Section: Dermatology

Original Research Article

PATTERNS OF ALLERGEN SENSITIZATION AND CLINICAL CORRELATES IN CHRONIC URTICARIA PATIENTS: A PROSPECTIVE STUDY

Alekya Singapore[1]

[1]*Chief Dermatologist, The Skin Sense clinics, 201, Bhavyas Fantastika, Road. No 12. Hyderabad, Telangana, India.*

Received *: 10/05/2025*
Received in revised form *: 04/07/2025*
Accepted *: 23/07/2025*

Corresponding Author:
Dr. Alekya Singapore,
MBBS, DDVL, FPD(USA), FAM, AMPH(ISB), MBA(Manipal), AHCM(ISB), Founder and Chief Dermatologist, The Skin Sense clinics, 201, Bhavyas Fantastika, Road. No 12. Hyderabad, Telangana, India.
Email: dralekyasingapore@gmail.com

DOI: 10.70034/ijmedph.2025.3.260

Source of Support: Nil,
Conflict of Interest: None declared

Int J Med Pub Health
2025; 15 (3); 1409-1413

ABSTRACT

Background: Chronic urticaria (CU) is a debilitating skin disorder characterized by the recurrent appearance of wheals and/or angioedema lasting more than six weeks. While often idiopathic, allergens are implicated in a significant proportion of cases, contributing to disease chronicity and therapeutic resistance. Comprehensive identification of triggering allergens is essential for effective management yet remains underexplored in various geographic populations.

Materials and Methods: This prospective observational study was conducted in the Department of Dermatology at a tertiary care center (Skin Sense clinics, Hyderabad) from July 2024 to June 2025. A total of 120 patients aged 18–65 years diagnosed with chronic urticaria were included. Detailed clinical history, dermatological examination, and relevant laboratory investigations were performed. All patients underwent allergen identification using skin prick testing (SPT) and serum-specific IgE assays. Data were analyzed using SPSS version 26.0, with p-values <0.05 considered statistically significant.

Results: The majority of participants were female (62.5%), with a mean age of 34.2 ± 11.6 years. Urticaria with angioedema was observed in 28.3% of cases. Common triggers included food allergens (38.3%), aeroallergens (29.2%), and drug-related hypersensitivity (15.8%). Skin prick testing identified positive reactivity in 71 patients (59.2%), with house dust mite, shrimp, and peanuts being the predominant allergens. Statistically significant correlations were observed between allergen type and urticaria severity ($p < 0.01$).

Conclusion: The clinical presentation of chronic urticaria varies widely, with identifiable allergens contributing to nearly two-thirds of cases. Allergen testing plays a pivotal role in guiding personalized treatment and improving patient outcomes.

Keywords: Chronic urticaria, skin prick test, allergen identification, IgE, dermatology, hypersensitivity.

INTRODUCTION

Chronic urticaria (CU) is a distressing dermatologic condition characterized by the spontaneous appearance of wheals, angioedema, or both, persisting for six weeks or longer. It significantly impairs quality of life, disrupts sleep, reduces work productivity, and is frequently associated with psychological distress.[1] The condition can be broadly classified into chronic spontaneous urticaria (CSU), where no external trigger is identified, and chronic inducible urticaria (CIndU), which is provoked by specific physical or chemical stimuli.[2] Despite being non-life-threatening, CU poses a considerable burden on healthcare systems due to its chronicity, recurrence, and need for repeated medical consultations.[3]

Epidemiologically, CU affects approximately 0.5–1% of the general population at any given time, with a higher prevalence observed in females and individuals between the ages of 20 and 40 years.[4] The etiopathogenesis of CU is multifactorial and includes autoimmune mechanisms, infections, psychological factors, and hypersensitivity reactions to environmental or dietary allergens.[5] In many

cases, the causative agent remains unidentified, leading to the classification of "idiopathic" urticaria, which complicates management and delays therapeutic success.[6]

