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Is Omalizumab Monotherapy Sufficient in Chronic Spontaneous Urticaria? Clinical Predictors of Treatment Insufficiency in a Cross-Sectional Cohort

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Abstract

Background: Omalizumab is an established third-line therapy for chronic spontaneous urticaria (CSU); however, a substantial proportion of patients require additional treatments despite standard dosing. Identifying clinical predictors of monotherapy insufficiency remains clinically relevant for optimizing treatment strategies. **Objective:** This paper aims to evaluate the sufficiency of omalizumab monotherapy in CSU patients and to identify clinical and laboratory predictors associated with treatment insufficiency. **Methods:** This cross-sectional study included 50 CSU patients receiving omalizumab therapy. Demographic characteristics, disease duration, angioedema presence, inducible urticaria, treatment parameters, and laboratory biomarkers were analyzed. Patients were stratified according to omalizumab monotherapy response as sufficient or insufficient. Comparative analyses and logistic regression were performed to determine predictors of treatment insufficiency. **Results:** Monotherapy response data were available for 48 patients. Omalizumab monotherapy was sufficient in 26 patients (54.2%) and insufficient in 22 patients (45.8%). Body mass index (BMI) was significantly higher in the monotherapy-insufficient group compared with sufficient responders (median 29.05 vs 25.40 kg/m^2, p = 0.006). Age, sex distribution, disease duration, angioedema presence, inducible urticaria, total IgE levels, inflammatory markers, and thyroid function tests were comparable between groups. Anti-thyroid peroxidase antibody levels were higher in insufficient responders (p = 0.022); however, patient numbers were limited. Most patients received standard 300 mg/month dosing. Dose escalation to 450 - 600 mg was rare and did not result in sufficient

disease control in escalated cases. Multivariate logistic regression identified higher BMI as the only independent predictor of omalizumab monotherapy insufficiency. **Conclusion:** Approximately half of CSU patients required additional therapy despite omalizumab treatment. Elevated BMI was independently associated with monotherapy insufficiency, suggesting a potential impact of adiposity on biologic treatment response. Dose escalation alone may not overcome treatment resistance, underscoring the need for phenotype-driven therapeutic strategies in refractory CSU.

Keywords

Chronic Spontaneous Urticaria, Omalizumab, Monotherapy

1. Introduction

Chronic spontaneous urticaria (CSU) is a debilitating inflammatory skin disorder characterized by the recurrent appearance of wheals, angioedema, or both for longer than six weeks without an identifiable external trigger. The disease significantly impairs quality of life, sleep, and work productivity, and its chronicity often necessitates long-term therapeutic strategies [1] [2].

Current international guidelines recommend a stepwise treatment approach beginning with second-generation H1-antihistamines, followed by dose escalation up to fourfold. In patients who remain symptomatic despite high-dose antihistamines, omalizumab, a recombinant humanized monoclonal anti-IgE antibody, is recommended as third-line therapy [3].

Omalizumab exerts its therapeutic effect by binding circulating IgE, reducing free IgE levels, and downregulating FcεRI receptor expression on mast cells and basophils. This mechanism ultimately suppresses mast cell activation and mediator release, leading to symptom control in CSU [4] [5].

Randomized controlled trials and real-world studies have demonstrated high efficacy rates, with response rates ranging between 60% and 80% at standard dosing [300 mg/month] [6] [7]. Nevertheless, a substantial subset of patients fails to achieve adequate disease control with monotherapy and requires additional therapies such as cyclosporine, systemic corticosteroids, or combination antihistamine regimens [8].

Identifying predictors of insufficient response to omalizumab remains an area of active investigation. Several clinical and laboratory parameters have been proposed, including baseline total IgE levels, disease duration, angioedema presence, autoimmune thyroid disease, and inducible urticaria [9]-[11]. However, findings across studies remain inconsistent.

More recently, metabolic factors—particularly obesity—have emerged as potential modifiers of biologic treatment response. Obesity is associated with chronic low-grade inflammation, altered adipokine signaling, and immune dysregulation, all of which may influence mast cell biology and therapeutic responsiveness [12] [13].

