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Aeroallergen sensitization in chronic spontaneous urticaria

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Abstract
Aim: To investigate the prevalence and distribution of aeroallergen sensitization in adult patients with chronic spontaneous urticaria (CSU), compared to a control group with allergic rhinitis (AR).
Method: This retrospective cross-sectional study included 158 CSU and 166 AR patients examined between October 15, 2023, and July 1, 2025. Demographics, clinical features, skin prick test (SPT) results for common aeroallergens (house dust mites, pollens, molds, animal danders), and hematological markers (e.g., basophil/eosinophil counts, total IgE) were examined.
Results: The CSU group was significantly older than controls ($p<0.001$), and female sex was similarly dominant in both groups ($p=0.296$). Aeroallergen sensitization was less frequent in CSU (39.24%) than AR (100%; $p<0.001$), with house dust mite (20.89% vs. 43.98%; $p<0.001$) and pollen (11.39% vs. 34.94%; $p<0.001$) detected as the primary allergens. Multiple sensitizations were also less common in the CSU group (5.06% vs. 21.08%; $p<0.001$). In terms of comorbidities, asthma was rarer among individuals with CSU (0.63% vs. 14.46%; $p<0.001$). Basophil ($p<0.001$) and eosinophil counts ($p<0.001$) were significantly lower in CSU, while total IgE levels were similar but trended lower in CSU ($p=0.159$).
Conclusion: Aeroallergen sensitization is less prevalent in CSU than AR, suggesting limited atopic involvement in this population, in contrast to several studies in the literature. Optimizing CSU management in this context will necessitate accurate assessment of sensitization profiles, since atopic characteristics appear to be heterogeneous.

Introduction

Chronic spontaneous urticaria (CSU) is a debilitating skin condition characterized by the recurrent appearance of wheals and/or angioedema that persist for six weeks or longer without identifiable external triggers.[1] This disorder represents the most common subtype of chronic urticaria, with a lifetime prevalence of approximately 1.8% and a point prevalence ranging from 0.5% to 1% in the general population.[2] Chronic spontaneous urticaria affects adults, especially females, and can persist for over a year in more than half of cases.[3] Persistent pruritus and unpredictable

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flares, as well as associated symptoms (sleep disturbance and emotional distress), reduce health-related quality of life (HRQoL), often resulting in reduced work productivity, social isolation, and increased healthcare utilization.[4] The level of morbidity is comparable to chronic skin diseases like psoriasis and appears to elevate risks of anxiety and depression.[5]

The pathogenesis of CSU is multifaceted and not fully elucidated, primarily involving inappropriate activation and degranulation of dermal mast cells, leading to the release of vasoactive mediators such as histamine.[6] Autoimmune mechanisms are recognized, including type I autoimmunity and type IIb autoimmunity,[7] which can cause sensory nerve activation, vasodilation, and plasma extravasation, ultimately leading to the hallmark wheals and angioedema.[8] Factors contributing to mast cell degranulation are incompletely understood, particularly in idiopathic cases.[9]

Atopy, defined as a genetic predisposition to develop immunoglobulin E (IgE)-mediated hypersensitivity to common allergens, has been increasingly implicated in CSU susceptibility.[10] Although CSU is traditionally viewed as less atopic compared to classic allergic diseases such as atopic dermatitis or allergic rhinitis (AR), some evidence implicates atopy as an underlying presence.[11] Prior reports have described high rates of aeroallergen sensitization in CSU patients, particularly to weeds, trees, and house dust mites, with a noted association between the presence of AR and positive skin prick test (SPT) results.[12] Similarly, in larger cohorts, positive specific IgE and elevated total IgE levels have been found to be significantly higher in patients with chronic urticaria compared to healthy controls.[13] Understanding this relationship could inform targeted therapies, such as anti-IgE biologics, and improve diagnostic strategies in clinical practice. However, data concerning the atopic features of CSU are inconsistent, particularly regarding the prevalence and clinical implications of aeroallergen sensitization in CSU versus AR populations.

