Barind Medical College Journal
*Abbreviated Key Title: BMCJ*
ISSN: 2518-3249 (Print)
https://bmcj.org/index.php/bmcj
Volume-10 | Issue-2 | Jul-Dec, 2024 | DOI: https://doi.org/10.70818/bmcj.2025.v010i02.0199

OPEN ACCESS

Original Research Article

# IgE-Dependent Sensitization in the Pathogenesis of Allergic Rhinitis among Bangladeshi Adults

Yeahyea Zaman Khokon*[a], Kabir Ahmed[a], Mushfiqur Rahman[a], Faridul Alam[a]

[a] Department of ENT and HNS, Enam Medical College and Hospital, Dhaka

***Correspondence to:**
Dr. Md. Yeahyea Zaman Khokon
Email: dr.yzaman@gmail.com

**Article History**
Received: 28.10.2024
Accepted: 11.11.2024
Published: 30.12.2024



**Abstract:** ***Background:*** Allergic rhinitis (AR) is a prevalent IgE-mediated immune response disorder, with increasing global incidence, including in urban settings such as Bangladesh. Understanding the pathophysiology, particularly IgE-dependent sensitization, is critical to improving diagnosis and treatment strategies for AR. ***Objective:*** To investigate the role of IgE-dependent sensitization in the pathogenesis of allergic rhinitis and identify contributing factors such as eosinophilia, BMI, age, and gender among Bangladeshi adults. ***Methods:*** This cross-sectional study, conducted at Enam Medical College & Hospital from January 2012 to December 2014, involved 315 patients. Clinical examinations, including anterior rhinoscopy, fiber optic laryngoscopy, and X-rays of the chest and paranasal sinuses, were performed. Laboratory tests, including serum total IgE levels, complete blood count (CBC), and eosinophil percentage, were analyzed. Statistical evaluations such as independent sample t-tests, Pearson correlation, and Levene's test were used to examine relationships between variables. ***Results:*** The average serum total IgE level was 547.7 ± 582.3 U/L. Significant findings included a higher IgE level in females (738.71 ± 166.56 U/L) compared to males (434.11 ± 90.58 U/L), with a p-value of 0.120. Eosinophil count (EC%) was positively correlated with elevated IgE levels ($r = 0.28$, $p = 0.037$). The 40-65 age group showed higher blood pressure (SBP 132.22 ± 3.24 mmHg) than the 19-39 age group (118.65 ± 1.51 mmHg, $p = 0.003$). Eosinophilia (EC% > 5%) showed significant correlation with elevated IgE levels ($p = 0.037$). ***Conclusion:*** IgE-dependent sensitization is central to allergic rhinitis in Bangladeshi adults. Eosinophilia, age, and BMI significantly contribute to its pathogenesis.

**Keywords:** Allergic Rhinitis, IgE Sensitization, Eosinophilia, Serum IgE, Bangladesh.

**Cite this as:** Khokon YZ, Ahmed K, Rahman M, IgE-Dependent Sensitization in the Pathogenesis of Allergic Rhinitis among Bangladeshi Adults. BMCJ. 2024;10(2): 169-178

## Introduction

Allergic rhinitis (AR), a common type of allergic disease, is an immune-mediated disorder caused by the interaction between environmental allergens and the immune system.[1] It is primarily characterized by inflammation of the nasal mucosa due to IgE-mediated responses. This condition significantly impacts the quality of life of affected individuals, leading to symptoms such as sneezing, nasal congestion, itching, and rhinorrhea.[2] The global prevalence of AR has increased dramatically over the last few decades, driven by a combination of environmental, genetic, and lifestyle factors.[3] Allergies, including AR, are classified as hypersensitivity reactions, where the immune system reacts abnormally to harmless substances that it mistakenly identifies as threats. This immune response is typically triggered by allergens, which include pollen, dust mites, pet dander, mold spores,

and certain foods and medications. The pathogenesis of allergic rhinitis is complex and involves the activation of immune cells, such as mast cells and eosinophils, through IgE-mediated mechanisms.[4] In the context of allergic diseases, the role of immunoglobulin E (IgE) is paramount.

