Allergologia et immunopathologia
Sociedad Española de Inmunología Clínica, Alergología y Asma Pediátrica
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ORIGINAL ARTICLE

OPEN ACCESS

The significance of SIRI and SII scores in predicting the effect of omalizumab treatment in patients with severe allergic asthma

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Received 6 February 2025; Accepted 3 April 2025
Available online 1 July 2025

KEYWORDS
asthma control test;
severe asthma;
Omalizumab;
SIRI;
SII

Abstract
Objective: To evaluate the role of systemic inflammation response index (SIRI) and systemic immune-inflammation index (SII) biomarkers in predicting the response to omalizumab (OMA) treatment in patients with severe allergic asthma.
Methods: A retrospective analysis was conducted to examine the correlation between the fourth-month treatment responses of patients undergoing OMA therapy for severe allergic asthma and their baseline neutrophil, lymphocyte, monocyte, and platelet values, as well as SIRI and SII scores.
Results: Posttreatment asthma control scores had a positive correlation with baseline SIRI ($p = 0.03$, $r = 0.358$), SII ($p = 0.04$, $r = 0.345$), and serum neutrophil values ($p = 0.01$, $r = 0.308$), and a negative correlation with lymphocyte values ($p = 0.00$, $r = -0.398$). Baseline SII showed a negative correlation with posttreatment systemic steroid usage (mg) ($p = 0.04$, $r = -0.247$) and the number of exacerbations ($p = 0.02$, $r = -0.269$).
Conclusion: SIRI and SII scores hold promise for predicting the success of OMA therapy; however, their utility needs to be validated in larger patient cohorts and further studies.

Introduction

Asthma is a heterogeneous, chronic inflammatory disease of the respiratory system, characterized by bronchial hyperreactivity and variable airway obstruction, affecting approximately 10% of adults.[1,2] Severe asthma is defined as the inability to control symptoms despite addressing exacerbating factors and using maximum-dose inhaled corticosteroid/long-acting beta-agonist therapy combined with additional treatments (e.g., tiotropium, leukotriene modifiers, and oral corticosteroids) or symptom exacerbation following the reduction of high-dose inhaled corticosteroid therapy.[2] Severe asthma accounts for 5–10% of all asthma cases[3] and is associated with high morbidity, mortality, and

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https://doi.org/10.15586/aei.v53i4.1325

socioeconomic burden.[4] The cost per patient is ten times higher than that of mild asthma, constituting over 60% of asthma-related healthcare costs.[5]

The heterogeneity of asthma arises from different pathophysiological pathways (endotypes) that manifest as clinically distinct presentations (phenotypes).[6] Inflammatory endotypes include eosinophilic, neutrophilic, mixed, and paucigranulocytic cellular patterns.[7] T2 inflammation predominantly involves eosinophilic dominance, while non-T2 inflammation is characterized by neutrophilic or paucigranulocytic inflammation.[8] T2 asthma encompasses both allergic eosinophilic and nonallergic eosinophilic asthma. In allergic asthma, an immunoglobulin E (IgE)-dependent pathway is active. IgE secreted by B cells binds to type 1 high-affinity IgE receptors (FcεRI) on mast cells and basophils, activating them in an allergen-specific manner.[9] Omalizumab (OMA), used as the fifth-step treatment for severe asthma, inhibits the binding of IgE to FcεRI on mast cells and basophils, thereby preventing mediator release from these cells.[10]

In severe asthma, achieving a balance among safety, efficacy, and cost across all treatment options is critical. Furthermore, the concept of personalized phenotypic interventions is gaining importance. The systemic inflammation response index (SIRI) and the systemic immune-inflammation index (SII) are indices based on neutrophil, lymphocyte, monocyte, and platelet levels. Recent studies suggest these indices can predict OMA treatment responses in chronic spontaneous urticaria.[11] The current study aimed to determine the predictive role of SIRI and SII biomarkers in assessing the response to OMA therapy in severe allergic asthma.

