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Clinical Characterization and Therapeutic Evaluation of Allergic Rhinitis

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Abstract: Allergic rhinitis (AR) is a chronic inflammatory condition characterized by nasal congestion, sneezing, rhinorrhoea, and itching that affects individuals of all ages, posing a substantial public health burden particularly in urbanizing and environmentally challenged regions like Telangana, India. This case-control study profiled fifteen AR patients and ten healthy volunteers through skin prick testing, serum Immunoglobulin E (IgE) quantification, and Absolute Eosinophil Count (AEC). All AR cases showed significantly elevated IgE levels (>500 IU/mL), with 40% exceeding 1000 IU/mL, indicating high allergic reactivity; however, none exceeded the 2000 IU/mL hyper-IgE threshold. Mean IgE was 784 ± 2.85 IU/mL, significantly higher than controls ($p<0.01$). AEC levels were significantly elevated in AR subjects (mean 784.33 ± 1.51 cells/µL) compared to controls (194 ± 3.67 cells/µL, $p<0.001$), showing moderate positive correlation with IgE ($r=0.648$, $p<0.01$). Gender-stratified analysis revealed females had significantly higher IgE levels (970 ± 2.27 IU/mL) than males (470 IU/mL; $p<0.01$). Therapeutically, antihistamines and intranasal corticosteroids were commonly prescribed, while allergen-specific immunotherapy remained underutilized. Additionally, 33.3% of patients reported using complementary approaches such as Ayurveda and homeopathy. Genetic analysis identified GSTM1 mutations, and ELISA showed elevated TNF-α levels. This study confirms IgE and AEC as accessible biomarkers and highlights the need for regionally personalized, patient-centered interventions that integrate pharmacologic efficacy with patient education for improved AR management.

Keywords: Allergens, Allergic rhinitis, Chronic rhinosinusitis, Antihistamines.

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1 Introduction

Allergic rhinitis (AR), a chronic inflammatory disorder of the nasal mucosa, is triggered by allergen exposure that leads to IgE-mediated inflammation. Clinically, it is characterized by four major symptoms i.e., rhinorrhea, sneezing, nasal itching, and nasal congestion. Additionally, it is associated with co-morbid conditions as asthma, atopic dermatitis and nasal polyps. Epidemiological studies indicate that approximately 20–30 % of the Indian population suffers from allergic rhinitis and from that 15 % of affected individuals develop asthma. The diagnosis, treatment and management of allergic rhinitis should adhere to the Allergic Rhinitis and its Impact on Asthma (ARIA) guidelines, while of asthma should follow the Global Initiative for Asthma) guidelines (GINA) [1].

The environmental etiological factors in AR refer to non-genetic factors that influences in contribution to disease development. Certain diseases serve as predisposing conditions for others, highlighting the interconnected nature of pathological processes. The etiological function of a disease entity explains its persistence and progression, shaping its clinical route. The etiologic classification of disease is based on the cause, when known, and is particularly significant in understanding biotic disease. On this basis, disease passes through distinct stages: Susceptibility, Pathological onset, Pre-symptomatic, Clinical, then Resolution. Each stage presents opportunities for intervention, including prevention, treatment, rehabilitation and self-care strategies, alongside necessary social adjustments.

Pharmacogenomic studies have evolved significantly, from candidate gene investigations to Genome-Wide Association Studies (GWAS) and sequencing-based

projects. Despite the relatively smaller sample sizes compared to genomic studies of polygenic diseases, pharmacogenomic investigations have demonstrated substantial drug-gene associations [2] Genetic polymorphisms affect drug-metabolizing enzymes and pharmacogenetic targets, such as receptors, have been identified. Pharmacogenetic testing may enable physicians to understand why patients react differently to various drugs and to make better decisions about therapeutic strategies [3]. Genotyping provides a more accurate approach than racial or ethnic categories to identify variations in drug response [4]. Unlike other factors that influences on drug response, genetic factors remain constant throughout an individual's lifetime. The use of pharmacogenetic data to support drug selection and dosing is emerging. Commercial testing is available for drug-metabolizing enzymes and pharmacodynamic targets such as VKORC1, stromelysin-1, and apolipoprotein etc.

Environmental determinants, such as allergen exposure, air pollution, climate change, ozone, smoking, viral infection, and environmental toxicants, may underlie much of the increase in prevalence of allergic rhinitis. Additionally, specific epigenetic changes induced by environmental factors may influence cellular homeostasis and contribute to the development of pathogenesis of allergic diseases [5]. Globally, allergic rhinitis affects approximately 15 to 30% of adults but higher prevalence among children. In addition to nasal and ocular symptoms that directly related to the allergic process impacts daily functioning and leading to daytime sleepiness and impaired quality of life. Patients miss their regular activities because of discomfortable symptoms but an even greater problem was interference with work productivity, which has been the biggest contributor to the total economic cost of allergic rhinitis. It was found that among the atopic disorders, allergic rhinitis was the most prevalent.

