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ASSESSMENT OF IMMUNOLOGICAL TEST RESULTS IN DIAGNOSING PATIENTS WITH ALLERGIC RHINOSINUSITIS

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ABSTRACT

Allergic rhinosinusitis is an inflammatory condition of the nasal mucosa triggered by exposure to allergens, mediated primarily by immunoglobulin E (IgE). Understanding the underlying pathogenic mechanisms is essential for prescribing effective and targeted therapy [18]. The pathogenesis of allergic rhinosinusitis is based on the development of an IgE-mediated hypersensitivity immune response initiated by allergens penetrating the nasal mucosa. This process occurs in two phases: early and late, which define the nature and progression of clinical symptoms. Both phases involve the activation of mast cells, eosinophils, lymphocytes, and basophils. The initial phase of allergic hyperreactivity is characterized by plasma exudation containing high levels of biologically active substances such as histamine, kinins, and immunoglobulins. Consequently, nasal congestion and rhinorrhea are prominent symptoms in the early phase. During the exudation process, histamine released onto the nasal mucosa irritates nerve endings through interepithelial junctions, causing itching and sneezing in the nose [12,17].

KEYWORDS

Introduction

Atopic diseases such as allergic rhinitis, bronchial asthma, and drug allergies are characterized by IgE-mediated immune responses against specific antigens (allergens). Both environmental factors and genetic predisposition contribute to disease development. IgE-mediated allergic reactions often affect mucous membranes (nasal, conjunctival, respiratory tract, gastrointestinal tract) or the skin, where mast cells coated with IgE molecules are abundant [3]. The primary diagnostic methods for allergies include skin prick tests conducted by trained specialists in allergology clinics. Additionally, levels of total and allergen-specific IgE in blood serum can be measured through various immunological laboratory techniques.

The treatment of allergic rhinosinusitis remains a pressing issue due to the increasing prevalence, exacerbation of allergic processes, occurrence of infectious complications, and the development of polysensitization [11,12,13,14]. Upon initiation of an allergic reaction, plasma cells produce allergen-specific IgE antibodies. Once formed and released into the bloodstream, IgE molecules bind to receptors on mast cells located in the skin, nasal, and respiratory mucosa, providing allergen-specific receptors for subsequent allergen exposure [6].

Regulatory mechanisms controlling IgE synthesis differ from those of other immunoglobulin classes. IgE levels gradually increase during infancy and typically reach adult values by the age of 15–20 years. These IgE levels are usually low, and allergen-specific IgE responses are commonly associated with a marked increase in total serum IgE concentration. It should be noted that even though total IgE levels in healthy individuals are generally low, higher concentrations of antigen-specific antibodies may exist and potentially lead to heightened sensitivity after exposure to corresponding foreign molecules [7].

Objective: To evaluate the role of cytokines IL-4, IL-13, and IgE molecules in the pathogenesis of allergic rhinosinusitis by comparing test results between patients and healthy individuals.

Materials and Methods

The study was conducted at the clinical base of Tashkent Medical Academy. Patients were examined and laboratory tests were performed in the Immunology and Human Genomics Laboratory. During the period from January to December 2022, a total of 80 participants were enrolled, including 60 patients diagnosed with allergic rhinitis (AR) and 28 healthy controls. Among the patients, 24 (48.65%) had intermittent allergic rhinitis (IAR) and 26 (51.35%) had persistent allergic rhinitis (PAR). The mean age of patients with AR was 45.59 ± 16.37 years (range 22–84), while the control group had a mean age of 42.96 ± 15.06 years (range 19–63). The patient group consisted of 32 (53.7%) females and 28 (46.3%) males; the control group comprised 14 (62.1%) females and 6 (37.9%) males. Patients with both forms of AR had not used any antiallergic drugs, antihistamines, topical or systemic corticosteroids, or non-steroidal anti-inflammatory drugs (NSAIDs) for at least one month prior to the study.

Results

Statistical analysis showed that serum IgE levels in patients with AR were higher compared to controls, although the difference was not statistically significant (237.84 ± 38.71 U/ml vs. 175.44 ± 79.89 U/ml, p = 0.474). Similarly, higher IL-4 levels were observed in the patient group compared to controls (29.48 ± 17.77 pg/ml vs. 7.88 ± 4.70 pg/ml, p = 0.188), but this difference was not significant (Figure 1). No significant difference was found in IL-4 levels between patients with AR and healthy controls when measured in plasma (3.02 ± 0.44 pg/ml vs. 2.31 ± 0.12 pg/ml, p = 0.668). Seasonal AR patients showed higher IL-4 levels than those with persistent AR (3.65 ± 0.67 pg/ml vs. 2.39 ± 0.2 pg/ml, p = 0.651).

