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Clinical Value Of Dietary Elimination Guided By Serum Food-Specific Ige In Chronic Urticaria And Pruritus: A Prospective Observational Study

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Abstract
Background. *Serum food-specific immunoglobulin E (sIgE) panels are frequently requested during the evaluation of chronic urticaria and persistent pruritus, yet laboratory sensitization correlates poorly with clinically relevant food allergy. International guidelines do not recommend routine food testing in chronic spontaneous urticaria, but elimination diets based solely on panel positivity remain common in everyday practice.*
Objective. *To evaluate the real-world clinical impact of eliminating foods identified by positive serum sIgE testing in patients presenting with chronic urticaria, chronic pruritus, or atopic dermatitis, and to examine how this impact varies with age.*
Methods. *In this prospective observational study conducted during 2024 at a specialized allergy center in Baghdad, 92 patients with chronic urticaria (>6 weeks), chronic pruritus, or atopic dermatitis and food-specific IgE ≥ class 2 were enrolled. Patients positive for the cross-reactive carbohydrate determinant (CCD)/bromelain marker (k202) and those with systemic causes of pruritus or relevant chronic drug use were excluded. Implicated foods were eliminated and clinical response was assessed by patient-reported improvement in itching or urticaria. Outcomes were compared across three pre-specified age strata (1–3, 4–12 and ≥13 years) using the Pearson chi-square test, a Freeman–Halton exact test, and a Cochran–Armitage test for trend; proportions are reported with 95% Wilson confidence intervals (CIs).*
Results. *Improvement after elimination occurred in 15/45 patients aged 1–3 years (33.3%, 95% CI 21.4–47.9%), 6/30 aged 4–12 years (20.0%, 95% CI 9.5–37.3%) and 0/17 aged ≥13 years (0%, 95% CI 0–18.4%); overall 21/92 (22.8%, 95% CI 15.4–32.4%). The association between age group and response was statistically significant ($\chi^2 = 7.98$, $df = 2$, $p = 0.018$; Freeman–Halton exact $p \approx 0.017$), and a monotonic decline with increasing age was confirmed by the trend test ($z = -2.81$, $p = 0.005$). No adolescent or adult improved, and elimination of apple, citrus, sesame, strawberry, tomato, chicken, mixed meat, rice, cacao and yeast produced no observable benefit; 17 patients had rarely or never consumed the implicated foods.*
Conclusions. *Serum food sIgE testing demonstrated limited clinical value in chronic urticaria and pruritus, with measurable benefit confined to a minority of young children and absent beyond early childhood. Positive results frequently reflect asymptomatic sensitization or cross-reactivity rather than clinically significant allergy. Dietary elimination should not be guided by laboratory positivity alone; it must be correlated with a convincing clinical history and, where feasible, confirmed by supervised oral food challenge.*
Keywords: *chronic spontaneous urticaria; food-specific IgE; sensitization; elimination diet; pruritus; cross-reactive carbohydrate determinants; oral food challenge*

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I. Introduction

Chronic urticaria is defined by the recurrence of wheals, angioedema, or both for more than six weeks and is among the most common conditions encountered in dermatology and allergy practice [1]. In the majority of cases no external trigger can be identified, and the disease is classified as chronic spontaneous urticaria (CSU) [1,2]. Contemporary evidence indicates that CSU is predominantly a mast cell–driven disorder of autoimmune or autoallergic origin, frequently involving autoantibodies directed against the high-affinity IgE receptor (FcεRIα) or against IgE itself, rather than a manifestation of food allergy [1,3].

Despite this, food allergy is among the first explanations proposed by patients—and frequently by clinicians—for persistent itching or recurrent wheals. As a result, multi-allergen serum food-specific IgE (sIgE) panels are ordered widely [4,5]. The interpretive difficulty is that a positive sIgE result demonstrates only sensitization—the presence of circulating IgE against a food antigen—and not clinical allergy [5,6]. Many sensitized individuals tolerate the implicated food without any symptoms. Sensitization without disease may arise from immunological recognition that does not trigger effector-cell degranulation, from low-affinity or non-functional IgE incapable of effectively cross-linking FcεRI, from intact oral tolerance maintained by regulatory

T-cell pathways, and from the high analytical sensitivity of modern panels, which routinely detect clinically irrelevant low-level reactivity [5,6].

