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Original Research Article

CORRELATION OF SERUM IGE AND ABSOLUTE EOSINOPHIL COUNT WITH ARIA SEVERITY AND DURATION OF THERAPY IN ADULT PATIENTS WITH ALLERGIC RHINITIS: A PROSPECTIVE HOSPITAL BASED STUDY

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ABSTRACT

Background: Allergic rhinitis is a common IgE-mediated inflammatory disorder of the nasal mucosa characterised by sneezing, rhinorrhoea, nasal obstruction and itching. Although clinical symptoms are commonly used to assess treatment response, objective biomarkers such as serum immunoglobulin E (IgE) and absolute eosinophil count (AEC) may help evaluate inflammatory activity and guide the duration of therapy. The aim is to assess the correlation of serum IgE and absolute eosinophil count with ARIA severity, clinical symptom improvement and duration of therapy in adult patients with allergic rhinitis. **Materials and Methods:** This prospective, single-centre, hospital-based study included 100 patients aged 18–60 years with allergic rhinitis attending the ENT outpatient department of a tertiary care centre in Jharkhand. Patients were classified as mild or moderate/severe according to ARIA guidelines and treated with conventional therapy. Clinical symptoms were assessed using a Visual Analogue Scale (VAS) at baseline and at 4, 8, 12 and 16 weeks. Serum IgE and AEC were measured at baseline, 12 weeks and 16 weeks. Data were analysed using appropriate descriptive and inferential statistical methods, with a p-value of less than 0.05 considered significant. **Results:** The mean age of the study population was 37.34 ± 12.09 years. Females constituted 59% of patients, while 62% belonged to urban areas. Mild allergic rhinitis was present in 52% and moderate/severe disease in 48%. The mean symptom duration was 23.7 ± 14.8 months, and the mean therapy duration was 10.8 ± 3.20 weeks. The mean VAS score decreased from 5.91 at baseline to 1.91 at 12 weeks and 0.94 at 16 weeks, representing an overall reduction of 84.1%. Mean AEC decreased from 716.71 ± 154.36 cells/µL to 501.20 ± 108.04 cells/µL and 429.61 ± 92.62 cells/µL at the corresponding follow-up points, also showing a 40.1% reduction. Most clinical and immunological improvement occurred during the first 12 weeks. **Conclusion:** Serum IgE and AEC decreased progressively in parallel with clinical improvement. These biomarkers, together with ARIA severity and VAS score, may serve as useful supportive tools for monitoring treatment response and determining the appropriate duration of therapy in allergic rhinitis.

INTRODUCTION

Allergic rhinitis is a chronic inflammatory disorder of the nasal mucosa produced by an immunoglobulin E–mediated immune response following exposure to sensitising allergens. It is characterised clinically by recurrent sneezing, watery rhinorrhoea, nasal itching and nasal

obstruction. Ocular symptoms, including itching, redness and excessive watering, may accompany the nasal complaints, while cough, throat irritation, postnasal discharge and sleep disturbances may occur in patients with persistent disease. Although allergic rhinitis is not generally life-threatening, its recurrent and chronic nature can substantially interfere with sleep, concentration, occupational performance, academic activities and overall quality of life. It is also frequently associated with conjunctivitis, sinus-related symptoms and asthma, emphasising the concept of a connected upper and lower airway. Contemporary recommendations therefore consider allergic rhinitis a clinically important airway disorder requiring accurate diagnosis, assessment of severity and structured long-term management rather than treatment based only on occasional symptom relief.[1] The development of allergic rhinitis involves a complex interaction between genetic susceptibility, allergen exposure, epithelial barrier function and type 2 immune inflammation. During the sensitisation phase, inhaled allergens are processed by antigen-presenting cells and presented to T-helper type 2 lymphocytes. These lymphocytes promote B-cell class switching and the production of allergen-specific IgE through cytokines such as interleukin-4 and interleukin-13. Allergen-specific IgE subsequently binds to high-affinity receptors on mast cells and basophils. On re-exposure, allergen-mediated cross-linking of surface-bound IgE leads to the release of histamine, leukotrienes, prostaglandins and other inflammatory mediators. These substances produce the early-phase symptoms of sneezing, itching and watery nasal discharge. A later inflammatory response involving eosinophils, lymphocytes and other cells contributes to persistent congestion, mucosal swelling and increased nasal reactivity.[2]