This study aimed to investigate the prevalence and distribution of aeroallergen sensitization in adult CSU patients compared to a control group with AR, utilizing retrospective data from SPT and laboratory evaluations to elucidate potential atopic contributions to CSU pathogenesis and guide future management approaches.

Material and Methods

Ethical considerations

This study was conducted in accordance with the principles outlined in the Declaration of Helsinki and its subsequent amendments. Ethical approval was obtained from the institutional review board of Samsun University Non-Interventional Clinical Research Ethics Committee (Date: 23/7/2025, No: GOKAEK 2025/15/9). The retrospective nature of the study allowed a comprehensive review of existing clinical data.

Study design and participants

This research was designed as a retrospective cross-sectional study and conducted at the Immunology and Allergy Department of Samsun Training and Research Hospital, Samsun, Turkey. The study population included patients with CSU and a control group of patients with AR examined at the Immunology and Allergy outpatient clinic of our hospital between October 15, 2023, and July 1, 2025.

Chronic spontaneous urticaria was defined as the recurrent appearance of wheals, angioedema, or both, lasting for six weeks or longer without identifiable external triggers.[1] Inclusion criteria for the CSU group were: being aged 18 years or older with a confirmed diagnosis, complete records of SPT, and availability of laboratory evaluations within the study period. The control group comprised patients with AR, as evidenced by rhinitis symptoms. All AR patients had undergone SPT for aeroallergen assessment. Exclusion criteria for both groups were: incomplete records, acute urticaria, inducible urticaria subtypes, concurrent systemic illnesses (e.g., active infections or immunosuppression) that could bias sensitization results, presence of rhinitis in patients with chronic spontaneous urticaria, or being younger than 18 years of age.

Data collection

Data were systematically gathered from electronic medical records post-ethical approval, encompassing demographic, clinical, and laboratory parameters to comprehensively assess aeroallergen sensitization and related factors in both CSU and control groups. Demographic information, such as age, sex, height, weight, and body mass index (BMI), was recorded. The presence of asthma was also noted.

Skin prick testing

Aeroallergen sensitization was evaluated using standardized SPTs, administered per European Academy of Allergy and Clinical Immunology (EAACI) guidelines,[14] with a panel covering common allergens, including house dust mites, pollens (grasses, trees, weeds), molds, animal danders (e.g., cat, dog), and others. Positivity was defined as a wheal diameter exceeding the negative control by ≥3 mm after application (15–20 minutes).[15]

Biochemical parameters

Hematological and immunological markers were obtained from routine blood tests performed in our central clinical laboratory. Complete blood count parameters, including white blood cell count, basophil count, neutrophil count, lymphocyte count, eosinophil count, monocyte count, thrombocyte count, and hemoglobin levels, were analyzed using the Sysmex XN-1000 automated hematology analyzer (Sysmex Corporation, Kobe, Japan). Total IgE levels were measured via a BII nephelometer (Siemens Healthineers, Erlangen, Germany).

Endpoints

The primary endpoint focused on the prevalence and distribution of aeroallergen sensitization in CSU patients compared to controls, who had allergic rhinitis.

Statistical analysis

All analyses were performed using IBM SPSS Statistics for Windows, Version 27.0 (IBM Corp., Armonk, NY, USA). The statistical significance value was accepted as p<0.05. Histograms and Q-Q plots were used to examine the conformity of the variables to normal distribution. Descriptive statistics were presented using mean ± standard deviation for normally distributed continuous variables, median (25th percentile–75th percentile) for non-normally distributed continuous variables, and frequency (percentage) for categorical variables. Student's t test was used to analyze normally distributed continuous variables, and the Mann–Whitney U test was used to analyze non-normally distributed continuous variables. The chi-square test or Fisher–Freeman–Halton test were used to analyze categorical variables.

Results

A total of 315 patients were included in the analysis, comprising 149 individuals with CSU and 166 with AR. The CSU group had a median age of 42 years (31–53), which was significantly higher than that of the AR group (p<0.001). Sex distribution showed a female dominance in both groups, with 112 females (75.17%) in the CSU group and 112 (67.47%) in the AR group (p=0.132). Asthma was present in 1 CSU patient (0.67%) versus 24 patients (14.46%) in those with AR (p<0.001) (Table 1).