A further and frequently overlooked source of false positivity is cross-reactivity mediated by cross-reactive carbohydrate determinants (CCDs)—α-1,3-fucose–containing N-glycans shared across plant- and insect-derived glycoproteins. Anti-CCD IgE produces broad, multi-food panel positivity without protein-specific sensitization and, in the great majority of cases, carries no clinical consequence; reactivity to bromelain is a recognized surrogate marker of CCD sensitization and should prompt re-evaluation of the entire panel [7,8]. Pollen-food cross-reactivity (for example, birch pollen sensitization producing positive IgE to apple, hazelnut and stone fruits) is a related phenomenon that inflates panel positivity without conferring true food allergy [5].

The clinical relevance of sIgE is further modulated by age. Food allergy is predominantly a phenomenon of early childhood; oral tolerance to most foods develops through infancy and childhood, and broad panel testing in adolescents and adults therefore carries a high false-positive burden [9,14]. International urticaria and food-allergy guidelines consistently emphasize that true food allergy accounts for only a small minority of chronic urticaria cases and that routine food testing has low diagnostic yield in this setting [1,5,9]. Nevertheless, elimination diets prescribed solely on the basis of laboratory positivity remain common, exposing patients—particularly children—to nutritional and psychosocial harm without demonstrated benefit [15,16].

Against this background, the present study evaluated the real clinical impact of eliminating foods identified through serum sIgE testing in patients presenting with chronic urticaria or persistent itching, and tested the hypothesis that any benefit would be concentrated in the youngest patients and would diminish with increasing age.

II. Methods

Study design and setting

This was a prospective, single-center observational study conducted during 2024 at the Al-Zahraa Specialized Center for Allergy and Respiratory Diseases, Baghdad, Iraq. The study reflects routine clinical practice in a specialized allergy setting and is reported in line with the STROBE recommendations for observational research.

Participants

Consecutive patients presenting with chronic urticaria (wheals and/or angioedema for more than six weeks), chronic pruritus, or atopic dermatitis with itching, and with a positive serum food-specific IgE result (≥ class 2), were enrolled. Inclusion and exclusion criteria are summarized in Table 1.

Inclusion criteria	Exclusion criteria
• Positive serum food sIgE (≥ class 2) • Chronic urticaria (>6 weeks) or persistent pruritus • Atopic dermatitis with itching	• Positive CCD/bromelain cross-reactivity marker (k202) • Systemic causes of pruritus (e.g., renal, hepatic, thyroid, haematological) • Relevant chronic drug use potentially causing pruritus/urticaria

Table 1. Eligibility criteria. Exclusion of CCD/bromelain-positive patients was intended to reduce false-positive panel results driven by cross-reactive carbohydrate determinants.

Food-specific IgE testing

Serum food-specific IgE was measured using the Polycheck multiparameter immunoassay. Consistent with the manufacturer's semi-quantitative scale, only results of class 2 or greater were considered potentially relevant and eligible for elimination. Reactivity to the bromelain/CCD marker (k202) was used to identify and exclude carbohydrate-driven cross-reactivity.

Intervention and outcome

Patients were advised to eliminate all foods corresponding to positive sIgE results. The primary outcome was clinical response, defined as patient-reported improvement in pruritus or urticarial activity following elimination. Responses were recorded as improved or not improved. No standardized severity instrument (e.g., UAS7 for urticaria or SCORAD/EASI for atopic dermatitis) was applied, and oral food challenge was not undertaken.