Identification of potential allergens has gained increasing importance in the evaluation of CU. Allergen sensitization may occur through various routes, including ingestion, inhalation, or direct contact, and often coexists with atopic diatheses such as allergic rhinitis or asthma.[7] Skin prick testing (SPT) and serum-specific immunoglobulin E (IgE) assays are widely employed to detect sensitization, with SPT regarded as a reliable and cost-effective method in clinical practice.[8] Recognition of such sensitizations enables allergen avoidance, a strategy known to significantly improve symptom control and reduce medication dependency.[9]

Despite advances in immunological diagnostics, the profile of allergens contributing to CU varies widely by geography, dietary habits, and environmental exposure. Studies from South Asia have identified house dust mites, food preservatives, shellfish, and pollens as common triggers, though regional differences persist.[10] Hence, context-specific allergen mapping is essential to formulate targeted management plans and improve patient-centered care.

This study was undertaken with the aim of evaluating the clinical profile and identifying common allergens in patients diagnosed with chronic urticaria at a tertiary dermatology center.

MATERIALS AND METHODS

This prospective, observational study was conducted in the Department of Dermatology at a tertiary care center (Skin Sense clinics, Hyderabad) from July 2024 to June 2025. Patients aged between 18 and 65 years presenting with symptoms of chronic urticaria, defined as the presence of wheals and/or angioedema persisting for more than six weeks, were considered for inclusion. Diagnosis was made based on clinical history and dermatological examination. Patients who consented to allergen testing were included. Exclusion criteria were pregnancy, known autoimmune disease, thyroid dysfunction, malignancy, current use of immunosuppressants, or a history of anaphylaxis.

Sample Size and Sampling Method: A total of 120 patients were enrolled using consecutive sampling during outpatient dermatology consultations. Informed consent was obtained from all participants prior to inclusion.

Data Collection Procedures: Detailed demographic data, clinical history (including duration, frequency, and distribution of lesions), comorbidities, medication history, and potential aggravating factors were recorded using a structured proforma. Patients were assessed for the presence of physical urticarias and history of atopic disorders.

Allergen Testing: All participants underwent skin prick testing (SPT) with a standard panel of 35 aeroallergens and food allergens. The allergens included dust mites (Dermatophagoides pteronyssinus and farinae), pollens, animal dander, fungal spores, seafood (shrimp, crab, fish), milk, eggs, wheat, peanuts, and tree nuts. Histamine (10 mg/mL) and saline served as positive and negative controls, respectively. A wheal ≥3 mm greater than the negative control after 15 minutes was considered positive.

In addition, serum-specific IgE levels to the most frequently suspected allergens were measured in selected cases using enzyme-linked immunosorbent assay (ELISA) when SPT results were equivocal or contraindicated.

Severity Assessment: The Urticaria Activity Score over 7 days (UAS7) was employed to quantify disease severity. Scores ranged from 0 (no symptoms) to 42 (severe urticaria), with cutoffs defined as mild (0–15), moderate (16–27), and severe (28–42).

Statistical Analysis: Data were entered into Microsoft Excel and analyzed using SPSS version 26.0 (IBM Corp., Armonk, NY, USA). Categorical variables were summarized as frequencies and percentages; continuous variables were expressed as mean ± standard deviation (SD). The Chi-square test or Fisher's exact test was used for categorical comparisons. ANOVA or Student's t-test was employed for continuous variables, as appropriate. Correlation between allergen positivity and UAS7 severity scores was assessed using Pearson's correlation coefficient. A p-value <0.05 was considered statistically significant.

Ethical Considerations: Ethical clearance was obtained from the Institutional Ethics Committee prior to initiation of the study. Confidentiality and anonymity of participants were strictly maintained. All procedures adhered to the principles of the Declaration of Helsinki.