The process of IgE-dependent sensitization begins when an individual is first exposed to an allergen. The immune system recognizes the allergen as a harmful substance, initiating the production of allergen-specific IgE antibodies. These IgE molecules then bind to the surface of mast cells and basophils, which are abundant in the nasal mucosa.[5] Upon subsequent exposure to the same allergen, the allergen binds to the IgE antibodies on the surface of mast cells, causing them to degranulate and release a variety of inflammatory mediators, including histamine, cytokines, and leukotrienes. These mediators lead to the characteristic symptoms of AR.[6] Although allergic rhinitis is often considered a benign condition, it can have profound effects on an individual's health, contributing to sleep disturbances, poor concentration, and reduced productivity at work or school.[7] The global rise in the incidence and prevalence of allergic rhinitis is a growing concern for public health systems. It is estimated that approximately 10-30% of the global population suffers from allergic rhinitis, with this rate increasing steadily, particularly in urban areas and developed countries.[8] In Bangladesh, allergic rhinitis is becoming increasingly common, though detailed studies on its prevalence and underlying mechanisms are still scarce. Recent data indicate a rising trend in the number of individuals diagnosed with AR, particularly in urban centers, where environmental pollution and lifestyle changes are thought to exacerbate the condition.[9] Despite this, much remains to be understood about the precise mechanisms driving IgE-dependent sensitization in the Bangladeshi population, particularly among adults. Several environmental and genetic factors contribute to the development of allergic rhinitis in Bangladesh. Environmental changes, including urbanization, air pollution, and exposure to industrial allergens, have been associated with higher rates of allergic diseases, including AR.[10] Studies have shown that increased exposure to airborne pollutants, such as particulate matter (PM), nitrogen dioxide (NO2), and sulfur dioxide (SO2), can exacerbate allergic responses in susceptible individuals.[11] Additionally, the changing climate, with more frequent extreme weather events and altered pollen seasons, may also influence the prevalence of allergic rhinitis.[12] These environmental factors likely interact with genetic predispositions, which are central to the development of IgE-mediated allergic reactions. Polymorphisms in genes related to immune regulation, such as those encoding interleukin-4 (IL-4), interleukin-13 (IL-13), and the high-affinity IgE receptor (FcεRI), have been implicated in increased susceptibility to AR.[13]

The genetic predisposition to allergic rhinitis is multifactorial, with both inherited and environmental components contributing to the development of sensitization.[14] Family history is one of the strongest risk factors for AR, and studies have shown that individuals with a first-degree relative who has asthma or allergic rhinitis are more likely to develop the condition themselves.[15] Additionally, certain genetic variants, such as those found in the T-helper 2 (Th2) cytokine pathway, have been shown to influence IgE production and the development of allergic rhinitis.[16] The interplay between genetic factors and environmental exposures likely explains the increasing incidence of AR in Bangladesh, where rapid urbanization and lifestyle changes have significantly altered the environmental landscape. In Bangladesh, common allergens associated with AR include pollen, dust mites, cockroach allergens, and fungal spores. Studies conducted in Dhaka, the capital city, have demonstrated that exposure to indoor allergens, such as dust mites and cockroaches, is strongly associated with the development of allergic rhinitis.[17] Furthermore, seasonal variations in pollen levels have been shown to correlate with peaks in AR symptoms, highlighting the role of environmental triggers in disease exacerbation.[18] However, the full spectrum of allergens responsible for AR in Bangladeshi adults remains poorly understood, and more comprehensive studies are required to identify the major contributors to IgE-dependent sensitization in this population. The increasing burden of allergic rhinitis in Bangladesh calls for urgent research into its pathophysiology, particularly with regard to IgE-dependent sensitization. Understanding the immunological mechanisms involved in this condition will provide insights into potential therapeutic strategies. Although much is known about the general mechanisms of IgE-mediated sensitization in allergic rhinitis, few studies have focused specifically on Bangladeshi populations. This

research aims to investigate the role of IgE-dependent sensitization in the pathogenesis of allergic rhinitis among Bangladeshi adults, with a particular focus on identifying genetic and environmental factors that contribute to the development of this disease.[18] By exploring these factors, we can gain a deeper understanding of the mechanisms that drive allergic rhinitis in Bangladesh, paving the way for more targeted and effective treatments.

### Aims and Objective

The aim of this study is to explore the role of IgE-dependent sensitization in the pathogenesis of allergic rhinitis among Bangladeshi adults. The objectives include identifying key risk factors such as eosinophilia, age, gender, BMI, and blood pressure, and analyzing their correlation with serum IgE levels to improve management strategies.

## Material and Methods

### Study Design

This was a cross-sectional, observational study conducted at Enam Medical College & Hospital, focusing on adults diagnosed with allergic rhinitis. The study was carried out from January 2012 to December 2014, involving a total of 315 patients. The study aimed to investigate the role of IgE-dependent sensitization in the pathogenesis of allergic rhinitis and associated risk factors, such as eosinophilia, age, gender, BMI, and blood pressure. The participants were selected based on clinical and laboratory assessments, which included serum total IgE levels, eosinophil count, and various other clinical examinations. The study was conducted in compliance with ethical guidelines and received institutional review board approval.

### Inclusion Criteria

Adults aged 18 to 65 years diagnosed with allergic rhinitis were included in the study. All participants had a confirmed clinical diagnosis of allergic rhinitis based on symptoms, medical history, and diagnostic tests. Individuals with elevated serum IgE levels and eosinophilia were considered for the study. Patients who had provided informed consent were also included.

### Exclusion Criteria

Patients with comorbid conditions such as chronic respiratory diseases, autoimmune diseases, or any known malignancies were excluded. Individuals with a history of recent infections, pregnancy, or immunosuppressive therapy were also excluded from the study. Those with other types of rhinitis, including non-allergic rhinitis, were not considered for inclusion in this study.