Statistical analysis

Patients were stratified into three pre-specified, clinically meaningful age groups (1–3, 4–12 and ≥13 years). The association between age group and clinical response was assessed using the Pearson chi-square test. Because one expected cell count was below five, the result was confirmed with a Freeman–Halton exact test. As

age groups are ordered and a dose-response (age-response) relationship was hypothesized a priori, a Cochran–Armitage test for trend was applied as the principal inferential analysis, since it is more powerful than an omnibus chi-square for detecting a monotonic gradient. Pairwise between-group comparisons used the Fisher exact test. Response proportions are presented with 95% Wilson score confidence intervals, which perform well for small samples and proportions near 0%. A two-sided p-value < 0.05 was considered statistically significant. Analyses were performed in Python (SciPy/statsmodels).

III. Results

Ninety-two patients met the eligibility criteria and were included. Overall, 21 of 92 patients (22.8%, 95% CI 15.4–32.4%) reported improvement after dietary elimination. Response rates declined steadily with increasing age and are summarized in Table 2 and Figure 1; participant flow is shown in Figure 2.

Age group	Total (n)	Improved	Not improved	Improvement % (95% CI)
1–3 years	45	15	30	33.3% (21.4–47.9)
4–12 years	30	6	24	20.0% (9.5–37.3)
≥13 years	17	0	17	0% (0–18.4)
Total	92	21	71	22.8% (15.4–32.4)

Table 2. Clinical improvement after dietary elimination by age group, with 95% Wilson confidence intervals.

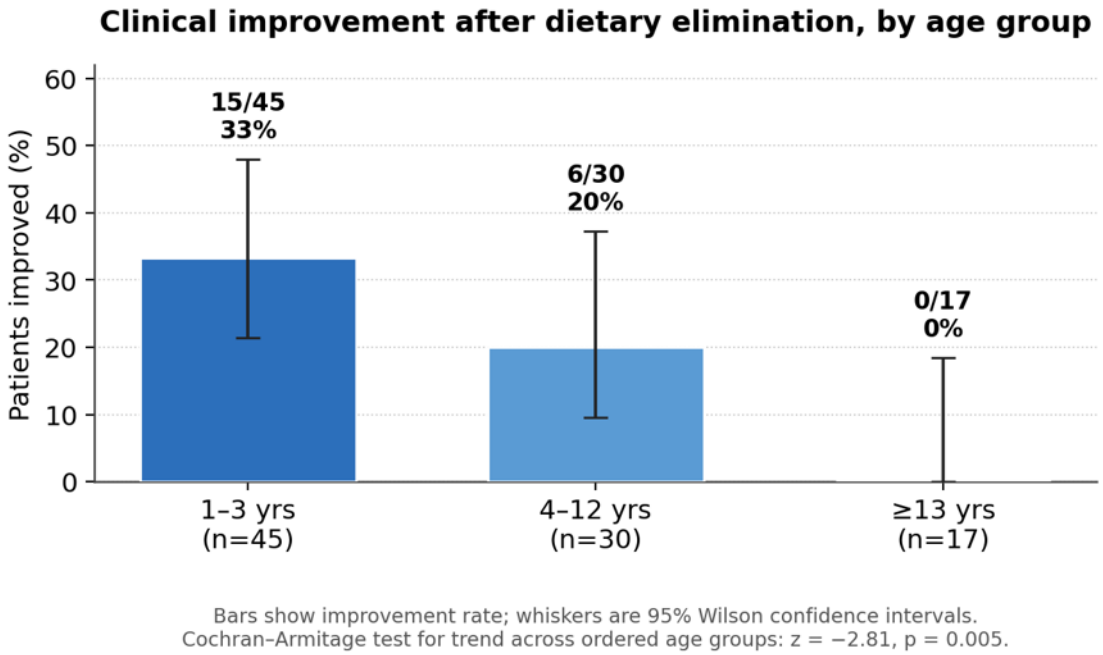

Figure 1. Improvement rate by age group with 95% Wilson confidence intervals. The gradient is significant by the Cochran–Armitage trend test (z = −2.81, p = 0.005).