Allergic rhinitis may be classified according to the temporal pattern and clinical severity of symptoms. The Allergic Rhinitis and its Impact on Asthma framework categorises the condition as intermittent or persistent and as mild or moderate-to-severe. This classification reflects the frequency of symptoms and their effects on sleep, daily activities, work, education and general well-being. Severity assessment is important because patients with apparently similar nasal symptoms may experience very different levels of functional impairment. The clinical history should include the pattern and duration of symptoms, suspected triggers, occupational and environmental exposures, seasonal variation, family history of allergic disease, associated ocular or respiratory symptoms, previous treatment and response to medication. Physical examination and allergy testing may support the diagnosis, although the findings must be interpreted in relation to the clinical presentation because sensitisation alone does not always establish clinically active allergic disease.[3] The ARIA severity classification provides a practical clinical framework, but symptom-based categories may not fully represent the underlying inflammatory burden. Some patients with mild symptoms may demonstrate substantial immunological sensitisation, whereas others with severe symptoms may have overlapping structural, infectious or non-allergic contributors. International consensus recommendations therefore emphasise a comprehensive diagnostic approach that distinguishes allergic rhinitis from non-allergic rhinitis, local allergic rhinitis, chronic rhinosinusitis, medication-related rhinitis and other causes of persistent nasal symptoms. Objective measures that reflect IgE-mediated sensitisation and eosinophilic inflammation may complement clinical assessment, particularly in patients with prolonged disease, uncertain treatment response or discordance between reported symptoms and examination findings. However, biomarkers should be interpreted as supportive components of evaluation and not as isolated substitutes for clinical diagnosis.[4]

Serum IgE is an important component of the allergic response and may be measured as total IgE or allergen-specific IgE. Allergen-specific IgE testing helps identify sensitisation to particular environmental allergens, whereas total serum IgE provides a broader indication of atopic activity. Nevertheless, serum IgE concentrations may be influenced by age, genetic background, parasitic infestation, smoking, environmental exposure and other allergic or immunological conditions. A normal total IgE value does not exclude allergic rhinitis, and an elevated value does not by itself prove that nasal symptoms are allergic in origin. Despite these limitations, serial serum IgE assessment may provide additional information regarding immunological activity when interpreted together with symptom severity, exposure patterns and other inflammatory markers. Its potential usefulness in monitoring treatment response and supporting decisions regarding therapy duration requires further evaluation in defined clinical populations. Eosinophils are major effector cells in type 2 airway inflammation and contribute to tissue injury, mucus production, epithelial dysfunction and persistent nasal hyperresponsiveness. The absolute eosinophil count is a readily accessible and comparatively inexpensive peripheral blood marker. An increased AEC may support the presence of eosinophilic inflammation, although it is not specific to allergic rhinitis and may also be elevated in asthma, parasitic disease, drug reactions, dermatological disorders and systemic eosinophilic conditions. Peripheral eosinophil counts may also vary over time and may not precisely reflect inflammation within the nasal mucosa. The combined assessment of serum IgE and AEC may therefore be more informative than either measurement alone, particularly when correlated with ARIA severity, symptom scores and response to treatment. Recent care pathways increasingly support personalised assessment using clinical

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information, patient-reported outcomes and relevant biomarkers.[5]

Management of allergic rhinitis includes allergen avoidance where feasible, patient education, saline nasal irrigation and pharmacotherapy selected according to symptom pattern and severity. Commonly used medications include oral or intranasal antihistamines, intranasal corticosteroids and, in selected circumstances, leukotriene receptor antagonists or combination intranasal preparations. Intranasal corticosteroids are particularly effective for persistent or moderate-to-severe symptoms because they act on multiple components of nasal inflammation. Treatment should be regularly reassessed to determine adherence, inhaler or spray technique, adverse effects, environmental exposure and the adequacy of symptom control. Step-up or step-down decisions should be individualised rather than based on a uniform fixed duration. Clinical improvement may occur before complete resolution of immunological inflammation, making the identification of reliable objective markers potentially valuable for deciding whether treatment should be continued, modified or gradually withdrawn.[6]

MATERIALS AND METHODS

This was a prospective, single-centre, hospital-based study with planned data collection. The total duration of the study was 24 months. The first four months were devoted to the preparation and approval of the synopsis, followed by 12 months of data collection and six months for data analysis, interpretation, and thesis writing. The study was conducted in the Department of Otorhinolaryngology, Rajendra Institute of Medical Sciences (RIMS), Ranchi, Jharkhand, which is a tertiary care centre. The study population included adult patients of either sex, aged between 18 and 60 years, who presented with symptoms of allergic rhinitis to the ENT outpatient department of Rajendra Institute of Medical Sciences, Ranchi, Jharkhand. The diagnosis and classification of allergic rhinitis were made according to the Allergic Rhinitis and its Impact on Asthma (ARIA) guidelines.

