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Effect of age on omalizumab response in chronic urticaria: a retrospective comparison between adult and geriatric populations

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Abstract
Background: Chronic urticaria (CU) can occur at any age, but comorbidities, age-related immune changes, and polypharmacy may influence treatment outcomes in older adults. Limited data exist on the effect of age on omalizumab efficacy. This study aimed to evaluate the impact of age on treatment response, safety, and clinical-laboratory features in adult (aged <65 years) and geriatric (aged ≥65 years) CU patients receiving omalizumab.
Methods: This retrospective study included 153 CU patients who initiated omalizumab between 2019 and 2024 because of resistance to antihistamines. Patients were categorized by age (<65 or ≥65 years) and by Urticaria Control Test (UCT) scores at the 4th month (responders: UCT ≥12; non-responders: UCT <12). Demographic, clinical, and laboratory parameters were compared between groups.
Results: The median age was 44 years (interquartile range: 35-54), and 63.4% were females. Of all the patients, 130 (85.0%) were aged <65 years and 23 (15.0%) were aged ≥65 years. The overall response rate was 83.7%, with no significant difference between age groups (P = 0.965). Geriatric patients had more comorbidities (P < 0.001), polypharmacy (P = 0.004), and malignancy (P = 0.045). Basophil (P = 0.013) and eosinophil (P = 0.024) counts were lower in the elderly. Among non-responders, autoimmune disease (P = 0.004), elevated anti-thyroglobulin (P = 0.016), and lower baseline UCT scores (P = 0.009) were more frequent. No serious adverse events occurred.
Conclusions: Omalizumab is effective and safe for CU even in older adults. Age, comorbidities, and polypharmacy do not affect treatment response, while autoimmunity and baseline disease activity may predict outcomes.

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Introduction

Urticaria is a skin disorder marked by well-demarcated, pink or red, edematous papules or plaques involving the superficial dermis that blanch with pressure. When deeper skin or mucosal tissues are affected, it is termed angioedema (AE).[1] Urticaria and AE coexist in about half of cases, while 40% present with urticaria alone and 11% with isolated AE.[1-3]

Episodes lasting for <6 weeks define acute urticaria (AU), whereas symptoms persisting >6 weeks on most days define chronic urticaria (CU). Inducible urticaria arises from physical or identifiable triggers, while spontaneous forms lack such triggers.[1-3] Epidemiologic data are limited and variable because of differing definitions and methods. Population-based studies indicate that 15–20% of individuals experience AU at least for one time.[4] CU is less common but clinically significant for its chronicity and treatment resistance, occurring most often between ages of 20–40 years, and twice as frequently in women.[4,5] Reported prevalence of CU ranges from 0.5% to 5%. Chronic spontaneous urticaria (CSU) accounts for approximately 50–75% of cases, and one-third are of physical types.[4-6]

Although CSU is rarely life-threatening condition, it severely impairs quality of life through pruritus, sleep disturbance, and psychiatric comorbidity.[7] Immune dysregulation underlies its pathogenesis via type I (autoallergic) immunoglobulin E (IgE)-mediated and type IIb autoimmune immunoglobulin G (IgG)-mediated mechanisms, which may coexist.[8-10]

First-line therapy is non-sedating second-generation H1-antihistamines up to four-fold standard doses.[1] Refractory cases may use corticosteroids or immunomodulators with limited benefit.[11] Omalizumab, a humanized anti-IgE monoclonal antibody, effectively treats CSU and is a guideline-recommended therapy for antihistamine-refractory patients.[1,12] However, most data concern younger adults, and evidence in older populations remains inconsistent.[13]

Given age-related immune changes, comorbidities, and polypharmacy, treatment response in geriatric CU patients may differ. Older individuals are often underrepresented in clinical trials, making the efficacy and safety of omalizumab in this population uncertain. Therefore, this study aimed to evaluate the impact of age on omalizumab response and safety by comparing adult (aged <65 years) and geriatric (aged ≥65 years) CU patients.

Materials and Methods

Study design and patient selection

This single-center, retrospective cohort study included patients diagnosed with CU, and were initiated omalizumab therapy between 2019 and 2024 at the Allergy and Immunology Outpatient Clinic of a tertiary care center because of antihistamine resistance. A total of 153 adult patients with confirmed CU and complete clinical and biochemical data were analyzed.

Exclusion criteria included: age <18 years; isolated angioedema treated with omalizumab; concurrent omalizumab and cyclosporine therapy; and incomplete treatment (<4 months) or missing follow-up data (e.g., UCT scores).