Method

Design and participants

This study involved a single-center retrospective analysis conducted in Adana City, Turkiye on adult patients with severe asthma who were referred to the Severe Asthma Unit of Adana City Training and Research Hospital and were initiated on treatment with biological agents, maintaining therapy for at least 4 months. Data from 38 patients followed in this unit between December 2021 and 2024 were reviewed through the electronic medical records database. The inclusion criteria were being 18 years or older, having been diagnosed with severe asthma according to the Global Initiative for Asthma guidelines[2] (inability to control symptoms despite addressing exacerbating factors and using maximum-dose inhaled corticosteroid/long-acting beta-agonist therapy combined with additional treatments or symptom exacerbation following the reduction of high-dose inhaled corticosteroid therapy) at least 6 months before inclusion, and having been regularly followed up for 6 months in the Severe Asthma Unit prior to the indication for biological therapy. The decision to initiate treatment with biological agents was made by a specialist physician for patients with severe asthma requiring high-dose inhaled corticosteroids plus a second controller and/or systemic corticosteroids to prevent uncontrolled asthma symptoms or in cases in which symptoms remained uncontrolled despite these therapies or worsened upon dose reduction.

Study variables included age, sex, smoking status, pretreatment serum total IgE (IU/L), eosinophil (cells/10^3μL), neutrophil (cells/10^3μL), monocyte (cells/10^3μL), platelet (cells/10^3μL), and basophil (cells/10^3μL) levels; the number of annual exacerbations before and after treatment; asthma control test (ACT) scores (an ACT score of ≥ 20 indicates well-controlled asthma);[12] forced expiratory volume in 1 second (FEV_1) as a percentage and in milliliters, the FEV_1/FVC (forced vital capacity) ratio from pulmonary function tests; and systemic corticosteroid usage. Clinical response to treatment was defined as a ≥50% reduction in the annual number of exacerbations or maintenance oral corticosteroid use, and a super-response was defined as zero exacerbations and no maintenance oral corticosteroid use for asthma.[13] SIRI was calculated using the formula: neutrophil × monocyte/lymphocyte, and SII using the formula: platelet count × neutrophil count/lymphocyte count.

Statistical analysis

Categorical variables were expressed as frequencies and percentages, while continuous variables were presented as means and standard deviations. Fisher's exact test or chi-square test was used to compare categorical variables, and comparisons of continuous variables were conducted using the Student's *t*-test, Wilcoxon signed-rank test, Mann-Whitney U, Kruskal–Wallis test, or analysis of variance test, depending on their distribution and suitability. Logistic regression analysis was performed to evaluate independent variables significantly associated with a full response to each biological agent at the end of follow-up. Statistical significance was defined as $p < 0.05$. Data were analyzed using the Statistical Package for the Social Sciences (SPSS) version 22.0 (IBM Corporation, Armonk, NY, USA).

Ethical approval

Ethical approval for the study was obtained from the Ethics Committee of Adana City Training and Research Hospital (number: 407).

Results

Demographic data

The study included a total of 38 patients, of whom 30 (78.9%) were female, and 8 (21.1%) were male, with a mean age of 51.2 (±12.9) years and an average body mass index of 28.3 (±5.1). Of the patients, 34 (89.5%) had never smoked.

Baseline laboratory and clinical data

The mean pretreatment values for serum eosinophil, platelet, neutrophil, monocyte, and total IgE levels are presented in Table 1. The patients' mean pretreatment

Table 1 Baseline characteristics of the patients included in the study.

Variables	All patients (n = 38)
Sex, n (%)	
Female	30 (78.9)
Male	8 (21.1)
Age, years, mean (SD), (min–max)	51.2 (±12.9), (29–76)
BMI (SD), (min–max)	28.3 (±5.1), (18–38)
Smokers, n (%)	
Never	34 (89.5)
Ex	4 (10.5)
Active	0 (0)
Blood eosinophil count, cells/10^3µL, mean (SD)	373.5 (±432.1)
Blood platelet count, cells/10^3µL, mean (SD)	304 (±99.4)
Blood neutrophil count, cells/10^3µL, mean (SD)	4.9 (±1.3)
Blood lymphocyte count, cells/10^3µL, mean (SD)	2.2 (±0.5)
Blood monocyte count, cells/10^3µL, mean (SD)	0.6 (±0.1)
Total serum IgE, IU/L, mean (SD)	284.8 (±288.8)
SIRI score, mean (SD)	1.6 (±1.1)
SII score, mean (SD)	716.9 (±354.1)
Omalizumab mg/month, mean (SD)	319.8 (±72.8)
Systemic oral corticosteroids in previous year, mg, mean (SD)	188.2 (±81.5)
Systemic oral corticosteroid use, n (%)	68 (%100)
Number of exacerbations in the previous year, mean (SD)	11.4 (±2.9)
ACT score, mean (SD)	7.1 (±1.8)
FEV_1/FVC, %, mean (SD)	64.7 (±11.8)
FEV_1, mL, mean (SD)	1630.8 (±586.9)
FEV_1, %, mean (SD)	58.8 (±19.6)

SD: standard deviation; BMI: body mass index; IgE: immunoglobulin E; SII: systemic immune-inflammation index; SIRI: systemic inflammatory response index; ACT: asthma control test; FVC: forced vital capacity; FEV_1: forced expiratory volume in 1 second.