Eligibility Criteria: Patients diagnosed with allergic rhinitis according to the ARIA guidelines were considered eligible for inclusion in the study. Patients of either sex aged between 18 and 60 years who were willing to participate and provide written informed consent were enrolled.

Patients with chronic rhinosinusitis, chronic infective rhinitis, chronic purulent postnasal drip, rhinitis medicamentosa, or nasal polyposis were excluded from the study. Patients with bronchial asthma and those with a history of anti-allergic treatment before enrolment were also excluded. Patients who were unwilling to provide written informed consent, as well as those below 18 years or above 60 years of age, were not included.

Method of Sample Collection and Study Procedure: Patients attending the ENT outpatient department of RIMS, Ranchi, for the management of allergic rhinitis during the study period were screened for eligibility. Patients who fulfilled the inclusion criteria and did not meet any of the exclusion criteria were enrolled after obtaining written informed consent.

All enrolled patients were classified as having mild or moderate-to-severe allergic rhinitis according to the ARIA guidelines. They were prescribed conventional pharmacological treatment for allergic rhinitis in accordance with the ARIA recommendations. The treatment prescribed by the attending physician, including the name, dose, frequency, and duration of each drug, was recorded in the study proforma.

The patients were followed up at four weeks, eight weeks, 12 weeks, and 16 weeks after the initiation of therapy. These follow-up assessments were designated as visit 2, visit 3, visit 4, and visit 5, respectively. Clinical improvement, changes in symptom severity, adherence to treatment, and duration of therapy were assessed during each follow-up visit.

Data Collection: Patients with allergic rhinitis who fulfilled the eligibility criteria were enrolled in the study. Information regarding demographic characteristics, medical history, duration and severity of symptoms, associated clinical conditions, concomitant medications, and previous treatment history was collected using a predesigned study proforma.

A detailed general physical examination, assessment of vital signs, systemic examination, and complete otorhinolaryngological examination were performed. Relevant laboratory investigations and details of the drugs prescribed by the treating physician were also recorded.

The severity of allergic rhinitis symptoms was assessed using a Visual Analogue Scale. The Visual Analogue Scale score was recorded before the initiation of treatment and during each follow-up visit to assess changes in symptoms and response to therapy. Serum total immunoglobulin E and absolute eosinophil count were measured at baseline before the initiation of treatment and subsequently at the 12-week and 16-week follow-up visits. The findings were correlated with the ARIA severity category, clinical response, and duration of therapy.

Statistical Analysis: The collected data were coded and entered into a Microsoft Excel spreadsheet. After checking the data for completeness and accuracy, statistical analysis was performed using the Statistical Package for the Social Sciences software. Categorical variables were presented as frequencies and percentages, whereas continuous variables were expressed as mean with standard deviation or median with interquartile range, depending on the distribution of the data. Appropriate tests of statistical significance were applied according to the nature and distribution of

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the variables. Correlation analysis was performed to determine the relationship of serum immunoglobulin E and absolute eosinophil count with ARIA severity and duration of therapy. A p-value of less than 0.05 was considered statistically significant.

RESULTS

The present study included 100 adult patients diagnosed with allergic rhinitis. The age distribution revealed that the majority of patients belonged to the younger age groups. The highest proportion of patients was observed in the 18–28 years age group, accounting for 32% of the study population, followed by the 29–38 years age group comprising 27%. Together, these age categories represented 59% of all participants, indicating that allergic rhinitis predominantly affected young adults. Patients aged 39–48 years and 49–58 years constituted 18% and 20% of the study population, respectively, while only 3% of patients were between 59 and 60 years of age. The mean age of the study population was 37.34 ± 12.09 years, suggesting that allergic rhinitis is commonly encountered during the most productive years of adulthood. Gender-wise analysis demonstrated a female predominance. Of the total participants, 59 (59.0%) were females, whereas 41 (41.0%) were males, yielding a female-to-male ratio of approximately 1.4:1. [Table 1]