This study aims to investigate the etiological factors and family history/tendency associated with allergic rhinitis. Additionally, seeks to evaluate therapeutic interactions based on their epidemiological findings and the pathological implications of the various biomarkers. A key focus is to determine/assess drug toxicity and efficacy based on the severity of symptoms such as mild, moderate, or severe, this is to identify novel therapeutic target and biomarkers. Treatment of allergic rhinitis has been difficult; therefore, to address this issue, this study proposes to find new potential biomarkers of the disease. While Immunoglobulin E (IgE) has been widely studied as a potential biomarker, this research aims to explore alternative ones that may enhance diagnostic precision and treatment efficacy.

2 Materials and Methods

The subjects were recruited from the population of Telangana, India, who were attending the Clinics of Mahavir Hospital and Research Centre, Hyderabad. Subjects with history of AR as determined by the Health Professionals were selected as cases and healthy subjects served as controls. Written informed consent was obtained from each of the subjects after explaining the objectives of the study. The study group consisted of 15 AR cases and 10 healthy control subjects.

Blood Sample collection; 2 ml blood samples were collected from AR cases and control subjects, after taking written consent from the study group. This project was approved by the Institutional Ethics Committee (IEC) of Bhagwan Mahavir Medical Research Centre. The blood samples collected were stored at 4°C in the fridge for further use. The subjects were enrolled after preliminary screening by our ENT physician in accordance to Standard criteria for diagnosis procedures following inclusion and exclusion criteria.

Inclusion criteria: This study enrolled only those patients who are suffering from allergic rhinitis caused by aeroallergens. Control subjects were selected from among healthy individuals with no known history of allergic or chronic conditions.

Exclusion criteria: Participants excluded were children, pregnant women, individuals with diabetic mellitus, or those were diagnosed with other allergic conditions such as food allergies and chronic asthma and other diseases.

2.1 Diagnostic Methods

Skin Prick Test (SPT) Method

Skin Prick Test (SPT) was used as a primary diagnostic tool to identify the specific aeroallergens that was responsible for allergic rhinitis. This common test is painless and accurate, though it may be a little uncomfortable. This test was performed on the patient's forearm, pre-marked with a sterile marker and then a small amount of different allergens extract was introduced into the skin via a gentle prick or scratch technique. If the patient/subject is allergic to a particular allergen, the area becomes red, itchy or localized erythematous reaction appeared within 15 to 30 minutes, often accompanied by itching and raised,

hive-like welts called wheals, that indicated an allergic reaction. This test was found to be a safe, reliable and effective way to determine which allergens was causing the symptoms [6].

Immunoglobulin E (IgE) test (ELISA-Based)

An allergen-specific immunoglobulin E (IgE) assay was performed using the Enzyme Linked Immuno Sorbant Assay (ELISA), which is considered the current gold standard test for allergy and allergy related blood testing, that shows the levels of different IgE antibodies in a patient's blood. This test detects IgE mediated responses to environmental allergens. Blood samples were collected and analysed to measure the antibodies to specific allergens. It can detect all types of allergies, including food allergies [7]. Commercially available assays have been cross-standardized to a common primary human IgE standard [8]. Total IgE values reported in International Units of IgE per volume (IU/mL); a conversion factor (1 IU = 2.42 ng) is sometimes applied. This study reported total IgE values in International Units per millilitre (IU/mL), with 1 IU equivalent to 2.42 ng of IgE.

Nasal Allergen Provocation Test (NAPT)

Nasal challenge tests were used with standardized allergen extracts for selected participants. These tests are particularly commonly used and most valuable in research context. Whereas, occasionally used to confirm occupational rhinitis diagnoses.

2.2 Aetiology of AR

Aetiological studies aimed to investigate the causal relationship between putative risk factors (or determinants) and the incidence of allergic rhinitis. Unlike prognostic research, that predicts clinical outcomes, the aetiological approach aimed to focused on understanding of underlying pathophysiological contributors of AR.

2.3 Therapeutic interventions

Second-generation oral antihistamines and intranasal corticosteroids were prescribed as the mainstay of treatments. Allergen immunotherapy is an effective immune-modulating treatment recommended for patients exhibiting suboptimal response or intolerance to pharmacological management, or for those preferring long-term immune modulation [9].

2.4 Biomarker Analysis

Study of Genetic Polymorphisms

To determine the role of potential genetic associations, two polymorphic genes (GSTs-Glutathione S-transferase gene) variants (GSTT1 and GSTM1) and the tumor necrosis factor-alpha (TNF-α) polymorphism were analysed in a case control design.

Sample Collection: Peripheral blood (2 mL) was collected in EDTA tubes and stored at 4°C. Genomic DNA was isolated using the phenol-chloroform method, and DNA quality was confirmed by Gel-Electrophoresis. Polymerase chain reaction (PCR) was performed using the primers as mentioned below, followed by SNP studies by standard procedures.

