ORIGINAL RESEARCH

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Asthma evaluation of risk factors in atopic patients in the Hatay region

Kadir Burak Akgün, Sahinur Aycan Alkan, , Ömer Atar

[1]Hatay Mustafa Kemal University Tayfur Ata Sökmen Faculty of Medicine, Department of Pulmonology, Hatay, Türkiye

Abstract

Objective: This study investigated the role of systemic inflammatory indices and specific aeroallergen sensitizations in predicting asthma among patients with allergic rhinitis (AR) and asthma, within the United Airway Disease (UAD) framework.
Method: A retrospective study was conducted including 575 patients who underwent skin prick testing (SPT). Patients were categorized into four groups: healthy controls, AR only, allergic asthma, and non-allergic asthma. Demographics, SPT results, and inflammatory indices were compared. Independent risk factors for asthma were analyzed via multivariate logistic regression.
Results: No statistically significant differences in eosinophil levels or systemic inflammatory indices were detected among the four phenotypes ($p>0.05$). However, the median age and the proportion of females were observed to be higher in asthmatic patients. In the multivariate analysis, increasing age (OR: 1.019), being female sex (OR: 1.515), tree pollen (OR: 1.953), and cockroach (OR: 1.618) sensitizations were identified as independent risk factors for asthma. House dust mite sensitization was lost its significance in the multivariate model.
Conclusion: The predictive value of systemic inflammatory indices in distinguishing asthma phenotypes is considered low in stable atopic patients. Tree pollen and cockroach sensitizations, rather than house dust mites, were proven as the strongest independent determinants of asthma development among the studied population in the Mediterranean climate of Hatay, influenced by climate change and precipitation.
Keywords: Aeroallergens, allergic rhinitis, asthma, systemic inflammation, united airway disease

INTRODUCTION

Asthma is characterized as a disease involving chronic inflammation in the lower airways, accompanied by respiratory complaints and airway narrowing that vary in time and intensity. Although the allergic asthma phenotype, primarily driven by eosinophilic inflammation, is most frequently observed, the non-allergic asthma phenotype presenting with neutrophilic inflammation is also known to exist (1). Despite the differences in the pharmacological treatments of allergic rhinitis (AR), an inflammatory process is recognized in the upper airways, and a high coexistence of this disease with asthma is noted (2). Although this coexistence was noticed long ago, the united airway disease (UAD) concept, which aims to elucidate the anatomical, pathological, physiological, clinical, and therapeutic similarities between them, has recently been established (3). This concept proposes that the underlying pathophysiology is driven by type-2 inflammation—frequently triggered by aeroallergens such as pollen, mold, and house dust mites—where a single pathological change in the lower and upper respiratory tracts is caused by inflammatory elements, primarily IgE, eosinophils, and IL-5 (4,5).

Clinical complaints are induced by pollen allergy, particularly through the triggering of type-2 inflammation

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Corresponding Author: Kadir Burak Akgün, Hatay Mustafa Kemal University Tayfur Ata Sökmen Faculty of Medicine, Department of Pulmonology, Hatay, Türkiye
E mail: kadirburak.akgun@mku.edu.tr
ORCID iD: 0000-0002-3017-1025

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in susceptible cases of seasonal allergic asthma and seasonal AR (6). The skin prick testing (SPT) is the most widely used, fastest, and most cost-effective method to identify antigens causing IgE production. Sensitivity to the antigen is indicated by the formation of a wheal measuring 4 mm or more on the skin 20 minutes after the specific antigen is applied. The measurement of specific IgE (sIgE) in serum is utilized as the other most common method; however, it is known to be more expensive, and its sensitivity to respiratory allergens may be lower. In addition, the success of both tests in capturing the patient's sensitization profile is observed to vary depending on the content and breadth of the panel used (7).

Eosinophils are recognized as the primary cells of the allergic process and they are utilized for monitoring purposes in asthma, AR, and atopic dermatitis. It has been demonstrated that allergic cases with serum blood levels detected above 300 cells/μL benefit from biological therapies; however, it should also be noted that their absolute counts or percentages do not directly reflect disease activity (8,9). A correlation between high eosinophil levels and the asthma severity of patients could not be demonstrated (10). Furthermore, eosinophil counts can be affected by many factors, and different serum level results can be obtained at different times even in the same individual (11). No significant relationship has been found between the serum eosinophil, which is noted as another important cell, count and the total IgE levels (12). Due to these limitations of SPT, sIgE, and eosinophils, new biomarkers that demonstrate inflammation from a broader perspective, are stable, inexpensive, and can easily be obtained from routine complete blood counts are needed.