Serum IgE levels were measured at baseline, 12 weeks, and 16 weeks to assess the immunological response to therapy. At the initiation of treatment, the mean serum IgE level was 389.77 ± 127.06 IU/mL, reflecting significant IgE-mediated allergic activity among study participants. Following 12 weeks of treatment, the mean serum IgE level decreased to 268.37 ± 88.96 IU/mL. This represented an absolute reduction of 121.40 IU/mL from baseline, corresponding to a 31.1% decline. At the end of 16 weeks, the mean serum IgE level further decreased to 239.47 ± 76.28 IU/mL. Compared with baseline values, this represented a total reduction of 150.30 IU/mL and an overall decrease of 38.6%. [Table 2]

Absolute eosinophil count (AEC) was assessed as an indicator of eosinophilic inflammation associated with allergic rhinitis. At baseline, the mean AEC was 716.71 ± 154.36 cells/µL, indicating marked eosinophilic activity among the study population. After 12 weeks of treatment, the mean AEC decreased to 501.20 ± 108.04 cells/µL. This corresponded to an absolute reduction of 215.51 cells/µL and a percentage reduction of 30.1% compared with baseline values. By the end of the 16-week follow-up period, the mean AEC further declined to 429.61 ± 92.62 cells/µL. The total reduction from baseline was 287.10 cells/µL, representing an overall decrease of 40.1%. [Table 3]

The combined analysis of serum IgE levels, absolute eosinophil count, and Visual Analogue Scale (VAS) scores demonstrated consistent improvement in both immunological and clinical parameters during the study period. The mean serum IgE level decreased from 389.77 IU/mL at baseline to 268.37 IU/mL at 12 weeks and further to 239.47 IU/mL at 16 weeks, resulting in an overall reduction of 38.6%. Similarly, the mean AEC decreased from 716.71 cells/µL at baseline to 501.20 cells/µL at 12 weeks and 429.61 cells/µL at 16 weeks, also representing an overall reduction of 40.1%. Clinical symptom severity, assessed using the Visual Analogue Scale, showed even more pronounced improvement. The mean VAS score decreased from 5.91 at baseline to 1.91 at 12 weeks and further to 0.94 at 16 weeks, corresponding to an overall reduction of 84.1%. These findings indicate that clinical improvement was accompanied by parallel reductions in immunological markers. [Table 4]

Analysis of interval-wise changes demonstrated that the greatest improvement occurred during the first 12 weeks of treatment. Serum IgE levels showed a reduction of 121.40 IU/mL between baseline and 12 weeks, whereas only an additional reduction of 28.90 IU/mL occurred between 12 and 16 weeks. The total reduction over the entire study period was 150.30 IU/mL. A similar trend was observed for absolute eosinophil count. Between baseline and 12 weeks, AEC decreased by 215.51 cells/µL, while a further reduction of 71.59 cells/µL occurred during the subsequent four weeks. The total reduction from baseline to 16 weeks was 287.10 cells/µL. Clinical symptom severity also improved markedly during the early phase of treatment. [Table 5]

Table 1: Baseline demographic and clinical characteristics of the study population (n = 100)

Variable	Category	Number of patients (n)	Percentage (%) / Mean ± SD
Age group	18–28 years	32	32.0%
	29–38 years	27	27.0%
	39–48 years	18	18.0%
	49–58 years	20	20.0%
	59–60 years	3	3.0%
Age	Mean age	100	37.34 ± 12.09 years
Sex	Female	59	59.0%
	Male	41	41.0%
Residence	Urban	62	62.0%
	Rural	38	38.0%
ARIA severity	Mild allergic rhinitis	52	52.0%
	Moderate/severe allergic rhinitis	48	48.0%
Duration of symptoms	0–12 months	26	26.0%

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	13–24 months	29	29.0%
	25–36 months	35	35.0%
	37–48 months	4	4.0%
	49–60 months	4	4.0%
	61–72 months	2	2.0%
Symptom duration	Mean duration	100	23.7 ± 14.8 months
Therapy duration	Mean duration	100	10.8 ± 3.20 weeks
	Range	100	8–16 weeks

Table 2: Change in serum IgE levels during treatment

Time point	Mean serum IgE	Standard deviation	Absolute reduction from baseline	Percentage reduction
Baseline	389.77 IU/mL	127.06	—	—
12 weeks	268.37 IU/mL	88.96	121.40 IU/mL	31.1%
16 weeks	239.47 IU/mL	76.28	150.30 IU/mL	38.6%

Table 3: Change in absolute eosinophil count during treatment

Time point	Mean AEC	Standard deviation	Absolute reduction from baseline	Percentage reduction
Baseline	716.71 cells/µL	154.36	—	—
12 weeks	501.20 cells/µL	108.04	215.51 cells/µL	30.1%
16 weeks	429.61 cells/µL	92.62	287.10 cells/µL	40.1%