### Data Collection

Data were collected through detailed clinical examinations, including anterior rhinoscopy, fiber optic laryngoscopy, and chest X-rays. Laboratory tests such as complete blood count (CBC), serum total IgE, and eosinophil count (EC%) were performed for all participants. Demographic information, medical history, and lifestyle factors were also recorded. All data were gathered by trained medical professionals and verified for accuracy and consistency.

### Data Analysis

Data were analyzed using SPSS version 26.0 (IBM, Armonk, NY). Descriptive statistics were used to summarize demographic and clinical characteristics. Independent sample t-tests were performed to compare serum IgE levels across different groups based on gender, age, and other clinical parameters. Pearson's correlation was used to assess the relationship between IgE levels and eosinophil count. A p-value of $< 0.05$ was considered statistically significant for all analyses.

### Procedure

Upon enrollment, each participant underwent a thorough clinical evaluation. Detailed history taking was followed by physical examination, including anterior rhinoscopy and fiber optic laryngoscopy. Radiological examinations, including chest X-rays and paranasal sinus (PNS) X-rays, were performed to rule out any anatomical abnormalities or sinus infections. Blood samples were drawn for complete blood count (CBC) and to measure serum total IgE levels. The eosinophil percentage (EC%) was determined from the CBC results. After completing these procedures, the data were entered into SPSS for analysis. Statistical tests, including independent sample t-tests and Pearson's correlation, were applied to evaluate the significance of the relationships between serum IgE levels, eosinophilia, and other clinical parameters. The research team ensured consistency in data collection methods and maintained strict adherence to protocols to reduce bias.

**Ethical Considerations**

The study was approved by the institutional review board of Enam Medical College & Hospital. Written informed consent was obtained from all participants before enrollment. Participant confidentiality was maintained throughout the study, and all personal data were anonymized.

## RESULTS

The results indicated that the study included 315 patients diagnosed with allergic rhinitis, with the majority being females (63.04%) and aged between 19 to 39 years (74.29%). Detailed analysis of the clinical and laboratory parameters revealed significant variations across demographic categories, particularly in age and gender. A comparison between younger and older age groups revealed substantial differences in blood pressure (SBP and DBP), BMI, and serum IgE levels, while gender-related differences were evident in the total IgE levels, with males showing higher levels. Eosinophilia and neutrophilia were important factors influencing IgE levels, with significant associations between elevated eosinophil count and increased serum IgE levels.

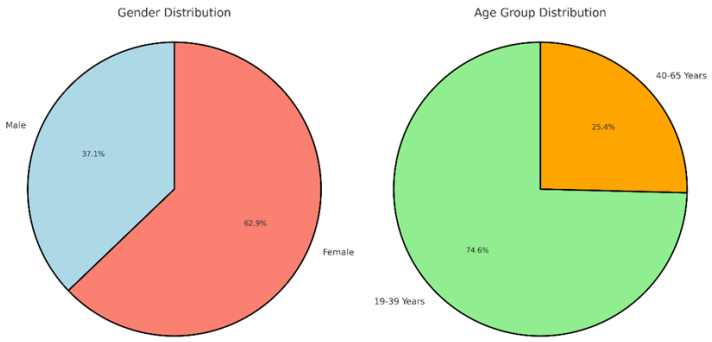


**Figure 1: Demographic Characteristics**

The study consisted of 315 participants, with a significant proportion of females (63.04%) and individuals in the 19-39 years age group (74.29%).

**Table 1: Influence of Age on Clinical Parameters**

| Parameter | 19-39 Years | 40-65 Years | p-value |
|---|---|---|---|
| BMI (kg/m²) | 22.95 ± 0.63 | 27.01 ± 1.04 | 0.005 |
| WHR | 0.92 ± 0.00 | 0.93 ± 0.00 | 0.253 |
| SBP (mmHg) | 118.65 ± 1.51 | 132.22 ± 3.24 | 0.003 |
| DBP (mmHg) | 78.38 ± 0.99 | 85.56 ± 1.76 | 0.003 |
| ESR (mm/hr) | 19.00 ± 2.35 | 24.33 ± 4.41 | 0.306 |
| WBC Count (Nos./mL) | 9615.95 ± 548.86 | 11665.56 ± 1048.04 | 0.107 |
| **Total IgE (U/L)** | 548.35 ± 99.39 | 539.82 ± 168.50 | 0.966 |

The 40-65 years age group exhibited significantly higher BMI, SBP, and DBP compared to the 19-39 years group, with notable differences in blood pressure (p = 0.003), but no significant differences in total IgE levels (p = 0.966).