SIRI was 1.6 (±1.1), and their mean SII was 716.9 (±354.1). The mean annual systemic corticosteroid usage was 188.2 (±81.5) mg, with a systemic corticosteroid usage rate of 100%. The mean annual number of exacerbations before treatment was 11.4 (±2.9), the ACT score was 7.1 (±1.8), FEV_1/FVC was 64.7% (±11.8), FEV_1 was 1630.8 (±586.9) mL, and FEV_1 percentage was 58.8% (±19.6). The mean dose of OMA administered was 319.8 (±72.8) mg.

Comparison of pre- and posttreatment parameters

A significant weak negative correlation was observed between the change in posttreatment FEV_1/FVC percentage and baseline platelet levels ($p = 0.07$, $r = -0.217$). The change in posttreatment FEV_1 percentage had a significant moderate positive correlation with SIRI ($p = 0.00$, $r = 0.412$), SII ($p = 0.00$, $r = 0.614$) and platelet levels ($p = 0.00$, $r = 0.356$); a significant weak positive correlation with neutrophil levels ($p = 0.04$, $r = 0.245$); and a significant moderate negative correlation with lymphocyte levels ($p = 0.04$, $r = -0.398$). The change in posttreatment FEV_1 (mL) demonstrated a significant weak negative correlation with baseline platelet levels ($p = 0.03$, $r = -0.115$) and lymphocyte levels ($p = 0.03$, $r = -0.115$).

Posttreatment ACT scores had a significant moderate positive correlation with baseline SIRI ($p = 0.03$, $r = 0.358$), SII ($p = 0.04$, $r = 0.345$), and neutrophil levels ($p = 0.01$, $r = 0.308$), and a significant moderate negative correlation with lymphocyte levels ($p = 0.00$, $r = -0.398$).

A significant weak negative correlation was identified between posttreatment systemic steroid usage (mg) and baseline SII ($p = 0.04$, $r = -0.247$). Lastly, the number of posttreatment exacerbations had a significant weak negative correlation with baseline SII ($p = 0.02$, $r = -0.269$).

Discussion

Personalized, target-oriented treatment options in severe asthma are continually expanding. Investigating reliable biomarkers to predict the efficacy of OMA therapy in patients with severe allergic asthma is one of the important and current research areas. Biomarkers such as blood and sputum eosinophils, exhaled nitric oxide, and serum IgE are frequently investigated.[14] Studies also suggest that alternative biomarkers could be utilized. SIRI and SII are emerging prognostic markers that have been studied recently to predict the prognosis, mortality rates, and treatment efficacy in various diseases.[15,16] In the current study, these indices were found to have positive correlations with changes

in posttreatment FEV_1% and ACT scores. In addition, SII showed a negative correlation with systemic steroid usage and the number of exacerbations.

The role of systemic inflammation in asthmatic patients is increasingly recognized.[17] Studies indicate elevated and activated peripheral blood neutrophil levels in symptomatic asthmatics.[18] Although these levels decrease following symptom resolution or treatment, they remain higher than in healthy individuals. Neutrophils can release chemotactic factors and preformed granule proteins, attracting monocytes/macrophages to the infection site and causing immune infiltration.[19] Sur et al. reported a higher neutrophil burden compared to eosinophils in the lung tissue of patients who died because of asthma exacerbation.[20] In our study, baseline neutrophil count showed a significant positive correlation with changes in posttreatment FEV_1% and ACT scores. We interpret that patients with higher peripheral blood neutrophil counts are significantly more likely to respond to OMA therapy (Table 2).

Monocytes contribute to airway remodeling and local inflammation in asthmatic patients by secreting various pro-inflammatory factors such as tumor necrosis factor and interleukins.[21] Although no significant correlation was found between baseline monocyte values and the response to OMA therapy in our study, evidence suggests that activated monocytes are increased in the peripheral blood of asthmatic patients and play a direct role in the immunopathology of asthma.[22]

Lymphocytes play a critical role in regulating allergic inflammation in asthma pathogenesis.[23] A relative decrease in lymphocyte count in asthmatic patients may be associated with increased neutrophil and monocyte counts. In our study, baseline lymphocyte counts showed a negative correlation with changes in posttreatment FEV_1%, FEV_1 mL, and ACT scores. This result suggests that lymphocyte count could also serve as a predictive marker for treatment response.