Table 4: Combined changes in serum IgE, AEC and clinical symptom score

Parameter	Baseline mean	Mean at 12 weeks	Mean at 16 weeks	Overall percentage reduction
Serum IgE	389.77 IU/mL	268.37 IU/mL	239.47 IU/mL	38.6%
Absolute eosinophil count	716.71 cells/µL	501.20 cells/µL	429.61 cells/µL	40.1%
VAS score	5.91	1.91	0.94	84.1%

Table 5: Interval-wise reduction in immunological parameters

Parameter	Reduction from baseline to 12 weeks	Reduction from 12 to 16 weeks	Total reduction from baseline to 16 weeks
Serum IgE	121.40 IU/mL	28.90 IU/mL	150.30 IU/mL
Absolute eosinophil count	215.51 cells/µL	71.59 cells/µL	287.10 cells/µL
VAS score	4.00 points	0.97 points	4.97 points

DISCUSSION

The age distribution in the present study demonstrated that allergic rhinitis primarily affected young and middle-aged adults. Of the 100 patients, 32% were aged 18–28 years and 27% were aged 29–38 years; therefore, 59% of patients were younger than 39 years. The mean age was 37.34 ± 12.09 years. These findings are comparable to those of Klossek et al. (2009), whose national survey found a higher prevalence of allergic rhinitis among younger adults: 39% in individuals aged 18–25 years and 35% in those aged 26–35 years, compared with 22% among individuals older than 65 years. Both studies indicate that the burden of allergic rhinitis is greatest during early and middle adulthood, although the present study was hospital-based and reported the distribution of diagnosed cases rather than population prevalence.[7] A female predominance was observed in the present study, with 59 females and 41 males, corresponding to a female-to-male ratio of approximately 1.4:1. A comparable distribution was reported by Erbay et al. (2023), who evaluated 472 patients undergoing skin-prick testing and found that 64.6% were females and 35.4% were males, with a mean age of 33.8 years. The female proportion in that study was slightly higher than the 59% recorded in the present study, while the mean age was relatively close to the present mean of 37.34 years. These findings suggest that adult clinical populations evaluated for allergic rhinitis or aeroallergen sensitisation may show a modest female predominance.[8] Regarding place of residence, 62% of the patients in the present study were from urban areas and 38% were from rural areas. Zheng et al. (2015) similarly reported a slightly higher confirmed prevalence of allergic rhinitis in urban residents than in rural residents, at 7.2% and 6.2%, respectively. Although the difference between urban and rural patients was considerably larger in the present hospital-based sample, the direction of the association was similar. The greater urban representation in the present study may reflect differences in exposure to traffic-related pollutants, indoor allergens, occupational agents, healthcare access, or referral patterns. However, because the present study did not calculate community prevalence or directly measure pollutant exposure, urban residence should be interpreted as a demographic association rather than proof of causation.[9] The present study classified 52% of patients as having mild allergic rhinitis and 48% as having moderate-to-severe disease according to ARIA criteria. Muddaiah et al. (2020), in a study of 160 allergic rhinitis patients, reported mild disease in 66.3%, moderate disease in 7.2%, and severe