Basophil and eosinophil counts were significantly lower in the CSU group (p<0.001 for both). Total IgE levels showed a non-significant trend toward lower values in CSU patients (p=0.159). In the CSU group, 54 patients (36.24%) tested positive for aeroallergens, including house dust mite sensitization in 27 (18.12%), pollen in 17 (11.41%), other allergens (such as cat, dog, or mold) in 3 (2.01%), and multiple sensitizations in 7 (4.70%) patients. Overall, 95 (63.76%) patients with CSU were negative in all aeroallergen sensitization tests. The entire AR group exhibited positive results for at least one aeroallergen (0% negative), with higher rates of house dust mite (73, 43.98%), pollen (58, 34.94%), and multiple sensitizations (35, 21.08%) relative to the CSU group (Table 1).

Discussion

Our findings indicate that aeroallergen sensitization is markedly less common in CSU patients than in AR, but with lower yet notable frequencies of sensitization to house dust mites and pollens. Additionally, CSU patients exhibited significantly lower basophil and eosinophil counts, alongside a non-significant trend toward lower total IgE levels, suggesting a limited role for classical atopic mechanisms in CSU pathogenesis. We therefore believe that CSU may emerge

Table 1 Summary of variables with regard to groups.

	Groups		
	CSU (n=149)	AR (n=166)	p
Age, years	42 (31-53)	32 (24-44)	**<0.001**‡
Sex			
Male	37 (24.83%)	54 (32.53%)	0.132§
Female	112 (75.17%)	112 (67.47%)	
Total IgE, IU/mL	120 (55-294)	167.5 (61-342)	0.110‡
WBC (x10^3)	7.10±1.70	7.12±1.84	0.924†
Basophil (x10^3)	0.02 (0.02-0.03)	0.03 (0.02-0.04)	**<0.001**‡
Neutrophil (x10^3)	4.03±1.26	4.04±1.54	0.965†
Lymphocyte (x10^3)	2.22±0.72	2.25±0.57	0.723†
Eosinophil (x10^3)	0.14 (0.08-0.22)	0.20 (0.12-0.34)	**<0.001**‡
Monocyte (x10^3)	0.51±0.16	0.55±0.17	0.064†
Thrombocyte (x10^3)	269.74±59.77	266.52±61.34	0.638†
Hemoglobin, g/dL	13.56±1.66	13.56±1.73	0.998†
Asthma	1 (0.67%)	24 (14.46%)	**<0.001**§
Skin prick test			
Negative	95 (63.76%)	0 (0.00%)*	**<0.001**¶
House mite	27 (18.12%)	73 (43.98%)*	
Pollen	17 (11.41%)	58 (34.94%)*	
Other	3 (2.01%)	0 (0.00%)	
Multiple	7 (4.70%)	35 (21.08%)*	

Descriptive statistics are presented using mean ± standard deviation for normally distributed continuous variables, median (25th percentile - 75th percentile) for non-normally distributed continuous variables and frequency (percentage) for categorical variables.
†Student's t test, ‡Mann Whitney U test, §Chi-square test, ¶Fisher-Freeman-Halton test, *Statistically significant category for the variables with three or more categories. Statistically significant p values are shown in bold.
AR: Allergic rhinitis, CSU: Chronic spontaneous urticaria, IgE: Immunoglobin E, SPT: Skin prick test, WBC: White blood cell.

due to mechanisms other than allergic pathways, which is supported by available recommendations and literature.[1,8]

