2)–associated cytokines, particularly interleukin-5 (IL-5) (16). Previous studies have reported associations between eosinophil counts and eosinophil-based indices and disease severity in allergic rhinitis (8). In the present study, the significantly higher eosinophil-based inflammatory indices observed in the mixed group compared with the seasonal group suggest that continuous allergen exposure may be associated with higher peripheral eosinophil-related inflammatory indices.

Another noteworthy finding of the present study was the higher lymphocyte count observed in the perennial allergic rhinitis group compared with the other phenotypes. This finding may indicate that, in the perennial phenotype, more chronic and regulatory immune mechanisms are predominant, with a lymphocyte-mediated immune response playing a more prominent role rather than acute eosinophilic inflammation. In parallel, the lower platelet-to-lymphocyte ratio (PLR) detected in the perennial group may suggest a relatively lower inflammatory burden in this phenotype (17). Consistent with this observation, previous studies have reported that PLR represents a nonspecific marker of inflammation and yields variable results in allergic diseases (9). The very high rate of grass pollen sensitization observed in the mixed allergic rhinitis group may be related to the regional aeroallergen profile of our center and the frequent coexistence of grass pollen sensitization with perennial allergen sensitization in polysensitized patients.

The findings related to asthma comorbidity strongly support the concept of "united airway disease." In the present study, the markedly higher prevalence of asthma in the mixed allergic rhinitis group, together with the identification of asthma as an independent predictor of non-seasonal allergic rhinitis in multivariable analysis, suggests that the upper and lower airways may be affected by potentially overlapping inflammatory pathways. As emphasized by Canonica et al. allergic rhinitis and asthma should be regarded as different anatomical manifestations of the same underlying inflammatory process (18).

Asthma represents a potential confounding factor in the interpretation of eosinophil-related inflammatory markers, particularly given the markedly higher prevalence of asthma in the mixed allergic rhinitis phenotype. Because asthma itself is commonly associated with eosinophil-predominant type 2 inflammatory responses, part of the observed increase in eosinophil-based indices may reflect concomitant lower airway inflammation rather than allergic rhinitis phenotype alone. However, in the present study, asthma comorbidity was included in the multivariable logistic regression model, and absolute eosinophil count remained independently associated with non-seasonal allergic rhinitis after adjustment for asthma. These findings suggest that the observed eosinophilic inflammatory pattern is not solely attributable to asthma comorbidity, although a partial contributory effect cannot be excluded.

In the multivariable logistic regression analysis, the finding that each 100 cells/μL increase in eosinophil count was associated with a 54% increase in the odds of non-seasonal allergic rhinitis suggests that eosinophil count may serve as a complementary biomarker in the phenotypic evaluation of allergic rhinitis, rather than a standalone diagnostic marker. Although ELR and PLR were significant in univariate analyses, their exclusion from the multivariable model represents a deliberate methodological decision. The substantial collinearity between these composite indices and their constituent parameters, along with the potential risk of compromised model stability in the context of a limited sample size, constitutes a well-recognized statistical concern in the literature. This analytical approach enhances the robustness of the findings and minimizes the risk of overfitting.

In a previous study, the mean absolute eosinophil count was reported as 315 cells/μL in patients with allergic rhinitis, compared with 147 cells/μL in healthy controls (8). In the same study, eosinophil counts were reported to be significantly higher in patients with persistent allergic rhinitis (339 ± 246 cells/μL) compared with those with intermittent allergic rhinitis (232 ± 195 cells/μL). These