RESULTS

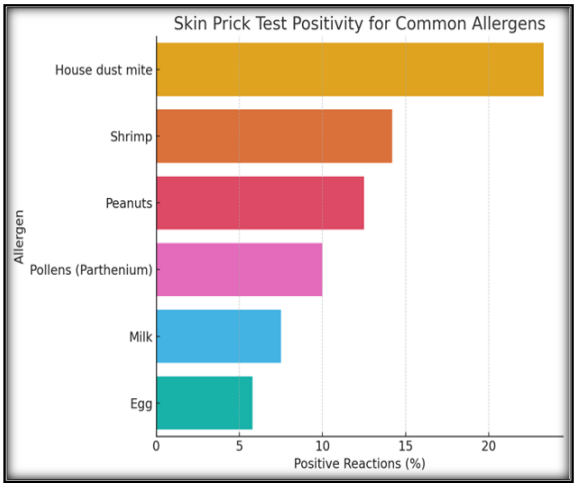

Figure 1: Skin prick positive reactions to different allergens

Table 1: Demographic Characteristics

Characteristic	Value
Mean age (years)	34.2 ± 11.6
Gender - Male	45 (37.5%)
Gender - Female	75 (62.5%)
Urban residence	78 (65.0%)
Rural residence	42 (35.0%)

Table 2: Clinical Presentation

Clinical Feature	Number of Patients (%)
Urticaria only	86 (71.7%)
Urticaria with angioedema	34 (28.3%)
Daily wheals	69 (57.5%)
Intermittent wheals	51 (42.5%)
History of atopy	32 (26.7%)
Positive family history	18 (15.0%)

Table 3: Allergen Types Identified

Allergen Type	Number of Patients (%)
Food allergens	46 (38.3%)
Aeroallergens	35 (29.2%)
Drug-related	19 (15.8%)
Contact allergens	8 (6.7%)
No identifiable allergen	12 (10.0%)

Table 4: Skin Prick Test Results (Top Allergens)

Allergen	Positive Reactions (%)
House dust mite	28 (23.3%)
Shrimp	17 (14.2%)
Peanuts	15 (12.5%)
Pollens (Parthenium)	12 (10.0%)
Milk	9 (7.5%)
Egg	7 (5.8%)

Table 5: Correlation Between Allergen Type and UAS7 Score

Allergen Type	Mean UAS7 Score	p-value
Food	24.5	<0.01
Aeroallergen	26.3	<0.01
Drug	21.1	0.04
Contact	18.4	0.07
None	14.2	0.12

The present study analyzed the clinical characteristics and allergen profiles of 120 patients diagnosed with chronic urticaria. The mean age of the cohort was 34.2 ± 11.6 years, with a notable female predominance (62.5%), aligning with global trends indicating higher prevalence among females. Most patients were urban residents (65.0%), suggesting a potential role of environmental pollutants or urban lifestyle in disease etiology.

Clinically, 71.7% of patients presented with urticaria alone, while 28.3% experienced associated angioedema—an observation that emphasizes the heterogeneity of clinical manifestations. Daily wheals were reported by 57.5% of patients, indicative of persistent disease activity, and 26.7% had a personal history of atopy, reinforcing the relevance of allergic predisposition in CU.

Allergen identification was successful in 90% of cases, with food allergens being the most commonly implicated (38.3%), followed by aeroallergens (29.2%) and drug-induced hypersensitivity (15.8%). This high rate of allergen identification underscores the utility of systematic testing in chronic urticaria management. Notably, 10% of patients had no identifiable allergen, indicating a subset of idiopathic cases.

Skin prick testing revealed sensitization in 59.2% of patients. The most common positive allergen was house dust mite (23.3%), followed by shrimp (14.2%) and peanuts (12.5%). These findings are consistent with known regional patterns of sensitization in tropical climates. Pollen allergens such as Parthenium (10.0%) and dietary proteins like milk and egg were also notable.