Additionally, omalizumab dosing in CSU is fixed rather than weight-based, raising the possibility that higher body mass index [BMI] may reduce effective drug exposure [14].

Another unresolved clinical question concerns the role of dose escalation in insufficient responders. While some real-world studies suggest benefit from increasing omalizumab doses beyond 300 mg, evidence remains limited and heterogeneous [15].

Given these uncertainties, real-world data evaluating monotherapy sufficiency and its predictors remain clinically valuable.

The present study aimed to evaluate the sufficiency of omalizumab monotherapy in patients with chronic spontaneous urticaria and to identify clinical and laboratory predictors associated with treatment insufficiency, with particular emphasis on body mass index and dose-response relationships.

2. Materials and Methods

2.1. Study Design and Patients

This cross-sectional study was conducted in Izmir City Hospital Dermatology Clinic and included patients diagnosed with chronic spontaneous urticaria (CSU) who were treated with omalizumab. A total of 50 patients receiving omalizumab therapy were retrospectively evaluated through electronic medical records. Patients who had received omalizumab treatment for at least three months and had available clinical follow-up data were included in the study. CSU diagnosis was established according to international guideline criteria and was defined as the spontaneous occurrence of wheals, angioedema, or both for longer than six weeks in the absence of an identifiable external trigger. Monotherapy response data were available for 48 patients, who constituted the primary analytical population.

2.2. Data Collection and Clinical Variables

Clinical, demographic, and laboratory data were extracted using a structured case report form. Recorded demographic and clinical variables included age, sex, height, weight, and body mass index (BMI). Disease-related characteristics comprised disease duration, the presence of angioedema, and the coexistence of inducible urticaria. At omalizumab initiation, all patients were antihistamine-refractory and had received up-dosed second-generation H1-antihistamines according to guideline recommendations. Although standardized disease activity scores (e.g., UAS7 or UCT) were not consistently available due to the retrospective design, all patients had clinically active disease requiring escalation to third-line therapy. Prior treatment intensity was comparable across patients, minimizing potential confounding effects on treatment response classification. Treatment-related parameters included duration of omalizumab therapy, monthly omalizumab dose (300 mg, 450 mg, or 600 mg), and the sufficiency of omalizumab monotherapy. Monotherapy sufficiency was defined as achieving adequate disease control without the need for additional systemic therapy. Omalizumab monotherapy suf-

ficiency was defined as achieving adequate disease control without the need for additional systemic therapy during follow-up. Adequate disease control was operationally defined as the absence of persistent wheals and/or angioedema requiring treatment escalation, as determined by the treating physician. Patients requiring additional therapies (e.g., cyclosporine, systemic corticosteroids, or high-dose antihistamines beyond standard dosing) were classified as monotherapy-insufficient. Treatment response classification was based on clinical evaluation at the third month of omalizumab therapy, consistent with routine clinical practice. Patients requiring concomitant systemic treatment were classified as monotherapy-insufficient. Concomitant therapies were also recorded, including antihistamines, cyclosporine, and systemic corticosteroids.

2.3. Laboratory Assessments

Baseline laboratory parameters were retrieved from patient records. These included total serum immunoglobulin E (IgE), erythrocyte sedimentation rate (ESR), C-reactive protein (CRP), thyroid stimulating hormone (TSH), anti-thyroid peroxidase (Anti-TPO), anti-thyroglobulin (Anti-TG), and complete blood count parameters. Due to the retrospective nature of the study, not all laboratory parameters were available for every patient. Analyses were performed using a complete-case approach, with each variable analyzed based on the number of patients with available data. No imputation methods were applied. Baseline laboratory parameters were defined as values obtained within 4 weeks before or after initiation of omalizumab therapy. When multiple measurements were available, the value closest to treatment initiation was selected for analysis.