Aeroallergen sensitization has long been implicated in the pathogenesis of allergic diseases, where exposure to environmental allergens such as house dust mites, pollens, and molds triggers IgE-mediated mast cell activation, leading to type 2 inflammation.[16] In the context of CSU, however, the role of such sensitization remains controversial, with some evidence suggesting it as a predisposing/co-existing factor rather than a direct cause.[17] Studies have shown varying rates of positivity in SPT among CSU patients, and these have often been linked to coexisting atopic conditions.[18] In our study, aeroallergen sensitization was observed in a minority of CSU patients, predominantly to house dust mites and pollens, which was in sharp contrast to the universal positivity in the control group of AR patients. Esmaeilzadeh et al., in a prospective study of 91 CSU patients, reported high aeroallergen sensitization rates, particularly to weeds, trees, and house dust mites, with a significant association with AR presence.[12] Similarly, in a cross-sectional analysis by Chen et al. involving 396 chronic urticaria patients, positive specific IgE was noted in a substantial proportion, exceeding levels in healthy controls.[18] In a retrospective study by Caliskaner et al. evaluating 259 patients with chronic idiopathic urticaria without allergic respiratory disease, immediate cutaneous reactivity to one or more allergens was detected in 27.4%, with house dust mites being the most common (24.7%), significantly higher than in healthy controls but lower than in atopic individuals.[19] These results from multiple studies show that classical atopic pathways may indeed be activated in a subpopulation of patients with CSU; however, the consistently lower frequencies relative to other atopic conditions indicate a divergent pathophysiology. For instance, another cross-sectional study by Cakmak et al., comparing 141 patients with symptomatic dermographism and 220 with CSU, found higher rates of aeroallergen positivity in dermographism patients.[10]

Our results demonstrate lower levels of sensitization in CSU compared to atopic controls, which agrees with the interpretation that the pathophysiological processes differ between CSU and other atopies. Nonetheless, it is crucial to consider the potential biases between studies on this topic. These include regional allergen exposure differences, race- or ethnicity-based variations, and selection biases in studied cohorts. We believe that the current results, taken together with the literature, indicate that sensitization does occur in CSU, but that it might have a non-mechanistic relationship relative to pure respiratory allergies, and that IgE-mediated impacts are secondary phenomena in CSU. Therefore, therapeutic strategies targeting IgE, such as omalizumab, may be more effective in CSU subsets with underlying atopy, which may be a crucial distinction to make regarding the personalization of treatment and to improve management.[20] From a pathophysiological perspective, aeroallergen sensitization in CSU may involve cross-linking of IgE bound to the high-affinity FcεRI receptor on mast cells, leading to degranulation and release of mediators such as histamine, leukotrienes, and prostaglandins, which promote vascular permeability and wheal formation. However, in non-atopic CSU, alternative pathways such as auto-IgE or IgG autoantibodies against FcεRI or IgE may instead take priority, bypassing classical allergen–IgE interactions and resulting in persistent mast cell activation without external influence.[21,22] Autoimmune features in CSU, including antithyroid antibodies, further complicate this relationship.[23]

Comorbid atopic diseases, such as AR and asthma, are frequently observed in CSU, suggesting shared immunological pathways, though their prevalence varies across populations.[9] To ensure evaluation of a pure CSU phenotype without overlapping allergic features, patients with CSU who had concomitant rhinitis were excluded from the present analysis. In our cohort, asthma was significantly less common in patients with CSU relative to the control group with AR, which is supported by lower eosinophil and basophil counts that indicate lower levels of type 2 inflammation. This pattern suggests that CSU in our population is less atopic-driven. It must be noted that females generally have lower eosinophil counts but greater eosinophilic activity.[24] As such, the higher female percentage in the CSU group (despite being statistically non-significant) could explain some of the variance between the groups, and a more accurate analysis would be to assess eosinophilic activity. In a retrospective study by Magen et al., CSU patients showed higher rates of atopy, including AR and asthma, compared to controls, with odds ratios indicating increased comorbidity.[8] In a systematic review by Kolkhir et al., analyzing 39 studies, the prevalence of atopy in CSU ranged from 0% to 75.4%, with higher rates of asthma and AR in CSU than in controls, and sensitization to aeroallergens such as *Dermatophagoides farinae* being more common.[9] Our findings show far lower frequencies of atopic findings/conditions in CSU, which could be attributed to our exclusion of inducible urticaria and focus on spontaneous cases. By demonstrating lower atopic markers in CSU versus AR controls, our study contributes evidence that non-atopic CSU may predominate in certain demographics.[23]