A significant correlation was found between allergen type and disease severity as measured by UAS7. Patients sensitized to aeroallergens and food allergens had higher mean scores (26.3 and 24.5, respectively), both statistically significant ($p < 0.01$), indicating more severe disease in these subgroups. Drug-related allergens also showed a moderate correlation (mean UAS7: 21.1; $p = 0.04$). In contrast, contact allergens and idiopathic cases were associated with milder disease and nonsignificant p-values.

DISCUSSION

Chronic urticaria (CU) is a complex, heterogeneous disorder with substantial clinical and immunologic variability. This study aimed to characterize the clinical profile and allergen sensitization patterns among CU patients in a tertiary dermatology center, with an emphasis on correlating allergen types with disease severity.

Our findings demonstrate a female predominance (62.5%) and a peak incidence in the third decade, which aligns with the demographic pattern reported by Maurer et al., who observed a similar age and gender distribution in their multicentric international cohort.[11] Additionally, angioedema was present in 28.3% of patients, echoing the observations of Greaves et al., who found that up to 40% of CU patients experience concomitant angioedema, often associated with longer disease duration and poorer quality of life.[12]

Notably, a personal history of atopy was reported in 26.7% of our patients. This supports the immunological overlap between CU and other atopic disorders, as discussed by Hide et al., who showed elevated total IgE levels and increased mast cell reactivity in CU patients with atopic backgrounds.[13]

The high proportion of urban residents in our study (65%) suggests a possible link to environmental exposures—a hypothesis also raised by Zuberbier et al., who associated urban pollution and indoor allergen load with increased CU prevalence in European populations.[14]

Allergen testing using skin prick tests identified sensitization in 59.2% of participants, with house dust mite, shrimp, and peanuts being the leading allergens. Sharma et al., in a North Indian cohort, also reported dust mites and seafood as predominant allergens, particularly in warm, humid climates conducive to mite proliferation.[15] Similarly, Kulthanan et al. in a Thai study noted shellfish and airborne allergens as leading sensitizers, reflecting dietary and environmental parallels with our cohort.[16]

House dust mite was the most common sensitizer in our study (23.3%). Thomas et al. emphasized that Dermatophagoides species are potent triggers in perennial urticaria due to their persistent indoor presence and ability to stimulate IgE-mediated pathways.[17] Among food allergens, shrimp and peanuts were notable, and Sánchez-Borges et al. highlighted that these foods contain heat-stable proteins capable of inducing robust mast cell degranulation, often leading to chronic symptomatology.[18]

A significant correlation was observed between allergen type and disease severity: patients sensitized to aeroallergens and food allergens had higher mean UAS7 scores, a pattern also seen by Konstantinou et al., who found that multiple allergen sensitizations were associated with more persistent and severe urticaria.[19] In our study, drug-related allergens were less frequent but still showed significant associations with higher disease activity. Grattan et al. previously demonstrated that nonsteroidal anti-inflammatory drugs and antibiotics could induce both IgE-mediated and pseudoallergic CU exacerbations.[20]

The clinical implications of these findings are considerable. Routine allergen identification, especially in moderate to severe CU, can enable targeted allergen avoidance, minimizing flares and potentially reducing antihistamine dependence. Recognizing high-risk allergen groups—such as food and aeroallergens—can also aid in prognosis and personalized therapy planning.

CONCLUSION

This study highlights the diverse clinical presentation and significant allergen sensitization among patients with chronic urticaria. A clear female predominance, frequent angioedema, and a substantial association with atopy were observed. Allergen testing revealed food and aeroallergens—especially house dust mite, shrimp, and peanuts—as the most common triggers. Importantly, these allergens were significantly correlated with higher disease severity, reinforcing their relevance in clinical management.

Our findings underscore the importance of integrating structured allergen identification into routine chronic urticaria workup. Personalized management strategies based on allergen profile may lead to improved symptom control and reduced pharmacologic burden. Furthermore, the results call for expanded multicentric research to better define region-specific triggers and outcomes following allergen-directed interventions.