Demographic, clinical, and laboratory data were retrospectively obtained from electronic medical records.

Data collection and evaluation

Collected data were classified as demographic, clinical, or laboratory variables.

- Demographics: age, gender, and smoking status.
- Clinical: comorbidities, polypharmacy (≥5 drugs), presence of angioedema, defined physical triggers, symptom duration before omalizumab, UCT scores before and at month 4, treatment response (UCT ≥12: responder; <12: non-responder), adverse effects, and relapse after discontinuation.
- Laboratory: total IgE (baseline or follow-up), eosinophil and basophil counts, C-reactive protein (CRP), erythrocyte sedimentation rate (ESR), thyroid function tests (thyroid-stimulating hormone [TSH] and free thyroxine [free T4]), thyroid autoantibodies (anti-thyroglobulin [anti-Tg] and anti-thyroid peroxidase [anti-TPO]), D-dimer, antinuclear antibody (ANA), *Helicobacter pylori* antigen, and autologous serum skin test (ASST) results.

Patient grouping

Analyses were performed by:

- Age: adults (18–64 years) versus geriatrics (aged ≥65 years).
- Response: responders (UCT ≥12 at 4th month) versus non-responders (UCT < 12 at 4th month).Clinical, laboratory, and follow-up data were compared between groups.

Omalizumab treatment protocol and follow-up

All patients received subcutaneous omalizumab 300 mg every 4 weeks as per guidelines. Response was evaluated using UCT scores at baseline and at month 4 of therapy.

Statistical analysis

Statistical analyses were performed using IBM SPSS Statistics version 22.0 (IBM Corp., Armonk, NY, USA). Continuous variables were expressed as median (interquartile range [IQR]) and categorical variables as numbers and proportions.Normality of continuous variables was assessed with the Shapiro–Wilk test. Group comparisons for non-normally distributed continuous data were conducted using the Mann–Whitney U test, while categorical variables were analyzed using the Chi-square (χ^2) or Fisher's Exact test, as appropriate. $P < 0.05$ was considered statistically significant.

Ethical approval

The study was approved by the Non-Interventional Clinical Research Ethics Committee of Necmettin Erbakan University (Meeting No: 255, September 12, 2025; Application ID: 26333). Data were retrospectively evaluated in accordance with the Declaration of Helsinki, ensuring patient confidentiality.

Results

The study included 153 patients with CU, with a median age of 44 years (IQR: 35–54). Of these, 63.4% were females (n = 97) and 36.6% males (n = 56). In 13.7% patients (n = 21), urticarial lesions were triggered by physical stimuli, such as pressure, heat, or cold.

The overall omalizumab response rate was 83.7% (n = 128), and relapse occurred in 32% cases (n = 49). Based on treatment response at the 4th month, patients were divided into two groups: responders (UCT ≥ 12, n = 128; 83.7%) and non-responders (UCT < 12, n = 25; 16.3%).

Non-responders had significantly lower baseline UCT scores than responders (median 4.0, P = 0.009), and post-4th month UCT scores were also markedly lower (P < 0.001) (Tables 1–2). Thyroglobulin antibody (TGAb) levels were significantly higher in non-responders (P = 0.016). A history of autoimmune disease was also more common in this group (P = 0.004).

When age was evaluated in relation to response, median age did not differ significantly between groups—40.0 years (IQR: 33.0–50.0) in non-responders and 45.0 years (IQR: 33.0–52.0) in responders (P = 0.748). No other significant differences were observed between response groups regarding gender, smoking status, comorbidities, malignancy, polypharmacy, angioedema, triggering factors, adverse events, ANA, *Helicobacter pylori* antigen, or ASST positivity. Likewise, there were no differences in symptom duration, total IgE (baseline or follow-up), basophil and eosinophil counts, CRP, ESR, TSH, free T4, thyroid microsomal antibodies (TMAb), or D-dimer levels (Tables 1 and 2).

By age, 130 patients (85.0%) were aged <65 years and 23 (15.0%) were aged ≥65 years. Treatment response rates were comparable: non-responders comprised 17.4% (n = 4) in the ≥65 group and 16.2% (n = 21) in the <65 group (P = 0.965) (Tables 3 and 4).