2.4. Statistical Analysis

All statistical analyses were performed using IBM SPSS Statistics version 27.0 (IBM Corp., Armonk, NY, USA). The distribution of continuous variables was assessed using the Shapiro-Wilk test, as well as histogram and Q-Q plot visualization. Normally distributed variables were expressed as mean ± standard deviation, whereas non-normally distributed variables were reported as median and interquartile range (IQR). Categorical variables were summarized as frequencies and percentages. Patients were stratified into two groups according to omalizumab monotherapy response: monotherapy-sufficient and monotherapy-insufficient. Between-group comparisons were performed using the independent samples t-test for normally distributed variables and the Mann-Whitney U test for non-normally distributed variables. Categorical variables were compared using the chi-square test or Fisher's exact test where appropriate. To identify independent predictors of omalizumab monotherapy insufficiency, binary logistic regression analysis was conducted. Variables entered into the regression model included body mass index, disease duration, angioedema presence, inducible urticaria, total IgE level, anti-TPO positivity, omalizumab treatment duration, and omalizumab dose. Odds ratios (ORs) and 95% confidence intervals (CIs) were calculated. A two-

tailed p value < 0.05 was considered statistically significant.

3. Results

3.1. Patient Characteristics and Treatment Response

A total of 50 patients with chronic spontaneous urticaria receiving omalizumab therapy were included in the study. Monotherapy response data were available for 48 patients, who constituted the primary analytical cohort. Omalizumab monotherapy achieved adequate disease control in 26 patients (54.2%), whereas 22 patients (45.8%) required additional systemic therapy and were classified as monotherapy-insufficient. Baseline demographic characteristics were comparable between groups. Median age did not differ significantly between monotherapy-insufficient and sufficient patients (46.5 vs 47.5 years, p = 0.41). Female predominance was observed in both groups without statistical significance (63.6% vs 69.2%, p = 0.76). Angioedema was present in the majority of patients overall and did not differ between response groups (77.3% vs 69.2%, p = 0.75). Similarly, the frequency of inducible urticaria was not associated with treatment sufficiency (22.7% vs 38.5%, p = 0.35). Detailed demographic and clinical characteristics are summarized in Table 1.

Table 1. Demographic and clinical characteristics.

Variable	Monotherapy Insufficient (n = 22)	Monotherapy Sufficient (n = 26)	p value
Age, years (median [IQR])	46.5 [43.3 - 53.0]	47.5 [28.3 - 52.0]	0.41
Female, n (%)	14 (63.6%)	18 (69.2%)	0.76
BMI, kg/m^2 (median [IQR])	29.05 [26.55 - 33.40]	25.40 [23.40 - 27.40]	0.006
Disease duration, years	3.0 [2.1 - 18.8]	3.0 [2.0 - 8.5]	0.63
Angioedema present, n (%)	17 (77.3%)	18 (69.2%)	0.75
Inducible urticaria, n (%)	5 (22.7%)	10 (38.5%)	0.35

3.2. Body Mass Index and Clinical Response

Body mass index differed significantly between groups. Patients with monotherapy insufficiency had higher BMI values compared with sufficient responders (median 29.05 kg/m^2 [IQR 26.55 - 33.40] vs 25.40 kg/m^2 [IQR 23.40 - 27.40], p = 0.006). No significant association was observed between disease duration and monotherapy response (p = 0.63). Similarly, omalizumab treatment duration did not differ between groups.

3.3. Laboratory Parameters

Comparative laboratory analyses revealed no statistically significant differences in total serum IgE levels between groups (p = 0.36). Inflammatory markers, including erythrocyte sedimentation rate and C-reactive protein, were comparable between monotherapy-insufficient and sufficient patients (p = 0.75 and p = 0.59,

respectively). Thyroid function tests (TSH) and anti-thyroglobulin antibody levels did not differ significantly. Anti-thyroid peroxidase antibody levels were higher in the monotherapy-insufficient group and reached statistical significance (p = 0.022). However, laboratory availability was limited to a subset of patients. Anti-TPO levels were available in 10 patients, whereas Anti-TG levels were available in 10 patients, and IgE levels were available in 34 patients. These varying sample sizes should be considered when interpreting laboratory comparisons. Laboratory findings are presented in Table 2.

Table 2. Laboratory parameters.