Statistical comparisons

The overall association between age group and clinical response was statistically significant on the Pearson chi-square test ($\chi^2 = 7.98$, df = 2, p = 0.018). Because one expected cell count was below five, a Freeman–Halton exact test was performed and yielded a concordant result (p ≈ 0.017), indicating that the small-cell limitation did not materially affect the inference. The pre-specified Cochran–Armitage trend test confirmed a significant monotonic decline in response with increasing age (z = −2.81, p = 0.005). In pairwise comparisons, the difference between the youngest (1–3 years) and oldest (≥13 years) groups was significant (Fisher exact p = 0.006), whereas the differences between adjacent groups did not reach significance (1–3 vs 4–12 years, p = 0.29; 4–12 vs ≥13 years, p = 0.07). These statistical results are summarized in Table 3.

Analysis	Statistic	p-value
Pearson chi-square (omnibus, df = 2)	$\chi^2 = 7.98$	0.018
Freeman–Halton exact test (confirmatory)	—	≈ 0.017
Cochran–Armitage test for trend (primary)	z = −2.81	0.005
1–3 vs ≥13 years (Fisher exact)	—	0.006
1–3 vs 4–12 years (Fisher exact)	—	0.29
4–12 vs ≥13 years (Fisher exact)	—	0.07

Table 3. Summary of statistical analyses. The trend test is the most appropriate primary analysis given the ordered age strata and the a priori age-response hypothesis.

Foods eliminated and tolerance history

No adolescent or adult patient (≥13 years) improved after elimination. Removal of a range of commonly implicated foods—apple, citrus, sesame, strawberry, tomato, chicken meat, mixed meat, rice, cacao and yeast—produced no observable clinical improvement in this study. Notably, 17 patients reported that they had either never consumed the implicated food items or consumed them only very rarely (for example, soybean or strawberry), yet these foods had nonetheless registered as positive on the panel—an observation consistent with sensitization or cross-reactivity rather than clinically active allergy.

Figure 2. Participant flow and outcomes by age group

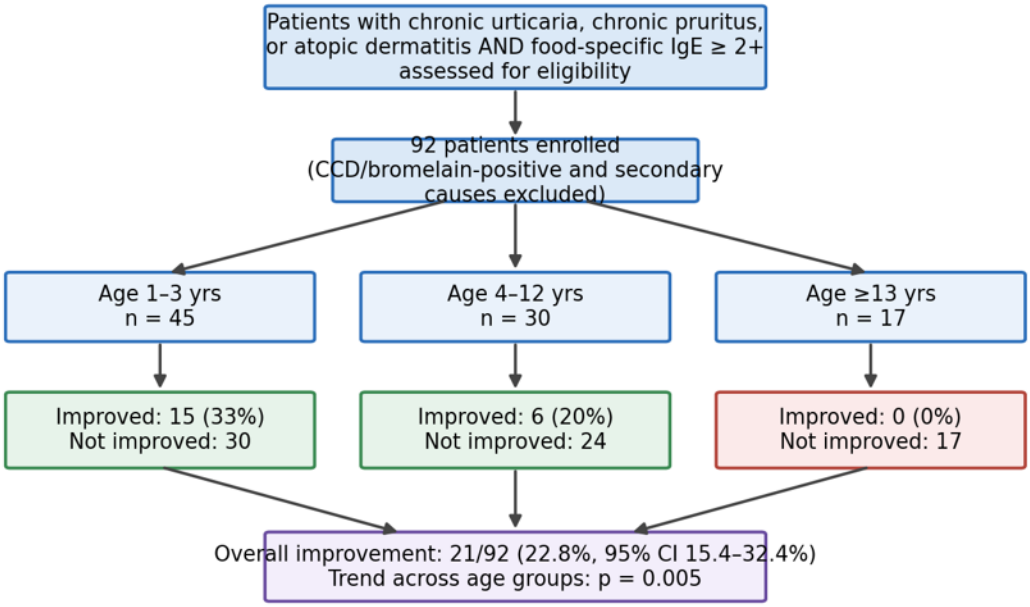

Figure 2. Participant flow and clinical outcomes by age group.