findings suggest that eosinophilic inflammation is closely associated with both the duration of allergic rhinitis and the overall inflammatory burden. In our study, the identification of a cutoff value of ≥195 cells/μL for absolute eosinophil count in distinguishing non-seasonal from seasonal allergic rhinitis, with significantly higher values observed in the non-seasonal group, further supports the notion that eosinophil count may represent a more prominent biomarker in phenotypes characterized by persistent and sustained inflammation. Moreover, in a pediatric cohort aged 3–10 years, Yücel Ekici demonstrated that the mean absolute eosinophil count was significantly higher in children with allergic rhinitis compared with healthy controls ($0.413 \pm 0.301 \times 10^3$/μL vs. $0.279 \pm 0.271 \times 10^3$/μL; p=0.003), reinforcing the potential utility of peripheral eosinophil count as an easily accessible marker reflecting eosinophilic inflammation in allergic rhinitis (19). In the same study, ROC analysis suggested a cutoff value of ≥0.34×10^3/μL (≥340 cells/μL) for eosinophil count in predicting allergic rhinitis (and/or sensitization), providing moderate specificity but relatively limited sensitivity. In contrast, the objective of the present study was not to discriminate the presence or absence of allergic rhinitis per se, but rather to distinguish between seasonal and non-seasonal phenotypes. Accordingly, the identification of a lower cutoff value for absolute eosinophil count (≥195 cells/μL), which was particularly prominent in non-seasonal allergic rhinitis, suggests that eosinophilic inflammation may be more pronounced in phenotypes characterized by year-round allergen exposure and a more persistent inflammatory burden. Thus, while the findings of Yücel Ekici broadly confirm the association between elevated eosinophil counts and allergic rhinitis, our results indicate that eosinophil count may have greater discriminatory value in persistent phenotypes and that optimal cutoff values may vary depending on the targeted clinical distinction (disease presence vs. phenotype differentiation). In the present study, a cutoff value of ≥195 cells/μL for absolute eosinophil count yielded a sensitivity of 77.8% and a specificity of 54.3% in distinguishing non-seasonal from seasonal allergic rhinitis.

In an adult cohort studied by Kant and Terzioğlu (8), comprising 205 patients with allergic rhinitis and 49 healthy controls, asthma comorbidity was reported to be comparable between persistent and intermittent allergic rhinitis groups (38.8% vs. 40.0%; p=0.879). In contrast, in our pediatric cohort, when allergic rhinitis was classified according to patterns of allergen exposure as seasonal and non-seasonal (year-round exposure), the presence of asthma emerged as an independent predictor of non-seasonal allergic rhinitis (NSAR) in multivariable analysis. This discrepancy may be attributable to the fact that continuous allergen exposure during childhood is more likely to be associated with sustained Th2-driven eosinophilic inflammation, thereby strengthening the coexistence of asthma.

This study has several limitations. The retrospective design and single-center setting may limit the generalizability of the findings. Additionally, the evaluation of complete blood count parameters exclusively during the non-pollen season precluded assessment of potential seasonal variability. Because direct airway inflammatory markers or cytokine profiles were not evaluated, the observed hematological indices should be interpreted as indirect indicators of inflammatory activity rather than direct evidence of systemic inflammation. The unequal distribution of patients among allergic rhinitis phenotypes, particularly the relatively small number of patients in the perennial allergic rhinitis group, may also have affected the statistical power of subgroup comparisons and the stability of regression analyses. Furthermore, although the multivariable logistic regression model demonstrated statistically significant discriminatory ability, the significant Hosmer–Lemeshow test result indicates a potential limitation in model calibration; therefore, the predictive performance of the model should be interpreted with caution.

In conclusion, this study demonstrates that, in pediatric allergic rhinitis, the mixed phenotype is associated with higher peripheral eosinophil-based inflammatory indices, and that eosinophil count may serve as a complementary inflammatory biomarker associated with non-seasonal allergic rhinitis phenotypes. These readily accessible parameters obtained from routine clinical practice may provide supportive information in the evaluation of allergic rhinitis phenotypes when interpreted together with clinical findings and sensitization profiles. Future prospective studies with larger sample sizes are warranted to validate these findings and to facilitate their integration into clinical decision-making algorithms.

Ethical Approval

The study was approved by the Fırat University Non-Interventional Clinical Research Ethics Committee (Decision No. 2026/01-03; meeting date: 15 January 2026; approval document issued on 22 January 2026).

Conflict of Interest

The authors declare no conflicts of interest related to this work.

Author Contributions

Concept: **FDS,** Design: **MK,** Data collection or processing: **HSS,** Analysis or Interpretation: **OK,** Literature search: **FDS,** Writing: **FDS,** Approval: **FDS, HSS, OK, MK.**

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