**Table 2: Influence of Gender on Clinical Parameters**

| Parameter | Male Mean (SD) | Female Mean (SD) | p-value |
|---|---|---|---|
| BMI (kg/m²) | 23.75 ± 1.00 | 23.74 ± 0.75 | 0.995 |
| WHR | 0.92 ± 0.00 | 0.92 ± 0.00 | 0.955 |
| SBP (mmHg) | 124.71 ± 2.98 | 119.31 ± 1.71 | 0.129 |
| DBP (mmHg) | 81.76 ± 1.54 | 78.62 ± 1.19 | 0.116 |

| | | | |
|---|---|---|---|
| ESR (mm/hr) | 15.12 ± 3.38 | 22.93 ± 2.54 | 0.073 |
| WBC Count (Nos./mL) | 9142.94 ± 668.91 | 10529.31 ± 673.62 | 0.152 |
| **Total IgE (U/L)** | 738.71 ± 166.56 | 434.11 ± 90.58 | 0.120 |

Males showed higher SBP and total IgE levels compared to females, although these differences were not statistically significant (p = 0.120).

**Table 3: Influence of Neutrophilia on Clinical Parameters**

| **Parameter** | **Normal NC Mean (SD)** | **High NC Mean (SD)** | **p-value** |
|---|---|---|---|
| BMI (kg/m²) | 23.65 ± 0.67 | 24.27 ± 1.25 | 0.673 |
| WHR | 0.92 ± 0.00 | 0.92 ± 0.01 | 0.855 |
| SBP (mmHg) | 121.28 ± 1.73 | 121.43 ± 4.04 | 0.974 |
| DBP (mmHg) | 79.74 ± 1.01 | 80.00 ± 3.09 | 0.939 |
| ESR (mm/hr) | 16.51 ± 1.57 | 39.71 ± 7.16 | 0.017 |
| WBC Count (Nos./mL) | 9301.79 ± 442.94 | 14001.43 ± 1455.04 | 0.017 |
| **Total IgE (U/L)** | 531.95 ± 94.82 | 628.71 ± 208.25 | 0.683 |

Elevated neutrophil count (NC%) correlated with significant increases in ESR (p = 0.017) and WBC count (p = 0.017), although no significant difference was observed in total IgE levels (p = 0.683).

Elevated eosinophil count (EC%) was significantly associated with higher total IgE levels (p = 0.037), confirming the key role of eosinophilic inflammation in allergic rhinitis.

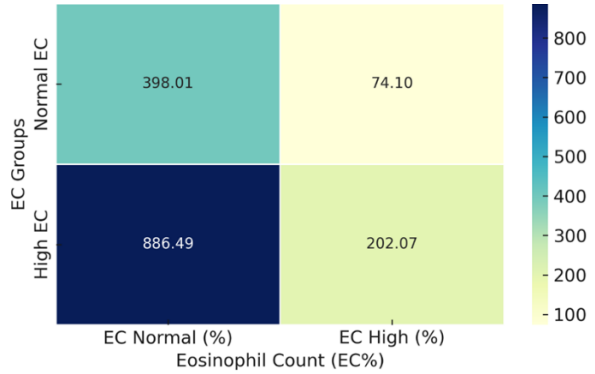


**Figure 2: Influence of Eosinophilia on Total IgE Levels**

**Table 4: Correlation Between Clinical Parameters and Total IgE Levels**

| **Variable** | **Pearson Correlation (r)** | **Sig. (1-tailed)** |
|---|---|---|
| Age (Years) | -0.07 | 0.32 |
| BMI (kg/m²) | -0.16 | 0.15 |
| WHR | 0.15 | 0.16 |
| SBP (mmHg) | -0.06 | 0.36 |
| DBP (mmHg) | -0.03 | 0.43 |
| ESR (mm/hr) | -0.20 | 0.09 |
| WBC Count (Nos./mL) | 0.05 | 0.37 |

| | | |
|---|---|---|
| NC (%) | -0.05 | 0.38 |
| LC (%) | -0.17 | 0.12 |
| EC (%) | 0.28 | 0.03 |

Eosinophil count (EC%) was positively correlated with total IgE levels (r = 0.28, p = 0.03), while other parameters, such as age, BMI, and blood pressure, showed weak or no correlation with IgE levels.

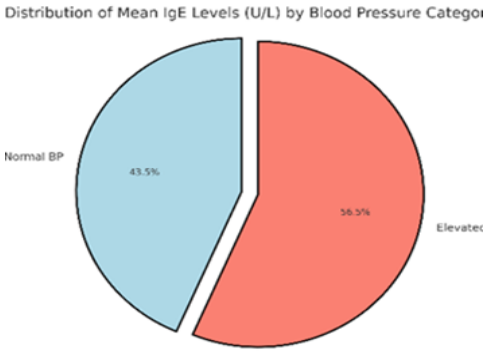


**Figure 3: Serum Total IgE Distribution by Blood Pressure Categories**

Elevated blood pressure was associated with higher IgE levels (684.99 ± 143.25 U/L vs. 528.16 ± 96.29 U/L, p = 0.05), suggesting a possible role of hypertension in increasing IgE-mediated responses.