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disease in 26.5%; thus, 33.7% had moderate or severe disease compared with 48% in the present study. The present population therefore contained a relatively greater proportion of clinically important moderate-to-severe cases. Muddaiah et al. also reported perennial symptoms in 56% and seasonal symptoms in 44% of patients. In the present study, the mean symptom duration was 23.7 ± 14.8 months, and 70% of patients had experienced symptoms for more than 12 months, supporting the chronic or persistent nature of disease in a substantial proportion of participants.[10] The mean duration of therapy in the present study was 10.8 ± 3.20 weeks, with a range of 8–16 weeks, indicating that treatment was continued until satisfactory clinical and biomarker improvement was achieved. Ellis et al. (2023) evaluated 503 adults with allergic rhinitis and observed significant improvement after only four weeks of treatment: the mean patient-reported VAS score decreased from 6.5 ± 2.4 to 2.6 ± 2.2, while the clinician-reported VAS decreased from 6.6 ± 2.2 to 2.0 ± 2.2. In comparison, the present study demonstrated a decline in mean VAS from 5.91 at baseline to 4.45 at four weeks, 2.89 at eight weeks, 1.91 at 12 weeks, and 0.94 at 16 weeks. The comparatively gradual improvement may be related to differences in treatment regimens, initial disease characteristics and criteria used to continue or discontinue therapy. Nevertheless, both studies demonstrate substantial clinical improvement with regular treatment and structured follow-up.[11] The baseline mean serum IgE level in the present study was 389.77 ± 127.06 IU/mL, which decreased to 268.37 ± 88.96 IU/mL at 12 weeks and 239.47 ± 76.28 IU/mL at 16 weeks. This represented reductions of 121.40 IU/mL at 12 weeks and 150.30 IU/mL at 16 weeks, equivalent to 31.1% and 38.6%, respectively. Gwalabe et al. (2024) reported a mean serum IgE level of 371.24 ± 82.63 IU/mL among patients with allergic rhinitis, which was close to the baseline value of 389.77 IU/mL observed in the present study. They also reported a mean age of 38.65 ± 14.34 years, comparable to the present mean age of 37.34 ± 12.09 years. Thus, the two studies involved clinically similar adult populations with elevated serum IgE, while the serial measurements in the present study additionally demonstrated that IgE declined progressively during treatment.[12] The mean absolute eosinophil count in the present study was 716.71 ± 154.36 cells/µL at baseline. It decreased to 501.20 ± 108.04 cells/µL at 12 weeks and 429.61 ± 92.62 cells/µL at 16 weeks, corresponding to absolute reductions of 215.51 and 287.10 cells/µL and percentage reductions of 30.1% and 40.1%, respectively. Sharma et al. (2019) also demonstrated greater peripheral eosinophilic activity in allergic rhinitis, reporting a mean eosinophil percentage of 4.49% among cases compared with 2.36% among controls. They additionally found a mean serum IgE level of 814.36 IU/mL in allergic rhinitis cases. Although eosinophils were expressed as percentages in their study and as absolute counts in the present study, both findings support the presence of systemic eosinophilic inflammation in allergic rhinitis. The serial decline observed in the present study further suggests that AEC may be useful for monitoring inflammatory response during treatment, although its clinical interpretation should be combined with symptom assessment.[13] A parallel reduction was observed in serum IgE and AEC in the present study. Serum IgE decreased by 38.6%, from 389.77 to 239.47 IU/mL, while AEC also decreased by 40.1%, from 716.71 to 429.61 cells/µL over 16 weeks. Demirjian et al. (2012), in an analysis of 125 patients with rhinitis, reported that serum IgE levels above 140 IU/mL and eosinophil counts above 80 cells/µL were associated with a high probability of an atopic cause. The baseline and 16-week mean values in the present study remained above these proposed thresholds, despite marked reductions during treatment. This suggests that clinical control and reduction of inflammatory activity may occur before complete normalisation of systemic biomarkers. Therefore, serum IgE and AEC may provide supportive objective information, but neither marker should be used independently to determine recovery or treatment cessation without considering symptoms, clinical findings and allergen exposure.[14] Clinical improvement was more pronounced than the changes in immunological markers. In the present study, mean VAS decreased from 5.91 at baseline to 1.91 at 12 weeks and 0.94 at 16 weeks, representing an overall reduction of 4.97 points or 84.1%. Most of this improvement occurred during the first 12 weeks, when the VAS fell by 4.00 points; the additional reduction between 12 and 16 weeks was 0.97 points. Similarly, serum IgE decreased by 117.40 IU/mL during the first 12 weeks and by a further 38.90 IU/mL during weeks 12–16, while AEC decreased by 215.51 and 71.59 cells/µL during the corresponding intervals. Van Weissenbruch et al. (2020) also reported rapid symptom improvement, with mean VAS falling from 73.2 mm at baseline to 57.2 mm on day 1, 46.3 mm on day 3, 36.1 mm on day 7 and 27.2 mm at the final assessment, an overall reduction of approximately 62.8%. The greater 84.1% reduction in the present study may be related to its longer 16-week follow-up. Collectively, these findings show that the largest therapeutic gain occurs early, followed by smaller additional improvements, supporting reassessment at approximately 12 weeks to determine whether therapy should be stopped, modified or extended to 16 weeks.[15]

CONCLUSION

The present study demonstrated that serum IgE and absolute eosinophil count decreased progressively with clinical improvement in adults with allergic rhinitis. Biomarkers IgE levels showed an overall reduction of 38.6% and AEC 40.1% by 16 weeks, while the VAS score decreased by 84.1%. Most

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improvement occurred within the first 12 weeks, with smaller additional benefits up to 16 weeks. Serum IgE and AEC may therefore be useful supportive markers, along with ARIA severity and symptom scores, for monitoring response and guiding the duration of therapy.

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