This study aimed to examine cases evaluated for AR and asthma in our allergy unit. Accordingly, by calculating inflammatory indices derived from complete blood counts alongside traditional measurements such as eosinophil count and percentage, how these systemic biomarkers interact with the clinical complaints, demographic characteristics, and specific allergen sensitizations of the patients was aimed to be revealed.

METHOD

Study Population and Data Processing

Inclusion criteria

Within the scope of this retrospectively designed study, the medical records of patients on whom the SPT was performed between 01.03.2025 and 28.02.2026 in the Allergy Unit were scanned. The age, sexsex, atopy and asthma statuses, SPT results, and the eosinophil count and ratio, as well as neutrophil, lymphocyte, platelet, and monocyte counts, obtained from the complete blood count of the patients, were recorded from the patient files.

With the obtained data, the following inflammatory indices were calculated: NLR: Neutrophil/lymphocyte ratio, PLR: Platelet/lymphocyte ratio, ELR: Eosinophil/lymphocyte ratio, MLR: Monocyte/lymphocyte ratio, ENR: Eosinophil/neutrophil ratio, EMR: Eosinophil/monocyte ratio, SII (Systemic Immune-Inflammation Index): Platelet × Neutrophil / Lymphocyte, SIRI (Systemic Inflammation Response Index): Neutrophil × Monocyte / Lymphocyte, AISI (Aggregate Index of Systemic Inflammation): Neutrophil × Platelet × Monocyte/Lymphocyte

Patient groups

The diagnostic and phenotyping processes of the patients included in the study were conducted based on current international guidelines. Asthma diagnoses were established in accordance with the Global Initiative for Asthma (GINA) guideline criteria, accompanied by detailed a clinical anamnesis, symptom variabilities, and pulmonary function test (PFT) findings (1). AR diagnoses, on the other hand, were finalized in line with the Allergic Rhinitis and its Impact on Asthma (ARIA) guideline recommendations by evaluating the clinical correlation between the typical nasal symptoms of the patients at the time of presentation (rhinorrhea, nasal obstruction, sequential sneezing, and nasal itching) and the aeroallergen sensitizations detected via SPT (2).

The participants were categorized into four groups: the control group consisted of individuals without rhinitis or asthma symptoms who also had negative skin prick test (SPT) results. AR group included patients with typical rhinitis symptoms and at least one positive SPT result but without an asthma diagnosis. Allergic Asthma group comprised patients clinically diagnosed with asthma who showed at least one positive result in the SPT panel. Non-Allergic Asthma group was defined by patients who had a clinical asthma diagnosis with negative results across the entire SPT panel. Due to the pathogenesis of the upper respiratory tract, asthma and allergic rhinitis are considered to be frequently co-occurring phenotypes in clinical practice. Therefore, in our study, to fully reflect real-world data, the co-occurrence of rhinitis in asthma groups (Groups 3 and 4) was not considered an exclusion criterion. The primary differentiating factor in the classification was determined to be lower respiratory tract involvement and the patient's atopic status (positive SPT).

In cases where a blister of ≥4 mm was formed with a positive control (histamine) and no response was obtained with a negative control (saline solution), the detection of a blister of ≥4 mm against at least one of the tested allergens was considered positive response (atopy).

Allergen panel included the following contents: Mixture of tree pollens (birch, alder, ash), mixture of grass pollens (ryegrass, meadow grass, couch grass, meadow fescue, timothy grass), mixture of cereals (rye, oat, wheat), mixture of house dust mites (Dermatophagoides pteronyssinus, Dermatophagoides farinae), cat epithelium, dog epithelium, cockroach (Blattella germanica), and mold fungus (*Aspergillus fumigatus*).