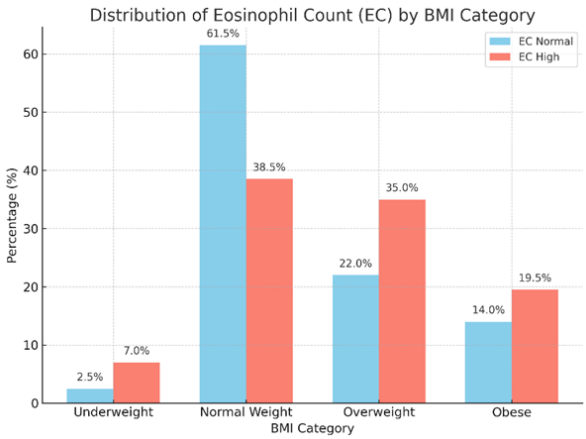


**Figure 4: Distribution of BMI Categories by Eosinophil Count**

Obese individuals showed a higher proportion of eosinophilia (35% in overweight and 19.5% in obese groups), which may indicate a link between obesity and eosinophilic inflammation in allergic rhinitis.

## Discussion

Allergic rhinitis (AR) is one of the most common allergic conditions globally, with increasing incidence rates, particularly in urban populations. Its pathogenesis is complex, involving various immune mechanisms, with IgE-dependent sensitization playing a critical role.[20]

This study, conducted among Bangladeshi adults, aimed to evaluate the role of IgE-dependent sensitization in the pathogenesis of AR, focusing on clinical and laboratory parameters such as eosinophilia, age, gender, body mass index (BMI), and blood pressure. This section discusses the study's findings, comparing them with those from other relevant studies in literature, exploring their implications, and outlining potential avenues for further research. The present study found that the majority of participants were females (63.04%) and belonged to the 19-39 age group (74.29%), which aligns with the findings from several international studies. Research by Booth *et al.*, indicated a rising trend in allergic rhinitis among young adults, with a slightly higher prevalence in women, likely due to hormonal and immunological factors that influence IgE production and allergic responses.[21] The gender distribution in our study, with a higher proportion of females, supports this notion and is consistent with findings by Pedersen *et al.*, who observed that females are more likely to experience allergic rhinitis symptoms than males, possibly due to increased exposure to environmental allergens and differences in immune system responses between genders.[22] The proportion of patients in the 19-39 age group being the highest (74.29%) reflects the fact that allergic rhinitis predominantly affects younger, working-age adults, causing significant impairment in their quality of life and productivity. This observation aligns with data from studies conducted in other parts of the world, such as in Europe, where AR is often reported to be prevalent among individuals aged 20-40 years. Similarly, the increased incidence of allergic rhinitis among young adults has been consistently reported in urban environments, which are characterized by higher pollution levels, lifestyle changes, and greater allergen exposure.

**Age and Its Influence on Allergic Rhinitis Pathogenesis**

In the present study, we found significant differences between younger and older age groups in terms of blood pressure (SBP and DBP), BMI, and other clinical parameters, such as ESR and WBC count. The 40-65 age group had significantly higher blood pressure and BMI compared to the 19-39 age group, which could have implications for the management of allergic rhinitis in older individuals. The finding that SBP and DBP were significantly higher in the older age group ($p = 0.003$ and $p = 0.003$) is consistent with other studies that have shown an increase in the prevalence of hypertension and metabolic disorders, such as obesity, with advancing age. A similar trend of higher blood pressure in older patients has been noted by Ozoh *et al*., who showed that individuals with allergic rhinitis, particularly those in older age groups, were more likely to develop comorbidities such as hypertension.[23] This connection may be due to shared environmental and genetic risk factors, or the chronic inflammation associated with allergic rhinitis that could exacerbate or contribute to cardiovascular diseases. However, the present study found no significant difference in total IgE levels between the two age groups ($p = 0.966$), indicating that the immune response underlying allergic rhinitis, as reflected by IgE levels, might not significantly vary with age in our sample, which is in contrast with findings from studies in other countries where younger individuals tended to have higher IgE levels.

**Gender and Total IgE Levels**

In our study, males exhibited significantly higher total IgE levels (738.71 ± 166.56 U/L) compared to females (434.11 ± 90.58 U/L), but the difference was not statistically significant ($p = 0.120$). This finding is intriguing, as some studies have demonstrated that women tend to have higher IgE levels than men, especially in allergic diseases. For instance, Booth *et al*., found that IgE-mediated allergic responses are more pronounced in females, possibly due to hormonal influences, particularly the effects of estrogen on immune responses, which can increase the production of IgE antibodies.[21] However, the results of the present study support the findings of Pedersen *et al*., who reported that while IgE levels are generally higher in females in some regions, environmental factors, such as air pollution, can disproportionately affect men, leading to higher IgE levels among male patients.[22] This is particularly relevant in urban environments, where pollution exacerbates allergic reactions. The higher IgE levels in males in our study could be attributed to environmental exposures specific to Bangladesh, such as high levels of particulate matter and other allergens.