Parameter	Monotherapy Insufficient	Monotherapy Sufficient	p value
Total IgE	282 [204 - 425]	205 [144 - 327]	0.36
ESR	13.5 [7.3 - 23.8]	15 [8 - 18]	0.75
CRP	3.0 [1.0 - 7.6]	2.0 [1.6 - 6.0]	0.59
TSH	1.92 [1.40 - 2.14]	1.71 [1.48 - 2.28]	1.00
Anti-TPO*	309 [300 - 410]	13.9 [13 - 104]	0.022
Anti-TG*	16 [8.6 - 90.5]	20 [17.5 - 32.5]	0.64

3.4. Omalizumab Dose and Treatment Outcomes

The majority of patients received standard omalizumab dosing of 300 mg/month. Among these, 18 patients were classified as monotherapy-insufficient, whereas 26 achieved adequate disease control. Dose escalation to 450 mg or 600 mg was observed in two patients. Both patients underwent dose escalation due to persistent disease activity despite standard dosing. The duration of treatment at higher doses ranged between 3 and 6 months, and both patients were receiving concomitant antihistamine therapy. Despite dose escalation, adequate disease control was not achieved in these cases. Given the small number of patients undergoing updosing, these findings should be considered descriptive rather than inferential.Dose distribution according to treatment response is shown in Table 3.

Table 3. Omalizumab dose distribution.

Dose	Monotherapy Insufficient	Monotherapy Sufficient
300 mg	18	26
450 mg	1	0
600 mg	1	0

3.5. Multivariate Analysis

Binary logistic regression analysis was performed to identify independent predictors of monotherapy insufficiency. Variables entered into the model included BMI, disease duration, angioedema presence, inducible urticaria, total IgE level, anti-TPO positivity, omalizumab treatment duration, and omalizumab dose.

Higher BMI emerged as the only independent predictor of monotherapy insufficiency, whereas other variables did not demonstrate statistical significance.

Overall, these findings indicate that while omalizumab monotherapy provides adequate disease control in approximately half of CSU patients, metabolic factors—particularly increased BMI—may influence treatment sufficiency.

4. Discussion

In this real-world cohort of patients with chronic spontaneous urticaria (CSU), approximately half of the patients required additional therapy despite omalizumab treatment. This finding is consistent with previously reported real-life response rates, which suggest that 30 - 50% of CSU patients may exhibit partial or insufficient response to anti-IgE monotherapy [6] [7].

4.1. BMI as a Predictor of Monotherapy Insufficiency

The most notable finding of our study was the significant association between higher body mass index (BMI) and omalizumab monotherapy insufficiency. Importantly, BMI remained the only independent predictor of insufficient response in multivariate analysis.

Obesity is increasingly recognized as a state of chronic low-grade inflammation characterized by adipokine imbalance, altered cytokine profiles, and immune dysregulation [12]. Adipose tissue secretes pro-inflammatory mediators such as leptin, TNF-α, and IL-6, which may enhance mast cell activation and perpetuate inflammatory signaling pathways relevant to CSU pathogenesis [13].

From a pharmacologic perspective, omalizumab dosing in CSU is fixed rather than weight-adjusted. Consequently, patients with higher BMI may receive relatively lower drug exposure per kilogram of body weight. This pharmacokinetic discrepancy has been proposed as a contributing factor to reduced treatment responsiveness [14].

Although limited CSU-specific data exist, similar obesity-related reductions in biologic efficacy have been reported in psoriasis and other inflammatory diseases, supporting the plausibility of this association.

4.2. Autoimmune Thyroid Disease Signal

We observed higher Anti-TPO levels in monotherapy-insufficient patients. While the number of tested individuals was limited, this finding aligns with the established association between CSU and autoimmune thyroid disease [11].

Autoimmune CSU endotypes, characterized by autoantibodies against FcεRI or IgE, may exhibit different therapeutic responsiveness compared with IgE-driven disease [10]. It is plausible that autoimmune-mediated mast cell activation may be less responsive to anti-IgE blockade alone, necessitating broader immunomodulatory strategies.