Pathophysiologically, comorbid atopic diseases in CSU may stem from a dysregulated Th2 immune response, where IL-4, IL-5, and IL-13 promote eosinophil recruitment and basophil priming, enhancing mast cell activation.[25] In atopic CSU, this could lead to increased vascular leakage via histamine and platelet-activating factor.[8] However, in our cohort with low atopy, autoimmune mechanisms such as IgG anti-FcεRI antibodies might cross-link receptors, causing basophil hyporesponsiveness and eosinophil apoptosis, as seen in reduced counts, thereby shifting toward type IIb autoimmunity with complement activation and C5a generation, which further stimulates mast cells via C5aR, perpetuating urticarial inflammation independent of atopy.[26]

Hematological and immunological markers, such as eosinophil and basophil counts and total IgE levels, can provide information regarding inflammatory or autoimmune activity.[24,27,28] In CSU, basopenia and eosinophilia have been variably reported, potentially linked to disease activity and response to therapy.[29] Our study revealed significantly lower basophil and eosinophil counts in CSU patients compared to controls, with a trend toward lower total IgE, suggesting decreased type 2 inflammation. This contrasts with some literature where elevated markers are noted. In a cross-sectional study by Jang et al. involving 88 CSU patients, higher serum free IgE levels were found in CSU than in healthy

controls, with atopics showing even higher levels.[30] Indeed, CSU patients have been described as having elevated total IgE in subsets with autoimmunity who showed a favorable response to omalizumab.[31] Another study by Sánchez et al., assessing 91 CSU patients, found IgE autoantibodies against eosinophil peroxidase and cationic protein in 28.8% and 26.6%, respectively, with cross-reactivity to thyroid peroxidase, suggesting autoimmune links.[23] Our lower counts may indicate a predominance of non-eosinophilic, autoimmune-driven CSU, as supported by a systematic review by Maurer et al. in ASSURE-CSU, where eosinophil count was not associated with disease activity and impaired QoL.[21] Lower basophil counts in CSU may result from trafficking to skin lesions, where C5a and chemokines such as CCL2 recruit them, leading to histamine release and wheal persistence. Reduced eosinophils could reflect apoptosis induced by autoantibodies or IFN-γ from Th1-skewed responses in autoimmune CSU, contrasting with Th2-dominant atopy where IL-5 sustains eosinophilia. The trend toward lower total IgE might indicate IgE consumption by autoantibodies or reduced production due to B-cell dysregulation.[11,25,31]

As a retrospective cross-sectional study relying on electronic medical records from a single tertiary center, the analyses and interpretations are prone to selection bias. The absence of urticaria activity scores precludes assessment of sensitization as a factor associated with disease severity or duration. Furthermore, specific IgE assays were not performed to corroborate SPT findings. The differences in age groups are indicative of the anticipated differences among these two disease groups. Although age could indeed alter immune characteristics and allergen response,[24] these are unlikely to have affected the present analyses, since neither group could be considered severely impacted by age-related variations. Additionally, a subgroup analysis comparing CSU patients with and without aeroallergen sensitization was not performed, which could have provided further insights into the clinical and immunological differences between atopic and non-atopic CSU phenotypes. Future studies should explore these distinctions to inform personalized management strategies.

In conclusion, we find that aeroallergen sensitization is less prevalent in CSU patients compared to those with AR. In CSU patients with sensitization, house dust mites and pollens were the primary allergens. CSU also manifested reduced basophil and eosinophil counts, suggesting limited atopic involvement. These findings highlight the need for targeted atopic profiling in CSU to optimize management, particularly in distinguishing atopic cases from those without atopy. We believe prospective multicenter research is warranted to validate and expand upon these observations for improved patient outcomes.

Mandatory Disclosure on Use of Artificial Intelligence

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Conflicts of Interest

The authors had no relevant financial interests to disclose.

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