Comorbidities were present in 40.5% (n = 62). The most common were metabolic/cardiovascular diseases (n = 30; 20.6%), followed by atopic disorders (n = 15; 10.3%), autoimmune diseases (n = 10; 6.8%), and psychiatric disorders or malignancy (each n = 3; 2.06%). One patient (0.68%) was human immunodeficiency virus (HIV)-positive (Table 5). In the ≥65 years group, chronic diseases were significantly more common than in younger adults (61.9% vs. 8.2%; P < 0.001), predominantly reflecting metabolic and cardiovascular comorbidities—consistent with the overall distribution presented in Table 5. Polypharmacy (26.3% vs. 4.0%; P = 0.004) and malignancy (10.5% vs. 0.8%; P = 0.045) were also significantly more frequent in the elderly group. Basophil (P = 0.013) and eosinophil (P = 0.024) levels were also significantly lower in the elderly group (Tables 3 and 4).

Relapse analysis among responders revealed no significant difference between age groups. Similarly, there were no differences between the age groups regarding gender, smoking status, autoimmune disease, angioedema, triggers, adverse events, ANA, *Helicobacter pylori* antigen, ASST positivity, duration of symptoms, baseline and 4th-month UCT scores, or laboratory parameters, including total IgE (baseline and follow-up), CRP, ESR, thyroid tests, TGAb, TMAb, and D-dimer (Tables 3–4).

Discussion

Recent reviews have suggested that advanced age (≥65 years) may be associated with a weaker therapeutic response to omalizumab because of age-related immune and pharmacodynamic alterations. In contrast, this

Table 1 Comparison of categorical clinical and laboratory variables according to omalizumab treatment response (responder vs. non-responder groups).

Variable	Non-responders (n = 25)	Responders (n = 128)	P value
Female	16 (64.0%)	81 (63.3%)	1.000
Male	9 (36.0%)	47 (36.7%)	1.000
Smoking	7 (53.8%)	26 (28.6%)	0.108
Chronic disease	10 (17.5%)	13 (17.1%)	0.955
Autoimmune disease	6 (26.1%)	6 (4.9%)	**0.004**
Malignancy	1 (4.3%)	2 (1.6%)	0.407
Polypharmacy	2 (8.7%)	8 (6.6%)	0.660
Concomitant angioedema	10 (43.5%)	45 (35.7%)	0.489
Triggered by physical stimuli	4 (16.0%)	17 (13.3%)	0.752
Adverse events	0 (0.0%)	1 (0.8%)	1.000
Antinuclear antibody positivity	3 (5.3%)	9 (11.8%)	0.177
Helicobacter pylori antigen positivity	9 (15.8%)	12 (15.8%)	0.996
Autologous serum skin test positivity	3 (5.3%)	6 (7.9%)	0.552

Note: Comorbidity details are provided in Tables 3–5, including age-specific distributions.

Table 2 Comparison of continuous clinical and laboratory variables according to omalizumab treatment response (responder vs. non-responder groups). Values are presented as median (IQR) or mean ± SD.

Variable	Non-responders (n = 25) Median (IQR) or mean ± SD	Responders (n = 128) Median (IQR) or mean ± SD	P value
Age (years)	40.0 (33.0–50.0)	45.0 (33.0–52.0)	0.748
Symptoms duration (months)	12.0 (6.0–48.0)	24.0 (6.0–60.0)	0.491
Baseline UCT† score	4.0 (4.0–5.0)	5.0 (4.0–6.0)	**0.009**
Post-4th dose UCT† score	9.0 (8.0–10.0)	15.0 (13.0–16.0)	**0.000**
Baseline total ımmunoglobulin E (IU/mL; ref.: 0–100)	67.0 (19.0–156.0)	111.0 (56.0–304.2)	0.120
Follow-up total ımmunoglobulin E (IU/mL; ref.: 0–100)	208.0 (50.5–371.5)	270.0 (157.5–579.5)	0.080
Basophil count (×10^9/L; ref.: 0–0.2)	0.02 (0–0.04)	0.03 (0.02–0.05)	0.129
Eosinophil count (×10^9/L; ref.: 0.2–0.6)	0.12 (0.01–0.18)	0.10 (0.04–0.17)	0.970
C-reactive protein (mg/L; ref.: 0–5)	3.0 (1.6–6.0)	3.0 (1.0–7.0)	0.678
Erythrocyte sedimentation rate (mm/h; ref.: 0–20)	10.0 (4.5–17.5)	9.0 (5.0–17.0)	0.968
TSH (mU/L; ref.: 0.27–4.2)	1.9 (1.4–2.6)	1.7 (1.1–2.5)	0.401
Free T4 (ng/dL; ref.: 0.93–1.7)	1.1 (1.0–1.3)	1.2 (1.1–1.3)	0.169
Anti-TPO (U/mL; ref.: 0–34)	10.2 (9.0–13.0)	11.1 (9.0–18.9)	0.549
Anti-Tg (U/mL; ref.: 0–115)	19.0 (15.0–26.8)	15.0 (13.0–15.2)	**0.016**
D-Dimer (ng/mL; ref.: 0–243)	180.2 ± 162.0	360.9 ± 281.2	0.236