Future studies incorporating basophil activation testing and functional autoantibody assays may further clarify this relationship.

4.3. Angioedema and Inducible Urticaria

In our cohort, angioedema and inducible urticaria were not associated with monotherapy sufficiency. The prognostic relevance of these features remains controversial in the literature. Some studies suggest angioedema predicts more severe disease, whereas others, similar to our findings, do not demonstrate a significant impact on biologic response [9].

4.4. Dose Escalation and Therapeutic Resistance

Another clinically relevant observation was the limited efficacy of dose escalation. Only two patients underwent dose escalation to 450 - 600 mg, and both remained insufficient responders. Real-world studies evaluating omalizumab updosing have produced heterogeneous results. While some cohorts report improved response rates, others demonstrate limited benefit [14]-[18]. Our findings support the notion that treatment insufficiency may reflect disease endotype rather than inadequate drug dosing alone. In resistant cases, autoimmune mechanisms, IgE-independent mast cell activation, or alternative inflammatory pathways may predominate, explaining the lack of response to higher anti-IgE dosing.

4.5. Clinical Implications

Taken together, our findings carry several practical implications:

Elevated BMI may serve as a readily accessible clinical predictor of omalizumab monotherapy insufficiency.

Dose escalation alone may not reliably overcome treatment resistance.

Patients with metabolic or autoimmune comorbidities may require earlier consideration of combination or alternative therapies.

These observations underscore the importance of phenotype-driven and potentially endotype-guided treatment strategies in CSU. The limited number of patients undergoing dose escalation precludes definitive conclusions regarding the efficacy of higher dosing strategies. Therefore, our findings should be interpreted as descriptive observations rather than evidence against dose escalation.

In conclusion, our study highlights the multifactorial nature of omalizumab treatment response in CSU. Metabolic status, particularly increased adiposity, appears to play a clinically meaningful role in therapeutic sufficiency. Integrating clinical, immunologic, and metabolic profiling into treatment algorithms may enhance personalized care and optimize outcomes in chronic spontaneous urticaria.

5. Conclusions

In this cohort of CSU patients, approximately half achieved adequate disease control with omalizumab monotherapy. Higher BMI emerged as an independent predictor of monotherapy insufficiency, highlighting a potential role of adiposity in modulating biologic treatment response.

Dose escalation beyond 300 mg/month did not guarantee treatment success in resistant cases, suggesting that treatment insufficiency may reflect disease endo-

type rather than inadequate dosing alone.

Personalized therapeutic approaches considering metabolic status and immunologic phenotype may improve treatment outcomes in chronic spontaneous urticaria.

6. Limitations

Several limitations of this study should be acknowledged. First, the relatively small sample size may limit the generalizability of the findings. Although the cohort reflects real-world clinical practice, larger multicenter studies are needed to validate the observed associations, particularly regarding predictors of omalizumab monotherapy insufficiency.

Second, the cross-sectional and retrospective design inherently carries the risk of selection bias and missing data. Some laboratory parameters, especially autoimmune thyroid markers such as Anti-TPO and Anti-TG, were not available for all patients, restricting the strength of conclusions related to autoimmune endotypes.

Third, disease activity and treatment response were evaluated based on clinical sufficiency rather than standardized scoring systems such as the Urticaria Activity Score over 7 days (UAS7) or Urticaria Control Test (UCT). The absence of validated disease severity indices limits the objectivity of response assessment.

Fourth, pharmacokinetic parameters and serum omalizumab levels were not assessed. Therefore, the relationship between body mass index and effective drug exposure could not be directly evaluated.

Fifth, dose escalation was observed in only a small number of patients, precluding robust statistical analysis regarding the efficacy of higher dosing strategies.

Finally, the study did not evaluate additional biomarkers, such as basophil activation tests or D-dimer levels, which may further refine disease endotyping and treatment prediction.

Despite these limitations, the study provides clinically relevant real-world insights into omalizumab monotherapy sufficiency and highlights the potential role of metabolic factors in treatment responsiveness.

Conflicts of Interest

The authors declare no conflicts of interest regarding the publication of this paper.

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