IV. Discussion

In this prospective observational cohort, dietary elimination guided by serum food-specific IgE produced clinical improvement in fewer than one quarter of patients overall, and the benefit was strongly age-dependent: it was greatest in the youngest children, modest in school-age children, and entirely absent in adolescents and adults. The statistically significant downward trend (p = 0.005) mirrors the established biology of food allergy and oral tolerance and reinforces the central message that laboratory sensitization is a poor proxy for clinically relevant disease [1,5,9].

Sensitization is not clinical allergy

The cornerstone of interpretation is the distinction between sensitization and clinical allergy. A positive sIgE test confirms only that the immune system has generated IgE against a food antigen; it does not establish that ingestion will provoke symptoms [5,6]. Several mechanisms explain why strongly sensitized patients may

tolerate a food daily: immunological recognition without effector-cell activation; low-affinity or non-functional IgE that cannot efficiently cross-link FcεRI; robust oral tolerance maintained by regulatory T-cell pathways, which is particularly well developed in older children and adults; and the high analytical sensitivity of contemporary multiplex panels, which detect low-level reactivity of no predictive value [5,6]. The finding that 17 patients had rarely or never eaten the foods to which they tested positive is difficult to reconcile with active, ingestion-driven allergy and is far more consistent with asymptomatic sensitization or cross-reactivity.

Cross-reactive carbohydrate determinants and pharmacologic triggers

Broad, multi-food panel positivity is frequently driven by CCDs rather than by genuine protein-specific sensitization. Anti-CCD IgE is generally clinically silent, and its presence—flagged by reactivity to bromelain—mandates cautious re-interpretation of the whole panel [7,8]. Although a minority of CCD-reactive sera can demonstrate biological activity in vitro, the clinical translation of such reactivity is limited [8]. Excluding bromelain/CCD-positive patients in the present study should have reduced, but cannot be assumed to have eliminated, this source of false positivity. Several of the foods removed without benefit are also recognized histamine-rich foods or histamine liberators (for example, tomato, citrus and strawberry); where these aggravate urticaria, they typically do so through non-immunologic, pharmacologic mechanisms rather than IgE-mediated allergy, which explains why their removal—and the removal of staples such as rice, chicken and yeast—yielded no measurable response in this cohort [1].

The age gradient and the atopic march

The pronounced age effect is biologically coherent. Food allergy is predominantly a disorder of infancy and early childhood, the stage of the atopic march at which epicutaneous sensitization through an impaired skin barrier is most relevant; oral tolerance to most foods is then progressively acquired, such that the diagnostic yield of food testing falls and the false-positive burden rises with age [9,13,14]. The improvement observed in a subset of young children may therefore reflect genuine allergy to common pediatric allergens such as cow's milk and egg [13], whereas the complete absence of benefit in patients aged ≥13 years is consistent with mature oral tolerance and with guideline statements that broad food testing in adolescents and adults is largely uninformative [1,9]. By contrast, most chronic urticaria and most atopic dermatitis are not primarily food-driven: CSU is predominantly idiopathic or autoimmune [1,3], and atopic dermatitis is dominated by skin-barrier dysfunction, microbial colonization and immune dysregulation rather than by dietary antigens.

Risk of misattribution and the case for oral food challenge

Both chronic urticaria and atopic dermatitis fluctuate spontaneously. When elimination coincides with a natural remission, improvement may be wrongly attributed to the dietary change—a classic post hoc ergo propter hoc inference. In the absence of a control group, an unknown proportion of the improvement reported here, particularly in the younger groups, may represent natural fluctuation or placebo response rather than a true treatment effect. This interpretive hazard underscores why the oral food challenge remains the only definitive test capable of confirming or excluding clinically relevant food allergy, and why no laboratory value, however elevated, can substitute for a structured, supervised challenge [5,17].