UCT: Urticaria Control Test; TSH: Thyroid-Stimulating HormoneFree; T4: Free Thyroxine; TPO: Thyroid Peroxidase; Anti-Tg: Anti-Thyroglobulin Antibody; CRP: C-Reactive Protein; ESR: Erythrocyte Sedimentation Rate.

Table 3 Comparison of categorical clinical variables according to age groups (<65 vs. ≥65 years).

Variable	<65 years (n = 130)	≥65 years (n = 23)	P value
Female	84 (63.6%)	13 (61.9%)	1.000
Male	48 (36.4%)	8 (38.1%)	1.000
Smoking	31 (33.7%)	2 (16.7%)	0.330
Chronic disease	10 (8.2%)	13 (61.9%)	**0.000**
Autoimmune disease	12 (9.5%)	0 (0.0%)	0.367
Malignancy	1 (0.8%)	2 (10.5%)	**0.045**
Polypharmacy	5 (4.0%)	5 (26.3%)	**0.004**
Concomitant angioedema	47 (36.4%)	8 (40.0%)	0.806
Triggered by physical stimuli	17 (12.9%)	4 (19.0%)	0.494
Relapse (after omalizumab discontinuation)	41 (31.1%)	8 (38.1%)	0.616
Treatment response	21 (15.9%)	4 (19%)	0.965
Adverse events	1 (0.8%)	0 (0.0%)	1.000
Antinuclear antibody positivity	10 (8.2%)	2 (9.5%)	0.889
Helicobacter pylori antigen positivity	19 (15.6%)	2 (9.5%)	0.447
Autologous serum skin test positivity	9 (7.4%)	0 (0.0%)	0.233

relationship was not observed in our study.[14] In our study, when patients were grouped according to their response to omalizumab treatment (UCT ≥ 12: responder; UCT < 12: non-responder), age was not found to have a statistically significant effect on treatment response. Similarly, when patients were stratified into <65 years and ≥65 years age groups, no significant difference was observed in response rates between the two groups. This finding was consistent with the results of a study done by Nettis et al., which included 322 patients and reported no significant differences in urticaria activity scores between age groups at weeks 4, 12, and 24 of treatment.[15] These results suggest that age may not be a determining factor in treatment response. On the other hand, a separate study evaluating patients who received only one or two cycles of omalizumab found that the likelihood of maintaining response was lower in individuals aged ≥60 years, compared to younger patients.[16]

In a study conducted by Kitao et al., which included 52 patients, both mean age and median disease duration were found to be lower among patients who achieved complete response, compared to those with partial or no response; however, these differences were not significant statistically.[17] On the other hand, when patients were classified into <65 years and ≥65 years age groups, the response rate to omalizumab was found to be significantly lower in the ≥65 years group, compared to the younger group. The authors attributed this difference to age-related

Table 4 Comparison of clinical and laboratory parameters by age group (<65 vs. ≥65 years). Values are presented as median (IQR) or mean ± SD, as appropriate.