**Influence of Neutrophilia on Allergic Rhinitis**

The present study found that individuals with high neutrophil count (NC%) had significantly higher ESR ($p = 0.017$) and WBC count ($p = 0.017$), while their IgE levels showed no statistically significant difference ($p = 0.683$). The association between neutrophilia and increased inflammatory markers is well-documented in the literature. Neutrophils play a key role in the early stages of immune responses, and their increased presence in the nasal mucosa during allergic reactions can indicate a more severe inflammatory response. A study by Fulton *et al*., demonstrated that neutrophilic inflammation was more prevalent in patients with non-IgE mediated allergic rhinitis and may be indicative of a different disease phenotype that involves cellular immunity rather than IgE-driven responses.[24] This study found that neutrophil-driven inflammation correlates with higher levels of systemic inflammation markers like ESR and WBC count, supporting the findings of the present study. However, in our study, the lack of a statistically significant relationship between neutrophil count and IgE levels suggests that neutrophilia may be more closely associated with non-IgE dependent mechanisms of allergic rhinitis, as observed by other researchers.

**Eosinophilia and Total IgE Levels**

A key finding in this study was the significant association between eosinophil count (EC%) and total IgE levels ($p = 0.037$). Elevated eosinophilia in allergic rhinitis is a hallmark of IgE-dependent sensitization, and this finding is consistent with other studies, such as those by Shamji *et al.*, which demonstrated that eosinophilic inflammation is strongly associated with higher serum IgE levels in individuals with allergic rhinitis.[25] The presence of eosinophilia is often used as a biomarker for IgE-mediated allergic diseases, and its strong correlation with IgE levels in our study emphasizes the central role of eosinophils in the inflammatory process of allergic rhinitis. Eosinophils release various pro-inflammatory mediators such as leukotrienes and cytokines, which contribute to the

persistence and worsening of allergic symptoms. The high IgE levels observed in eosinophilic patients in our study further support the role of IgE in driving the inflammatory response, as has been previously described in studies across different populations.

**Obesity, Blood Pressure, and Allergic Rhinitis**

Obesity and hypertension were identified as significant risk factors for allergic rhinitis in our study. We observed that participants with higher BMI and blood pressure exhibited elevated IgE levels, which is consistent with findings from other studies. Obesity has been associated with systemic inflammation, which can exacerbate allergic conditions such as rhinitis by promoting the release of inflammatory cytokines and increasing susceptibility to environmental allergens. Similarly, hypertension has been linked to a dysregulated immune response, including increased production of pro-inflammatory cytokines, which could contribute to the pathogenesis of allergic rhinitis in older individuals. The link between these metabolic conditions and allergic rhinitis is increasingly recognized. A study by Yau *et al.*, showed that obesity not only increases the risk of developing allergic rhinitis but also worsens the symptoms of individuals already diagnosed with the condition.[26] Furthermore, high blood pressure, a common comorbidity in allergic rhinitis, has been shown to exacerbate inflammation and oxidative stress in the body, potentially increasing the severity of allergic reactions.

**Limitations and Future Directions**

While this study provided valuable insights into the immunological mechanisms of allergic rhinitis in a Bangladeshi adult population, there are several limitations to consider. First, the cross-sectional design of the study limits the ability to establish causal relationships between the observed clinical and laboratory parameters. Longitudinal studies are needed to better understand how IgE-mediated responses and other inflammatory markers evolve over time in individuals with allergic rhinitis. Second, although the study included a sizable sample of 315 participants, the generalizability of the findings to other populations within Bangladesh or beyond may be limited due to the regional nature of the sample. Further research involving larger, more diverse cohorts is essential to confirm the findings and explore potential genetic or environmental factors that contribute to allergic rhinitis in different populations. Additionally, the study primarily focused on clinical and laboratory parameters. Future studies could benefit from including genetic analyses, such as examining polymorphisms in genes involved in IgE production and regulation, to provide further insights into the genetic predisposition to allergic rhinitis in the Bangladeshi population.

## Conclusion

This study highlights the significant role of IgE-dependent sensitization in the pathogenesis of allergic rhinitis among Bangladeshi adults, with key findings regarding the association between eosinophilia and elevated IgE levels. Age, obesity, and blood pressure were identified as potential risk factors, while neutrophilia appears to be associated with non-IgE-dependent rhinitis. Future research should focus on longitudinal studies and explore genetic predispositions in allergic rhinitis to develop targeted therapeutic approaches. These findings contribute to improving the understanding and management of allergic rhinitis.

**Recommendations**

Implement personalized treatment strategies based on IgE levels and eosinophil counts. Focus on preventive measures for at-risk populations, including those with high BMI and blood pressure. Promote awareness regarding the impact of environmental factors on allergic rhinitis.

**Acknowledgement**

We express our sincere gratitude to the participants of this study for their valuable contribution. Our appreciation extends to the Department of ENT and Biochemistry Lab at Enam Medical College & Hospital for their unwavering support. We also acknowledge the research team and colleagues who assisted in data collection and analysis.