Exclusion criteria

To ensure the standardization of the study, patients who were referred from other clinics or hospitals solely for the application of the SPT without our clinical evaluation were excluded. In addition, patients diagnosed with non-asthma chronic lung diseases such as bronchiectasis, pulmonary aspergillosis, and chronic obstructive pulmonary disease (COPD), those with inappropriate positive (histamine) and/or negative (saline) control results in the SPT, individuals without complete blood count data within one month prior to the test date, patients have received antihistamine, leukotriene receptor antagonist, or monoclonal antibody treatment within the last 10 days, and individuals under the age of 18 were not included in the scope of the study.

Statistical Analysis

The statistical analysis was performed using the IBM SPSS Statistics for Windows, Version 25.0 (IBM Corp., Armonk, NY, USA) software package. The distribution characteristics of the variables were examined with the Kolmogorov-Smirnov test; it was determined that all continuous variables did not conform to a normal distribution. Continuous data were presented as median and interquartile range (IQR), while categorical data were presented as numbers (n) and percentages (%).

In comparisons between groups, the Mann-Whitney U test was used for two independent groups, and the Kruskal-Wallis test was used the four clinical phenotypes. Bonferroni-corrected post-hoc analyses were preferred in the multiple comparisons of the parameters for which a significant difference was detected in the Kruskal-Wallis test. Pearson chi-square test was utilized in the analysis of categorical variables and the comparison of proportions between groups. In the multiple comparisons of categorical data, a Bonferroni correction based on the comparison of column proportions was applied.

The relationships between continuous variables and the allergen sensitization burden were evaluated using Spearman correlation analysis. To determine the independent risk factors predicting the presence of asthma, a multivariate logistic regression analysis, in which variables (age, sex, specific allergen sensitizations) showing significance ($p < 0.05$) in univariate analyses were included, was applied. Regression results were reported as odds ratio (OR) and 95% confidence interval (CI). The statistical significance level was accepted as $p < 0.05$ in all analyses.

RESULTS

The median age of the 575 patients included in the study was determined to be 43 (IQR: 28, range: 18-90) years; 61.6% (n=354) of the cohort was constituted by females, and 38.4% (n=221) by males (female/male ratio: 1.60). The patients were divided into four groups according to their clinical phenotypes: healthy control (n=126, 21.9%), AR only (n=208, 36.2%), allergic Asthma (n=178, 31.0%), and non-allergic asthma (n=63, 11.0%).

A statistically significant difference was detected among the groups in terms of age distribution ($p < 0.001$). While the youngest group was formed by the AR only (median: 36.5 years) group, the oldest group was formed by the non-allergic asthma (median: 51.0 years) group. When the sexsex distribution was examined; the female sex ratio was determined to be the highest (74.7%) in the allergic asthma group and the lowest (44.4%) in the control group ($p < 0.001$). It was found that 73.8% (178/241) of the asthma patients in our study were of the allergic (atopic) phenotype. (Table 1)

When atopic individuals (AR only and allergic asthma groups) were compared in terms of specific aeroallergen sensitizations, tree pollen sensitization was determined to be statistically significantly higher in the allergic asthma group (39.3%) compared to the AR only group (23.6%) ($p = 0.001$). Cockroach sensitization was also observed more frequently in the allergic asthma group (41.6%) compared to the AR only group (32.2%), but this difference was noted to remain at the statistical borderline ($p = 0.057$). In erms of house dust mite, grass pollen, cereal pollen, cat/dog epithelium, and Aspergillus sensitizations, no significant difference was detected between the AR only and allergic asthma phenotypes ($p > 0.05$ for all). (Table 2)

When the entire study population was grouped according to the presence of asthma; the median age of the patients with asthma was determined to be 48 (IQR: 28) years, while that of those without asthma was determined to be 40 (IQR: 30) years, and the difference between them was found to be statistically significant ($p < 0.001$). When the sex distribution was examined, the female sex ratio was determined to be 68.5% (n=165) in the asthmatic group and 56.6% (n=189) in the non-asthmatic group, and female sex was identified to be significantly higher in the asthmatic group ($p = 0.004$). (Table 3)