No statistically significant differences in IL-5 plasma levels were found between AR patients and controls (6.31 ± 1.24 pg/ml vs. 5.61 ± 2.2 pg/ml, p = 0.842). However, patients with intermittent AR tended to have higher IL-5 levels (13.43 ± 4.77 pg/ml) compared to others (3.84 ± 1.81 pg/ml, p = 0.088).

Though not statistically significant, IL-13 levels were lower in controls (5.04 ± 3.75 pg/ml) compared to patients with intermittent AR (15.05 ± 5.77 pg/ml, p = 0.296) and persistent AR (16.76 ± 7.4 pg/ml, p = 0.433). Overweight patients tended to have higher IL-4 plasma levels (61.80 ± 40.0 pg/ml) than normal weight (2.81 ± 0.58 pg/ml, p = 0.160) and underweight patients (10.30 ± 5.59 pg/ml, p = 0.220). IL-13 concentrations were significantly elevated in overweight patients compared to those with normal weight (24.99 ± 8.9 pg/ml vs. 7.57 ± 3.73 pg/ml, p = 0.093).

When comparing patients with AR based on the presence of nasal polyposis, some differences in cytokine levels were noted. Patients with nasal polyposis showed a trend toward lower IL-4 (4.20 ±

1.6 pg/ml) and IL-5 (5.77 ± 1.3 pg/ml) levels compared to those without polyposis (IL-4: 35.00 ± 21.59 pg/ml, p = 0.165; IL-5: 8.8 ± 3.66 pg/ml, p = 0.459) (Figure 5). Conversely, IL-13 levels were higher in patients with nasal polyposis compared to those without (31.43 ± 17.68 pg/ml vs. 13.69 ± 4.55 pg/ml, p = 0.493).

Discussion

Interaction between environmental allergens and specific IgE antibodies is a key factor in allergic airway reactions and represents a major pathogenic mechanism triggering allergic rhinitis and bronchial asthma symptoms. The initial immunopathogenic response required for disease onset is increased production of IgE antibodies specific to environmental allergens such as house dust and pollen.

Several studies have shown that IgE production is regulated by antigen-specific helper and suppressor T cells and is influenced by isotype-specific factors. Regulation of IgE synthesis involves Th2 cells and cytokines produced by B lymphocytes, which play a critical role in initiating and enhancing specific IgE antibody production, explaining the dominance of Th2 helper cells in allergic conditions [8].

Ohashi et al. reported no significant difference in total IgE levels among healthy individuals, atopic patients during clinical symptoms, and atopic patients outside clinical periods [10]. According to Finkelman et al., allergen-specific IgE levels are more relevant in allergic diseases than total IgE. IL-4 plays a crucial role in inducing and amplifying primary polyclonal and secondary IgE responses by B lymphocytes [11].

Li et al. demonstrated that nasal lavage fluid from patients showed significantly elevated levels of both total and specific IgE compared to healthy controls [12]. Our study did not find statistically significant differences in serum total IgE between patients with AR and controls, although approximately 25% of controls had low levels of antibodies.

Ohashi and colleagues also studied IL-4 involvement in modulating atopic responses and treatment effects, reporting decreased plasma IL-4 and IgE concentrations after subcutaneous immunotherapy in patients with AR [15]. Other authors confirmed that immunotherapy reduces IL-4 and IgE levels regardless of clinical symptom severity [16,17].

Our results indicate elevated serum IL-4 in AR patients compared to controls, with higher levels in seasonal AR than in persistent AR patients, suggesting increased inflammatory activity driven by pollen allergens. Additionally, overweight patients showed higher IL-4 levels compared to normal and underweight subjects.

Several studies reported increased IL-4 and IL-13 in nasal secretions of patients with allergic rhinitis. For example, Scavuzzo et al. found significantly elevated IL-4 levels in AR patients compared to healthy individuals [19]. However, some literature notes no increase in IL-4, IL-6, IL-8, leukotrienes C4, B4, and prostaglandin E2 after two weeks of intranasal corticosteroid therapy, with changes observed only in eosinophil counts [11,12].

Conclusion

Although significant differences in cytokine concentrations were not consistently observed between groups, our findings suggest that IL-4 and IL-13 serum levels are elevated in patients with allergic rhinitis compared to controls. Moreover, patients with seasonal allergic rhinitis exhibit higher cytokine

concentrations than those with persistent forms. Overweight patients also showed increased IL-4 levels relative to normal and underweight subjects.

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