Harms of unnecessary elimination

Eliminating foods on the basis of laboratory positivity alone is not a benign default. Unnecessary restriction can cause nutritional deficiency and impaired growth in children, generate eating-related anxiety and social restriction, and reduce quality of life [15,16]. Prolonged avoidance may also be counterproductive immunologically: the LEAP randomized trial demonstrated that avoidance of an allergenic food in high-risk infants increased, rather than decreased, the subsequent development of allergy, whereas early introduction was protective [15]. Indiscriminate, panel-driven elimination therefore risks both the established harms of restriction and a paradoxical loss of tolerance.

Clinical implications

Taken together, these findings argue against reflexive food-panel testing and laboratory-driven elimination in chronic urticaria and chronic pruritus, especially in adolescents and adults. A pragmatic interpretive framework is summarized in Table 4: features that genuinely support food allergy—an immediate, reproducible reaction to a single identifiable food, especially in a young child with severe atopic dermatitis—should be distinguished from features that suggest overdiagnosis, such as broad panel positivity in a highly atopic patient without a temporally convincing history. Where the history is compelling, confirmation by supervised oral food challenge should be pursued before durable restriction is imposed [5,17].

Features supporting true food allergy	Features suggesting sensitization / overdiagnosis
• Immediate reaction (≤30–60 min) after a specific food • Reproducible on independent re-exposures • Urticaria, angioedema or systemic features on exposure • Infant/young child with severe atopic dermatitis • Single identifiable culprit food	• Adolescent/adult onset with multiple "allergies" and no clear history • Symptoms inconsistent in timing with food exposure • Broad positive panels in a highly atopic patient • Chronic urticaria attributed solely to diet • Positivity to rarely/never-eaten foods or CCD marker

Table 4. Interpretive framework distinguishing clinically relevant food allergy from asymptomatic sensitization (adapted to the clinical context of this study).

V. Limitations

Several limitations should be considered. First, the absence of a control or comparison group means that natural fluctuation, regression to the mean, and placebo effects cannot be separated from a true effect of elimination; this is the most important constraint on causal interpretation. Second, the primary outcome relied on patient-reported improvement rather than validated severity instruments (such as UAS7, SCORAD or EASI), introducing potential measurement and recall bias and precluding assessment of effect size. Third, oral food challenge—the diagnostic reference standard—was not performed, so neither true allergy nor true tolerance could be confirmed. Fourth, the study was conducted at a single specialized center with a modest sample, particularly in the oldest stratum (n = 17), which limits precision (reflected in the wide confidence intervals) and generalizability. Fifth, component-resolved diagnostics were not used, and residual CCD- or pollen-driven cross-reactivity may persist despite exclusion of bromelain-positive patients. Finally, neither the duration of elimination nor formal reintroduction was standardized. These limitations notwithstanding, the cohort reflects real-world practice in a specialized allergy center and yields an internally consistent, biologically plausible signal.

VI. Conclusion

Serum food-specific IgE testing demonstrated limited clinical value in chronic urticaria and persistent pruritus. Benefit from elimination was confined to a minority of young children and was absent in adolescents and adults, with a significant decline in response across age groups. Positive panel results frequently reflect asymptomatic sensitization or carbohydrate-driven cross-reactivity rather than clinically significant allergy. Dietary elimination should not be guided by laboratory positivity in isolation; decisions must be anchored in a convincing clinical history and, wherever feasible, confirmed by supervised oral food challenge, so that genuine triggers are identified while patients are protected from the avoidable harms of unsubstantiated dietary restriction.

Declarations

Ethics. The study was conducted in accordance with the principles of the Declaration of Helsinki. Patients (or guardians for minors) provided informed consent for the anonymized use of their clinical data. Formal approval/registration details of the institutional review board should be inserted prior to submission.

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Conflicts of interest. The author declares no conflict of interest. [Confirm prior to submission.]

Data availability. De-identified data are available from the author on reasonable request.

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