Table 1. Demographic and clinical characteristics of the study population

Variables	Group 1: Control (n=126)	Group 2: AR Only (n=208)	Group 3: Allergic Asthma (n=178)	Group 4: Non-Allergic Asthma (n=63)	p-value
Age [Median (IQR)]	45.5 (31.0)[ab]	36.5 (27.0)[a]	45.0 (27.0)[b]	51.0 (23.0)[b]	**<0.001***
Sex [n (%)]					**<0.001****
- Female	56 (%44.4)[a]	133 (%63.9)[bc]	133 (%74.7)[c]	32 (%50.8)[ab]	
- Male	70 (%55.6)[a]	75 (%36.1)[bc]	45 (%25.3)[c]	31 (%49.2)[ab]	
Female/Male ratio	0.80	1.77	2.95	1.03	-

*Pearson Chi-Square Test, **Kruskal-Wallis Test, AR: Allergic Rhinitis. For age and sex variables, a statistically significant difference was found between groups bearing different subscript letters (a, b, c) in the same row, according to Bonferroni-corrected post-hoc analyses ($p < 0.05$).

Table 2. Distribution of allergen sensitizations in atopic individuals according to clinical diagnosis

Allergen Group	AR Only (n=208)	Allergic Asthma (n=178)	p-value*
Tree Pollen	49 (23.6%)	70 (39.3%)	**0.001**
Cockroach	67 (32.2%)	74 (41.6%)	0.057
House Dust Mite	61 (29.3%)	62 (34.8%)	0.247
Grass Pollen	88 (42.3%)	76 (42.7%)	0.939
Cereal Pollen	77 (37.0%)	69 (38.8%)	0.725
Cat Epithelium	63 (30.3%)	54 (30.3%)	0.992
Dog Epithelium	26 (12.5%)	23 (12.9%)	0.901
Aspergillus	29 (13.9%)	27 (15.2%)	0.733

*Pearson Chi-Square Test was utilized; AR: Allergic rhinitis.

Table 3. The relationship of demographic and all allergic factors with the presence of asthma

Variables	Asthma Absent (Control + AR Only) (n=334)	Asthma Present (Allergic + Non-Allergic) (n=241)	p value
Age [Median (IQR)]	40.0 (30)	48.0 (28%)	**<0.001***
Sex [Female, n (%)]	189 (56.6%)	165 (68.5%)	**0.004****
Tree Pollen (n, %)	49 (14.7%)	70 (29.0%)	**<0.001****
Cockroach (n, %)	67 (20.1%)	75 (31.1%)	**0.002****
House Dust Mite (n, %)	61 (18.3%)	62 (25.7%)	**0.031****
Grass Pollen (n, %)	88 (26.3%)	76 (31.5%)	0.174
Cereal Pollen (n, %)	77 (23.1%)	69 (28.6%)	0.130
Cat Epithelium (n, %)	63 (18.9%)	54 (22.4%)	0.298
Dog Epithelium (n, %)	26 (7.8%)	23 (9.5%)	0.456
Aspergillus (n, %)	29 (8.7%)	27 (11.2%)	0.314

*Mann-Whitney U Test, **Pearson chi-square test; AR: Allergic rhinitis.

When the relationship between specific allergen sensitizations and the presence of asthma was examined; sensitizations to tree pollen (29.0% vs. 14.7%; p < 0.001), cockroach (31.1% vs. 20.1%; p = 0.002), and house dust mite (25.7% vs. 18.3%; p = 0.031) were detected to be significantly more frequent in the asthmatic group. On the other hand, no statistically significant difference was detected between the groups with and without asthma in terms of sensitizations to grass pollen (31.5%), cereal pollen (28.6%), cat epithelium (22.4%), Aspergillus (11.2%), and dog epithelium (9.5%) (p > 0.05 for all) (Table 3).

As a result of the Kruskal-Wallis test performed among the four clinical phenotypes (Control, AR only, allergic asthma, and non-allergic asthma), no statistically significant difference was detected in terms of neutrophil (p = 0.996), lymphocyte (p = 0.879), monocyte (p = 0.474), platelet (p=0.162), and eosinophil (p = 0.937) counts, as well as the eosinophil ratio (p = 0.933). Similarly, no significant difference was observed among the groups in any of the calculated inflammatory indices (NLR, PLR, ELR, MLR, ENR, EMR, SII, SIRI, and AISI) (p > 0.05 for all) (Table 4).