Variable	<65 years (n = 132) Median (IQR) or mean ± SD	≥65 years (n = 21) Median (IQR) or mean ± SD	P value
Symptom duration (months)	24.0 (6.0–60.0)	36.0 (12.0–102.0)	0.180
Baseline UCT score	4.0 (4.0–6.0)	5.0 (4.0–6.0)	0.423
Post-4th dose UCT score	14.0 (12.0–16.0)	15.0 (13.0–16.0)	0.372
Baseline total ımmunoglobulin E (IU/mL; ref.: 0–100)	109.0 (55.0–274.0)	122.5 (27.0–185.2)	0.360
Follow-up total ımmunoglobulin E (IU/mL; ref.: 0–100)	252.5 (157.2–585.2)	113.5 (43.8–324.0)	0.079
Basophil count (×10^9/L; ref.: 0–0.2)	0.03 (0.02–0.05)	0.02 (0–0.03)	**0.013**
Eosinophil count (×10^9/L; ref.: 0.2–0.6)	0.11 (0.0575–0.17)	0.03 (0–0.14)	**0.024**
C-reactive protein (mg/L; ref.: 0–5)	3.0 (1.0–6.0)	3.0 (1.4–6.4)	0.849
Erythrocyte sedimentation rate (mm/h; ref.: 0–20)	10.0 (5.0–16.0)	9.0 (6.0–22.0)	0.668
TSH (mU/L; ref.: 0.27–4.2)	1.7 (1.2–2.5)	2.0 (1.6–2.5)	0.426
Free T4 (ng/dL; ref.: 0.93–1.7)	1.2 (1.0–1.3)	1.1 (1.1–1.2)	0.341
Anti-TPO (U/mL; ref.: 0–34)	11.0 (9.0–16.2)	11.0 (9.2–80.0)	0.513
Anti-Tg (U/mL; ref.: 0–115)	17.0 (15.0–21.9)	13.5 (11.8–89.8)	0.472
D-Dimer (ng/mL; ref.: 0–243)	369.7 ± 267.3	67.7 ± 17.0	0.071

UCT: Urticaria Control Test; TSH: Thyroid-Stimulating Hormone; Free T4: Free Thyroxine; TPO: Thyroid Peroxidase; Anti-Tg: Anti-Thyroglobulin Antibody.

Table 5 Distribution of comorbidity types in the study population.

Type of comorbidity	Number of patients (n)	Percentage (%)
Any comorbidity	62	40.5
Metabolic/cardiovascular	30	20.6
Atopic diseases	15	10.3
Autoimmune diseases	10	6.8
Psychiatric disorders	3	2.06
Malignancy	3	2.06
HIV positive	1	0.68

alterations in pharmacokinetics and pharmacodynamics that may affect treatment response.[17] The same study also documented the literature reporting a higher prevalence of autoimmune urticaria in elderly individuals and proposed that the difference in treatment response may be associated with underlying autoimmunity.[17,18] Furthermore, other potential contributing factors such as higher prevalence of comorbidities and organ dysfunction in older adults were discussed. Nevertheless, Kitao et al. reported no significant differences between age groups with respect to comorbidities, organ failure, or ASST positivity, which they attributed to the limited sample size in their cohort.[17]

In our study, unlike the findings of Kitao et al.,[17] the sample size was larger, and the rates of comorbidities and polypharmacy were significantly higher in the ≥65 years group (P < 0.001 and P = 0.004, respectively). Additionally, our results showed no autoimmune diseases in the ≥65-year group, and there was no significant difference in response to omalizumab treatment between the two age groups (P > 0.05). Interestingly, although autoimmune diseases were significantly more common in the non-responder group overall, none of the elderly patients (aged ≥65 years) had autoimmune comorbidities. This finding differs from previous reports suggesting a higher prevalence of autoimmune urticaria in older individuals. Our observations may reflect sample-size limitations or population-specific characteristics, and larger studies are needed to clarify age-related autoimmune patterns in CSU.

When patients were stratified according to treatment response, no significant differences were found between responders and non-responders in terms of comorbidities or polypharmacy (P > 0.05). These findings suggest that factors, such as age, comorbid conditions, and multiple drug use, may not have a substantial impact on the efficacy of omalizumab.

Our findings contrast with those of Curto-Barredo et al., who suggested that elderly CSU patients may represent a distinct phenotype because of the presence of comorbidities not commonly observed in younger individuals.[19] Similarly, our results do not align with those of Magen et al., who reported lower omalizumab response rates in CSU patients with comorbidities, such as obesity and hypertension.[20] On the other hand, our findings are consistent with the study done by Martina et al., which included 63 patients aged ≥65 years and demonstrated that omalizumab was effective and safe despite the presence of comorbidities.[21]

There are conflicting findings in the literature regarding the effect of age on response to omalizumab treatment. Therefore, larger prospective studies are needed to evaluate the efficacy and safety of omalizumab specifically in the elderly population.When baseline UCT scores were analyzed, scores were found to be significantly lower in the non-responder group (P = 0.009). This suggests that individuals with poor disease control and higher clinical severity may have an increased risk of non-response to omalizumab. Accordingly, in severe CSU cases, initiating treatment more aggressively or considering early dose escalation strategies may be warranted.