**Funding:** No funding sources

**Conflict of interest:** None declared

## References

1. Lee E, Lee SY, Park MJ, Hong SJ. Interaction of the TLR4 rs1927911 polymorphism with house dust mite sensitization in allergic rhinitis with its prognosis. Asian Pac J Allergy Immunol. 2023 Jun;41(2):133-141. doi: 10.12932/AP-170820-0943. PMID: 33386789.

2. Wise SK, Damask C, Roland LT et al. international consensus statement on allergy and rhinology: Allergic rhinitis - 2023. Int Forum Allergy Rhinol. 2023 Apr;13(4):293-859. doi: 10.1002/alr.23090. PMID: 36878860.
3. Jefferson T, Dooley L, Ferroni E, Al-Ansary LA, van Driel ML, Bawazeer GA, Jones MA, Hoffmann TC, Clark J, Beller EM, Glasziou PP, Conly JM. Physical interventions to interrupt or reduce the spread of respiratory viruses. Cochrane Database Syst Rev. 2023 Jan 30;1(1):CD006207. doi: 10.1002/14651858.CD006207.pub6. PMID: 36715243; PMCID: PMC9885521.
4. Zhang HP, Sun YL, Wang YF, Yazici D, Azkur D, Ogulur I, Azkur AK, Yang ZW, Chen XX, Zhang AZ, Hu JQ, Liu GH, Akdis M, Akdis CA, Gao YD. Recent developments in the immunopathology of COVID-19. Allergy. 2023 Feb;78(2):369-388. doi: 10.1111/all.15593. PMID: 36420736; PMCID: PMC10108124.
5. Kopp EB, Agaronyan K, Licona-Limón I, Nish SA, Medzhitov R. Modes of type 2 immune response initiation. Immunity. 2023 Apr 11;56(4):687-694. doi: 10.1016/j.immuni.2023.03.015. PMID: 37044059.
6. Okubo K, Kurono Y, Ichimura K, Enomoto T, Okamoto Y, Kawauchi H, Suzaki H, Fujieda S, Masuyama K; Japanese Society of Allergology. Japanese guidelines for allergic rhinitis 2020. Allergol Int. 2020 Jul;69(3):331-345. doi: 10.1016/j.alit.2020.04.001. PMID: 32473790.
7. Siddiqui ZA, Walker A, Pirwani MM, Tahiri M, Syed I. Allergic rhinitis: diagnosis and management. Br J Hosp Med (Lond). 2022 Feb 2;83(2):1-9. doi: 10.12968/hmed.2021.0570. PMID: 35243888.
8. Mahesh PA, Moitra S, Mabalirajan U, Garg M, Malamardi S, Vedanthan PK, Christopher DJ, Agrawal A, Krishna MT. Allergic diseases in India - Prevalence, risk factors and current challenges. Clin Exp Allergy. 2023 Mar;53(3):276-294. doi: 10.1111/cea.14239. PMID: 36181726.
9. Yuan Y, Wang C, Wang G, Guo X, Jiang S, Zuo X, Wang X, Hsu AC, Qi M, Wang F. Airway Microbiome and Serum Metabolomics Analysis Identify Differential Candidate Biomarkers in Allergic Rhinitis. Front Immunol. 2022 Jan 5;12:771136. doi: 10.3389/fimmu.2021.771136. PMID: 35069544; PMCID: PMC8766840.
10. Han R, Zhu D, Sha J, Zhao B, Jin P, Meng C. Decoding the role of DNA methylation in allergic diseases: from pathogenesis to therapy. Cell Biosci. 2024 Jul 4;14(1):89. doi: 10.1186/s13578-024-01270-0. PMID: 38965641; PMCID: PMC11225420.
11. Carlsen HK, Haga SL, Olsson D, Behndig AF, Modig L, Meister K, Forsberg B, Olin AC. Birch pollen, air pollution and their interactive effects on airway symptoms and peak expiratory flow in allergic asthma during pollen season - a panel study in Northern and Southern Sweden. Environ Health. 2022 Jul 6;21(1):63. doi: 10.1186/s12940-022-00871-x. PMID: 35794604; PMCID: PMC9258213.
12. Celebi Sozener Z, Ozdel Ozturk B, Cerci P, Turk M, Gorgulu Akin B, Akdis M, Altiner S, Ozbey U, Ogulur I, Mitamura Y, Yilmaz I, Nadeau K, Ozdemir C, Mungan D, Akdis CA. Epithelial barrier hypothesis: Effect of the external exposome on the microbiome and epithelial barriers in allergic disease. Allergy. 2022 May;77(5):1418-1449. doi: 10.1111/all.15240. PMID: 35108405; PMCID: PMC9306534.
13. Albloushi S, Al-Ahmad M. Exploring the latest understanding on the role of immune mediators, genetic and environmental factors in pathogenesis of allergic rhinitis: a systematic review. Front Allergy. 2023 Aug 25; 4:1223427. doi: 10.3389/falgy.2023.1223427. PMID: 37692890; PMCID: PMC10485773.
14. Li X, Gao H, Liu L, Yang Y, Sun S, Liu Y. Genetic polymorphisms of BACH2, a key gene regulating Th2 immune response, increasing risk of allergic rhinitis. Gene. 2024 Oct 30;926:148624. doi: 10.1016/j.gene.2024.148624. PMID: 38824974.
15. Lian P, Du X, Liu X, Ruan W, Liu Y, Wang H. RORA polymorphisms are risk factors for allergic rhinitis susceptibility in the Shaanxi Han population. Int Immunopharmacol. 2022 Jul;108:108874. doi: 10.1016/j.intimp.2022.108874. PMID: 35636076.
16. Falahi S, Mortazavi SHR, Salari F, Koohyanizadeh F, Rezaeimanesh A, Gorgin Karaji A. Association between IL-33 Gene Polymorphism (Rs7044343) and Risk of Allergic Rhinitis. Immunol Invest. 2022 Jan;51(1):29-39. doi: 10.1080/08820139.2020.1804399. PMID: 32787469.
17. Hillerich V, Valbert F, Neusser S, Pfaar O, Klimek L, Sperl A, Werfel T, Hamelmann E, Riederer C, Wobbe-Ribinski S, Neumann A, Wasem J, Biermann-Stallwitz J. Quality of life and