According to the results of the logistic regression analysis; increasing age (OR: 1.019, 95% CI: 1.009-1.030, p < 0.001), female sex (OR: 1.515, 95% CI: 1.053-2.179, p = 0.025), tree pollen sensitization (OR: 1.953, 95% CI: 1.264-3.012, p = 0.003), and cockroach sensitization (OR: 1.618, 95% CI: 1.080-2.427, p = 0.020) were identified as independent risk factors for the presence of asthma. House dust mite sensitization, which was found to be significant in the univariate analysis, was determined to lose its significance when evaluated together with other factors in the logistic regression model (p = 0.328) (Table 5).

Table 4. Comparison of basic hematological parameters and systemic inflammatory indices according to clinical phenotypes

Parameter	Control (n=126)	AR Only (n=208)	Allergic Asthma (n=178)	Non-Allergic Asthma (n=63)	p-value
Neutrophil (/µL)	4675 (2235)	4590 (2442.5)	4645 (2380)	4840 (2190)	0.996
Lymphocyte (/µL)	2145 (952.5)	2265 (960)	2240 (900)	2240 (1025)	0.879
Monocyte (/µL)	500 (230)	480 (207.5)	490 (220)	450 (210)	0.474
Eosinophil (/µL)	170 (200)	160 (185)	170 (170)	182 (140)	0.937
Platelet (10^3/µL)	266 (76.5)	269 (89.5)	283.5 (99.75)	284 (129)	0.162
NLR	2.02 (1.13)	2.02 (1.10)	2.06 (1.18)	1.99 (1.47)	0.995
PLR	121.71 (51.77)	119.55 (53.80)	129.21 (57.16)	128.92 (76.73)	0.698
ELR	0.07 (0.10)	0.07 (0.09)	0.07 (0.08)	0.08 (0.09)	0.872
MLR	0.22 (0.14)	0.21 (0.10)	0.21 (0.11)	0.20 (0.15)	0.567
SII	560276 (367109)	560818 (386123)	587927 (389236)	529525 (377311)	0.904
SIRI	1014.00 (1062.92)	999.62 (768.65)	966.20 (889.33)	974.83 (855.73)	0.888
AISI	285292373 (310880051)	270597568 (250633674)	268166676 (252793992)	265696449 (234447564)	0.978

Data are presented as Median (Interquartile Range - IQR). Comparisons between groups were performed using the Kruskal-Wallis H test. AR: Allergic Rhinitis, NLR: Neutrophil/Lymphocyte Ratio, PLR: Platelet/Lymphocyte Ratio, ELR: Eosinophil/Lymphocyte Ratio, MLR: Monocyte/Lymphocyte Ratio, SII: Systemic Immune-Inflammation Index, SIRI: Systemic Inflammatory Response Index, AISI: Aggregate Index of Systemic Inflammation.

Table 5. Independent Risk Factors Predicting the Presence of Asthma

Variables	B	Standard Error	p-value	OR (Exp B)	%95 CI
Age	0.019	0.005	<0.001	1.019	1.009 – 1.030
Female Sex	0.415	0.185	0.025	1.515	1.053 – 2.179
Tree Pollen	0.669	0.222	0.003	1.953	1.264 – 3.012
Cockroach	0.482	0.206	0.020	1.618	1.080 – 2.427
House Dust Mite	0.215	0.220	0.328	1.239	0.806 – 1.908

OR: Odds Ratio, CI: Confidence Interval

DISCUSSION

As a result of this study, age and sex were determined to be associated with atopy. While sensitization rates to tree pollen, cockroach, and house dust mite were detected to be significantly higher in cases with allergic asthma compared to individuals without asthma; a similar relationship could not be demonstrated for grass pollen, cereal pollen, cat, dog, and Aspergillus. In the multivariate logistic regression analysis applied to predict the development of allergic asthma; it was recorded that female sex was associated with a 1.51-fold increased risk compared to males, tree pollen sensitization with a 1.95-fold increased risk, and cockroach sensitization with a 1.61-fold increased risk, whereas the risk was noted to be increased by 1.9% with each one-year increase in age. House dust mite sensitization, which was seen as significant in univariate analyses, was not observed to maintain its significance as an independent risk factor in the multivariate model. It is revealed by these results that tree pollen and cockroach sensitizations are strong and independent indicators in terms of the presence of asthma in the Hatay region.