Type IIb autoimmune CSU is recognized as a distinct phenotype, typically associated with high disease activity, coexisting autoimmune diseases, positive ASST, low total IgE levels, elevated anti-TPO antibodies, basopenia, eosinopenia, poor response to antihistamines and omalizumab, and favorable response to cyclosporine.[8-10] In our study, some findings suggestive of this phenotype were observed. Notably, autoimmune comorbidities were significantly more common in the non-responder group (P = 0.004), which is a noteworthy observation. While no significant difference was found between the groups in terms of anti-TPO levels, anti-Tg levels were significantly higher among non-responders (P = 0.016). These findings suggest that autoimmunity and associated autoantibodies may influence response to omalizumab. Therefore, assessing autoimmune history and evaluating autoantibody levels prior to treatment may aid in the individualization of therapeutic strategies.

Eosinopenia (<50 cells/μL) and basopenia are observed in approximately 10% of patients with CU. Recent studies have suggested that eosinopenia could be associated with more severe disease, coexisting autoimmunity, and poor response to both antihistamines and omalizumab. Similarly, basopenia has been reported in up to 50% of patients and has been linked to a more severe clinical course.[9,10,22-25]

In the study done by Kitao et al., basophil and eosinophil counts were significantly lower in patients aged ≥65 years compared to those aged <65 years, and this difference was proposed to be potentially associated with reduced omalizumab efficacy in older individuals.[17] Similarly, in our study, basophil and eosinophil levels were also found to be significantly lower in the ≥65 years group, compared to the <65 years group (P = 0.013 and P = 0.024, respectively). However, despite these hematological differences, no statistically significant difference was observed in omalizumab treatment response between the age groups. This suggests that age-related hematological parameters may not always be sufficient to determine clinical response. Therefore, making treatment decisions based solely on eosinophil levels in elderly patients may be misleading. On the other hand, when compared with previous reports linking basopenia and eosinopenia to reduced omalizumab responsiveness, our findings should be interpreted with caution. Our results suggest that differences in patient populations, comorbidity profiles, and sample characteristics may exert multidimensional and interactive effects on treatment response.

Studies in the literature have reported elevated CRP and D-dimer levels in patients with CSU, with significantly higher levels observed in those with severe disease.[26-29] Cugno et al. found that baseline total IgE and D-dimer levels were higher in patients who responded to omalizumab treatment.[30] However, this difference was not observed in our study. This discrepancy may be attributable to patient heterogeneity, methodological differences, or variations in response criteria.

While post-treatment total IgE levels tended to be lower in patients aged ≥65 years compared to younger adults, this difference was not statistically significant. Omalizumab-associated IgE reductions may vary by baseline IgE levels, metabolic clearance, or age-related changes in IgE production. Our findings suggest that the pharmacodynamic impact of omalizumab on IgE may differ slightly with age, although this did not translate into differences in clinical response. Large-scale, prospective studies are needed to identify more reliable biomarkers.

The efficacy and safety of omalizumab in the treatment of CSU have been demonstrated in numerous randomized, placebo-controlled trials,[7,13,31-34] and these findings have been supported by real-world data as well.[35-42] However, data on its safety in elderly individuals are limited. In our study, only one patient experienced mild adverse effects—nausea, fatigue, and dizziness—associated with treatment; no serious systemic reactions, including anaphylaxis, were reported. Additionally, no significant difference in proportions of adverse event was observed between <65- and ≥65-year age groups. Similar findings have been reported in the literature. Narváez-Fernández et al., in a cohort of 89 patients, found no significant difference in adverse events between those aged <70 and ≥70 years, and concluded that omalizumab is safe for elderly individuals.[43]

Conclusion

In conclusion, this study demonstrates that omalizumab is an effective and safe treatment option for CSU in the elderly population. Factors such as age, comorbidities, and polypharmacy were not found to be determinants of treatment response. Moreover, the absence of serious adverse events supports the safe use of omalizumab in older adults. These findings suggest that treatment decisions in elderly patients with CSU should not be based solely on age, comorbid conditions, or polypharmacy. Further large-scale prospective studies are needed to identify the key factors influencing treatment response in the elderly population.

Mandatory Disclosure on Use of Artificial Intelligence

The authors declare that AI-assisted tools were used as follows: Grammarly, for linguistic editing and proofreading. All references have been manually verified for accuracy and relevance.

Author Contributions

All authors contributed equally to this article.

Conflicts of Interest

The authors declared no potential conflict of interest with respect to research, authorship, and/or publication of this article.

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