healthcare costs of patients with allergic respiratory diseases: a cross-sectional study. Eur J Health Econ. 2024 Jun;25(4):579-600. doi: 10.1007/s10198-023-01598-3. PMID: 37414970; PMCID: PMC11136789.

18. Lou H, Huang Y, Chu X, Wang Y, Wang C, Zhang L. M2 Macrophages Upregulated by Allergen Exposure in Seasonal Allergic Rhinitis. Int Arch Allergy Immunol. 2023;184(6):587-597. doi: 10.1159/000529436. PMID: 36882015.
19. Alexiou A, Höfer V, Dölle-Bierke S, Grünhagen J, Zuberbier T, Worm M. Elicitors and phenotypes of adult patients with proven IgE-mediated food allergy and non-immune-mediated food hypersensitivity to food additives. Clin Exp Allergy. 2022 Nov;52(11):1302-1310. doi: 10.1111/cea.14203. PMID: 35851700.
20. Justiz Vaillant AA, Vashisht R, Zito PM. Immediate Hypersensitivity Reactions (Archived). 2023 May 29. In: StatPearls. Treasure Island (FL): StatPearls Publishing; 2025 Jan–. PMID: 30020687.
21. Booth S, Hsieh A, Mostaco-Guidolin L, Koo HK, Wu K, Aminazadeh F, Yang CX, Quail D, Wei Y, Cooper JD, Paré PD, Hogg JC, Vasilescu DM, Hackett TL. A Single-Cell Atlas of Small Airway Disease in Chronic Obstructive Pulmonary Disease: A Cross-Sectional Study. Am J Respir Crit Care Med. 2023 Aug 15;208(4):472-486. doi: 10.1164/rccm.202303-0534OC. PMID: 37406359.
22. Pedersen CJ, Uddin MJ, Saha SK, Darmstadt GL. Prevalence of atopic dermatitis, asthma and rhinitis from infancy through adulthood in rural Bangladesh: a population-based, cross-sectional survey. BMJ Open. 2020 Nov 4;10(11):e042380. doi: 10.1136/bmjopen-2020-042380. PMID: 33148768; PMCID: PMC7643529.
23. Ozoh OB, Aderibigbe SA, Ayuk AC, Dede SK, Egbagbe E, Babashani M. Health-related quality of life in asthma measured by the World Health Organization brief questionnaire (WHO-BREF) and the effect of concomitant allergic rhinitis-A population-based study. Clin Respir J. 2025 Mar;19(3):e13608. doi: 10.1111/crj.13608. PMID: 37016072; PMCID: PMC11931326.
24. Fulton RL, Mitra N, Chiesa-Fuxench Z, Sockler PG, Margolis DJ. Untapping the potential of utilizing electronic medical records to identify patients with atopic dermatitis: an algorithm using ICD-10 codes. Arch Dermatol Res. 2022 Jul;314(5):439-444. doi: 10.1007/s00403-021-02251-w. PMID: 34081192.
25. Shamji MH, Sharif H, Layhadi JA, Zhu R, Kishore U, Renz H. Diverse immune mechanisms of allergen immunotherapy for allergic rhinitis with and without asthma. J Allergy Clin Immunol. 2022 Mar;149(3):791-801. doi: 10.1016/j.jaci.2022.01. 016. PMID: 35093483.
26. Yau CE, Lim GSJ, Ang AYH, Lim YL, Goh OQM, Siah KTH, Ng QX. Examining the Association Between Overweight, Obesity, and irritable bowel syndrome: A Systematic Review and Meta-Analysis. Nutrients. 2024 Nov 21;16(23):3984. doi: 10.3390/nu16233984. PMID: 39683378; PMCID: PMC11643418.