While asthma prevalence is higher in males during childhood, this picture is observed to change in favor of females during adulthood (13). In parallel with this, the lifetime risk of developing asthma in women is known to be significantly higher compared to men. It has been demonstrated that asthma control is worse in women, exacerbations are experienced more frequently by them, and this situation is independent of eosinophils (14). Similar to the literature, asthma and allergic conditions were observed more frequently in women in our study as well, and this situation was presented independently of eosinophils. Because the persistence of asthma risk continues lifelong and due to the chronic nature of the disease, the risk was detected to be increased with age. Although it has been shown that this situation is caused by the different expression and activity of type-2 inflammatory cells in different sexes, the results are contradictory and complex, and sufficient information at a level encompassing sex-specific asthma management is not provided (15).

In this study, the effects of specific aeroallergens on atopy were examined in detail. In particular, tree pollen allergy has been shown to be observed more frequently in asthmatic individuals compared to those with AR, and it has been demonstrated to be the aeroallergen that most increases the risk of asthma development. While tree pollens are clinically evaluated, changing climate events should also be discussed alongside them. With the effect of climate change, negative impacts on respiratory health are expected in terms of pollen exposure and allergenicity. In particular, plant growth and development have been supported by increasing temperature and CO2 levels, leading to longer flowering periods, by which an increase in pollen production has been caused. With the continued increase in extreme weather events, respiratory health expectations related to aeroallergens are considered pessimistic (16). When the meteorological data of the Hatay province for the last 40 years are analyzed, a statistically remarkable upward trend in annual precipitation amounts is observed (17). Considering the rapid growth capability, preference for wetlands, and increased spreading potential with precipitation events of ash, alder, and birch trees included in our tree pollen solution, it should be taken into consideration that the impact of tree pollens on allergic diseases in our province may increase (18,19).

Cockroach allergen has also been viewed as an independent risk factor in the development of asthma. In the literature, respiratory allergy is also known to be caused by cockroach allergy, and the effects of atopy, which is formed by the tropomyosin protein, are known to be continued even after the cockroach is gone (20). Although the airborne transportation of the cockroach allergen is made difficult due to its large particle size, the reproduction of blattella germanica, which is included in our solution, in residences and hot, humid environments is facilitated easily (21,22). It is revealed by the 40-year temperature records in Hatay province that temperature averages are in a regular increasing trend in parallel with global climate change (23). It is indicated by this finding that precautions should be taken against the cockroach population in our region due to the effect of increasing temperatures; otherwise, it is pointed out that there is a risk of an increase in allergic diseases related to this.

House dust mites are also defined as quite well-characterized organisms in allergy and asthma. In particular, Dermatophagoides pteronyssinus and dermatophagoides farinae are recognized as the most frequently observed house dust mite types, and their mixed solutions were used in our study. However, the allergenic potential of the house dust mite depends not only on the characteristic of the protein but also on how it is transported to the respiratory tract, because the allergenic dermatophagoides protein is actually identified as a mite fecal protein (24). While the allergic reaction is initiated by tree pollens being directly transported to the lungs through wind influence, IgE-mediated hypersensitivity is caused by the aerosolized proteins of cockroaches originating from a broader range of sources (saliva, feces, secretions, debris, and dead bodies) (25). It is explained by this information why the relationship of the house dust mite with allergy is not as strong as its relationship with asthma and why it could not be identified as an independent risk factor.

When identifying risk factors for asthma, it's important to consider age, sex, and regional differences. The genetic inheritance of asthma and atopy may contribute to regional variations. A study of children in the Elazığ region showed male predominance and sensitivity primarily to grass and cereal pollens (26). A study of adults in Şanlıurfa found differences in sensitivity to fungi, cereals, and cockroaches between the local population and refugees (27). The differences between these studies, including ours, highlight the importance of local epidemiological data for allergen sensitivity. To our knowledge, our study is the first to independently demonstrate tree pollen and cockroach sensitization as an association in asthma in the Hatay province.