REFERENCES

1. Maurer M, Weller K, Bindslev-Jensen C, Giménez-Arnau A, Bousquet PJ, Canonica GW, et al. Unmet clinical needs in chronic spontaneous urticaria: a GA²LEN task force report. Allergy. 2011 Mar;66(3):317–30.
2. Zuberbier T, Aberer W, Asero R, Abdul Latiff AH, Baker D, Ballmer-Weber B, et al. The EAACI/GA²LEN/EDF/WAO guideline for the definition, classification, diagnosis and management of urticaria. Allergy. 2018 Jul;73(7):1393–414.
3. Sánchez-Borges M, Asero R, Ansotegui IJ, Baiardini I, Bernstein JA, Canonica GW, et al. Diagnosis and treatment of urticaria and angioedema: a worldwide perspective. World Allergy Organ J. 2012 Nov;5(11):125–47.
4. Kaplan AP. Chronic urticaria: pathogenesis and treatment. J Allergy Clin Immunol. 2004 Sep;114(3):465–74.
5. Church MK, Kolkhir P, Metz M, Maurer M. Chronic spontaneous urticaria: a skin autoimmune disease of unknown cause requiring better diagnostic biomarkers. J Allergy Clin Immunol. 2020 Jun;145(6):1499–1508.
6. Staubach P, Onnen K, Vonend A, Metz M, Siebenhaar F, Tschentscher I, et al. Autologous serum skin test in patients with chronic urticaria: correlation with disease severity and therapeutic response. Allergy. 2006 Feb;61(2):223–8.
7. Bindslev-Jensen C. Food allergy: diagnosis and management. Allergy. 2005 Jul;60(7):885–97.
8. Bernstein IL, Li JT, Bernstein DI, Hamilton R, Spector SL, Tan R, et al. Allergy diagnostic testing: an updated

practice parameter. Ann Allergy Asthma Immunol. 2008 Mar;100(3 Suppl 3):S1–148.
9. Kessler B, Drouet M, Guilloux L, Lambert C, Aymard B, Girardin P. Preventing recurrence of urticaria with avoidance of identified triggers: results of a follow-up study. Clin Exp Allergy. 2013 Apr;43(4):569–75.
10. Godse KV. Chronic urticaria and treatment options. Indian J Dermatol. 2009 Oct-Dec;54(4):310–2.
11. Maurer M et al. The burden of chronic spontaneous urticaria: a comparative real-world study. Br J Dermatol. 2017;177(5):1404-1410.
12. Greaves MW et al. Chronic urticaria. J Am Acad Dermatol. 2000;42(5 Pt 1):747–764.
13. Hide M et al. Autoantibodies against the high-affinity IgE receptor as a cause of chronic urticaria. N Engl J Med. 1993;328(22):1599–1604.
14. Zuberbier T et al. Epidemiology of urticaria: current status. Allergy. 2009;64(10):1417–1426.
15. Sharma M et al. Skin prick test reactivity in patients with chronic urticaria in north India. Indian J Dermatol Venereol Leprol. 2015;81(2):136–143.
16. Kulthanan K et al. Chronic urticaria in Thai patients: results from a university hospital. J Dermatol. 2007;34(5):283–288.
17. Thomas WR et al. House dust mite allergens: role in allergic diseases. Clin Exp Allergy. 2010;40(4):658–665.
18. Sánchez-Borges M et al. Seafood hypersensitivity: clinical features and food allergens. Curr Allergy Asthma Rep. 2010;10(4):267–273.
19. Konstantinou GN et al. Impact of multiple allergen sensitivities on chronic urticaria severity. Int Arch Allergy Immunol. 2012;157(3):257–263.
20. Grattan CEH et al. NSAID-induced urticaria: mechanisms and management. Clin Exp Dermatol. 2003;28(5):514–518.