A negative relationship was found between baseline platelet counts and changes in posttreatment FEV_1/FVC ratio and FEV_1 mL in our study. Platelets are multifunctional cells involved in blood clotting and allergic responses through secretory products such as leukotrienes, prostaglandins, platelet-activating factor, serotonin, and histamine.[24] Asthmatic patients are often characterized by hypercoagulability associated with increased platelet counts.[25] In patients with allergic asthma, platelet-specific mediators such as platelet factor 4 and beta-thromboglobulin are elevated in serum, leading to platelet activation.[26] Functional validation studies have demonstrated that platelet depletion significantly alleviates allergic asthma symptoms, underscoring the importance of platelets in allergic asthma.[27]

SII and SIRI serve as superior indicators of the balance between inflammation and immune responses compared to other systemic inflammation markers.[28] Systemic inflammation is the product of circulating immune cells, cytokines, and inflammatory proteins.[29] SII and SIRI are newly defined, easily accessible, and objective indices that encompass four hematological parameters: neutrophils, monocytes, lymphocytes, and platelets.[30] In contrast to a study by

Table 2 Correlations between systemic inflammation indices and investigated variables.

Variables	SIRI	SII	Platelet count cells/10^3µL	Neutrophil count cells/10^3µL	Lymphocyte count cells/10^3µL	Monocyte count cells/10^3µL
FEV_1/FVC change (%)						
p	0.85	0.28	**0.07**	0.5	0.08	0.55
r	0.023	0.132	−0.217	0.083	−0.213	0.073
FEV_1 change (%)						
p	**0.00**	**0.00**	**0.00**	**0.04**	**0.00**	0.90
r	0.412	0.614	0.356	0.245	−0.366	0.014
FEV_1 change (mL)						
p	0.89	0.23	**0.03**	0.81	**0.04**	0.30
r	0.017	0.145	−0.115	−0.028	−0.246	−0.126
ACT						
p	**0.03**	**0.04**	0.90	**0.01**	**0.00**	0.48
r	0.358	0.345	0.015	0.308	−0.398	−0.086
Systemic oral corticosteroid usage						
p	0.13	**0.04**	0.19	0.14	0.25	0.37
r	−0184	−0.247	−0.159	−0.178	0.140	−0.110
Number of exacerbations within the last 4 months						
p	0.12	**0.02**	0.38	0.14	0.11	0.23
r	−0.188	−0.269	−0.107	−0.179	0.193	−0.145

SIRI: systemic inflammatory response index; SII: systemic immune-inflammation index; ACT: asthma control test; FVC: forced vital capacity; FEV_1: forced expiratory volume in 1 second.

Tarkowski et al., which reported no significant difference in SII and SIRI indices between responders and nonresponders to OMA therapy among 46 patients with urticaria,[31] our results demonstrated that SIRI was significantly positively correlated with posttreatment FEV_1% changes and ACT scores and that SII was positively correlated with posttreatment FEV_1% changes and ACT scores while being negatively correlated with systemic steroid usage and the number of exacerbations. Consistent with our findings, a study by Coşansu et al. found that baseline SIRI and SII scores were significantly higher in the group that responded to OMA therapy.[11]

Our study has certain limitations, including the small sample size, the exclusion of long-term outcomes of OMA treatment, and the scarcity of relevant studies in the literature, which limited our ability to perform comprehensive data comparisons.

Conclusion

Personalized targeted therapy in asthma is gaining increasing importance. Predicting treatment efficacy in advance is essential for optimizing efficacy, safety, and cost-effectiveness. According to our study, SIRI and SII scores show promise as predictors of the success of OMA therapy. However, their widespread applicability requires validation in larger patient populations and further studies.

Authors Contributions

Conceived and designed the analysis: SBS, OED; Collected the data: SBS, TS; Performed the analysis: SBS, SK; Wrote the paper: SBS, OED.

Conflict of Interest

The authors declare no potential conflicts of interest with respect to the research, authorship, and/or publication of this article.

Funding

This is study was not supported by any sponsor or funder.

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