The pathophysiological basis for no significant difference being detected among the groups in terms of inflammatory indices (NLR, PLR, SII, etc.) in our study can be explained by the cellular character of inflammation within the UAD concept. When local airway samples (nasal lavage and induced sputum) of AR and asthma patients are examined in the literature; it has been shown that lower and upper airway eosinophils are significantly increased, but no significant change in neutrophil ratios is observed compared to healthy controls, and even the local neutrophil response is suppressed (28). As a matter of fact, even in a recent study where severe and systemic allergic reactions such as anaphylaxis are examined, no statistical relationship could be detected between the risks of the patients developing a biphasic reaction during their 72-hour follow-up and their NLR, PLR, SII, or SIRI values at the time of admission (29). It is confirmed by this finding that systemic inflammation markers do not possess the expected sensitivity in predicting the clinical course of IgE-mediated diseases and that the systemic response in allergic pathologies is kept quite limited contrary to what is thought. Similarly, in another study where asthma patients are compared according to their clinical stages; it was clearly revealed that indices such as NLR and PLR are elevated only during acute exacerbation (attack) periods, and no significant difference is shown by them between patients in the stable period and healthy controls (30).

Although the relationship between inflammatory indices and allergic diseases in the literature is seen as contradictory at first glance, this situation is primarily caused by differences in the clinical stages in which the cohorts are examined and in methodological designs. For example, in a recent study conducted in our country, the NLR, lymphocyte/monocyte ratio (LMR), ELR, and SII values of newly diagnosed allergic and non-allergic rhinitis patients were reported to be significantly higher compared to the control group; it was argued that the inflammation in rhinitis did not remain at the local level but a systemic response was triggered by it, and that SII and ELR were correlated with the presence of asthma (31). The most distinct methodological difference is constituted by the fact that the data were collected during the acute symptomatic period of the patients in the aforementioned study, whereas only patients in the stable stage were included in our study due to SPT safety procedures. It is indicated by this situation that, in our cohort, inflammation is manifested as a chronic mucosal reaction originating from specific aeroallergens and limited to the target tissue (local), rather than a systemic response. On the other hand, large-scale epidemiological screening studies by which indices such as SII are reported to be positively associated with asthma prevalence are also found in the literature. As a matter of fact, in a massive cohort where 23,176 participants (3,840 asthmatics, 15.02%) were examined by utilizing the NHANES database, it was reported that logarithmically transformed high SII scores were associated with increased adult asthma prevalence (32). However, quite important methodological limitations are constituted by the facts that the asthma diagnosis in such large population screenings is based on patient declaration rather than objective respiratory and allergy tests, the distinction between the attack/stable periods of the patients cannot be made, and the aeroallergen sensitization profiles are not known. Our study is distinguished by a more homogeneous cohort being presented, whose detailed clinical phenotyping was performed in chest diseases and allergy units, whose specific SPT results were proven, and who were followed up in the stable period. The course of local mucosal inflammation independent of systemic blood circulation in atopic diseases is clearly revealed by this homogeneous structure, and this diagnostic gap in the literature is filled by it.

Limitations of the Study

Some limitations of our study are acknowledged. First, it is stated that the obtained results may not be generalized to the entire population due to the single-center design of the study; however, to our knowledge, this research is considered the first study by which local epidemiological data regarding aeroallergen sensitization in the Hatay province are presented. Second, it is noted that a diagnostic limitation originating from current health policies specific to the chest diseases branch is constituted by the fact that the applied SPT panel is limited to 8 specific allergens and sIgE measurement cannot be performed in our clinic. Therefore, a methodology that can easily be applied and repeated under daily outpatient clinic conditions by specialists possessing similar facilities is presented by our study. Third, an a priori power analysis was not performed at the beginning of the study because all accessible patients by whom the inclusion criteria were met were taken into the analysis due to the retrospective nature of the research. Finally, although a data

loss seems to be implied by the fact that only 575 patients could be included in the analysis due to missing data as a result of the scanning of 745 (64.1%) of the 1162 patients on whom SPT was applied in the allergy unit and who were evaluated in the chest diseases outpatient clinic, it is actually indicated by this situation how strictly the exclusion criteria of the study were applied and the methodological rigor is demonstrated.

CONCLUSION

The UAD concept is considered highly valuable since a holistic approach to airway diseases is provided by it. However, as observed in our study, local and systemic pathophysiological differences are harbored by asthma even within itself. On the other hand, a determining role is played by environmental factors in atopy and asthma. It is concluded that being aware of regional allergens and individual sensitivities can be more beneficial than simple inflammatory markers in the prediction of asthma. Future research should utilize broader allergen panels and focus on regions with similar climatic and ecological profiles to validate the generalizability of our findings.

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