PCR for Genotyping: Genomic DNA was isolated from peripheral blood samples of Rhinitis cases and controls using salting out method. Genotyping for GSTM1 and GSTT1 was carried out by PCR. The primer sequences used for the detection of GSTM1 and GSTT1 genotypes were as follows, along with Albumin as experimental marker.

A total PCR reaction volume of 20 μL containing 25 units/mL Taq DNA polymerase, 200 μM dNTPs, 1.5 mM $MgCl_2$, 0.25 μM of each primer, nuclease-free water, and 89 ng of genomic DNA were prepared. The desired genes were amplified under the following PCR conditions: 95°C (1 min), 60°C (1 min), 72°C (1 min) for 40 cycles, with a final extension step at 72°C for 10 min in a PTC-200 thermal cycler.

Biochemical Correlations: Circulating Biomarker such as IgE plays an important role in disease predisposition. Biochemical parameters were measured and data were correlated with genetic biomarkers.

Demographic characteristics of the patients: A structured questionnaire was designed to collect participant information. This information collected was used to understand the disease epidemiology (Supplementary Information 1).

Statistical analysis: Results were statistically evaluated, using Microsoft Excel and Python softwares. Chi-Square test was used to analyse data of genetic polymorphism data to compare frequencies between cases and controls. P values less than <0.05 were considered statistically significant.

3 Results

3.1 Patients Demographics/Epidemiology

The study comprised of 15 clinically confirmed cases (males and females) of allergic rhinitis and an equal number of healthy controls were matched by age and

Primer Sequences

Gene	Direction	Sequence (5′ to 3′)
GSTT1	Forward	TTC CTT ACT GGT CCT CAC ATC TC
	Reverse	TCA CCG GAT CAT GGC CAG CA
GSTM1	Forward	GAA CTC CCT GAA AAG CTA AAG C
	Reverse	GTT GGG CTC AAA TAT ACG GTG G
β-globin (control)	Forward	CAA CTT CAT CCA CGT TCA CC
	Reverse	GAA GAG CCA AGG ACA GGT AC
TNF-α (-308 G>A SNP)	Forward	AGG CAA TAG GTT TTG AGG GCC AT
	Reverse	TCC TCC CTG CTC CGA TTC CG

gender. The age distribution of subjects was ranged from 14 to 47 years, with a mean age of approximately 26.7 years, this represents a predominance of young adults. A slightly higher proportion of females of about 53% was observed, with mostly patients residing in urban or semi-urban Telangana (Table 1).

The age distribution revealed that the majority of participants (46.66%) fell within the 21-23 years age group, followed by 26.66% in the 14-20 years group range. These observations show a peak prevalence of allergic rhinitis among young adults, whereas, only one patient (6.66%) was in the 41-50 years range, indicating a lower disease prevalence in older individuals. Regarding the onset of symptoms, 46.66% of patients reported experiencing allergic manifestations since their childhood, while 26.66% reported symptoms persisting for 4-5 years while 20% described durations between 3-4 years. These findings suggested that chronic condition of allergic rhinitis with early life sensitization as a predominant patter. The gender distribution showed a higher prevalence among females i.e., 60% compared to males i.e., 40%, indicate females are more susceptible to allergic rhinitis. Tobacco or smoking history was negligible with only one patient i.e., 6.66%, reported minimal influence of active smoking on allergic rhinitis development in this group.

Occupational analysis showed that students comprised the largest subgroup (40%), followed by housewives (33.33%) and businessman (26.66%). This occupational diversity suggests that both indoor and outdoor environmental exposures may contribute to disease expression.

The symptom profiles also emphasized sneezing was the most frequent reported complaint ie., 66.66%, followed by nasal congestion (46.66%), runny nose (40%) and wheezing (33.33%). Less common symptoms included headache (26.66%), 13.33% cough, 6.66% nasal polyps and 6.66% asthma. These findings imitate a classic allergic rhinitis phenotype with potential overlap into lower respiratory tract hypersensitivity.

However, Absolute Eosinophil count (AEC) levels indicated that 46.66% of patients exhibited moderately elevated counts ie., 200-600 cells/cu mm, while a smaller fraction (13.33%) had AEC levels exceeding 600 cells/cu mm, suggested that they were suffering from eosinophilia and systemic allergic activity. The remaining 40% had AEC levels above 200 cells/cu mm, which may still reflect underlying allergic inflammation (Figure 1A). These findings of the study suggest that allergic rhinitis is more prevalent in younger individuals perticularly females are susceptible to allergic rhinitis than males.

3.2 Immunological profiling

Serum IgE quantification revealed high immunological patterns among patients diagnosed with allergic rhinitis. Figure 1 B presents the stratified distribution of Serum IgE concentrations among fifteen patients diagnosed with allergic rhinitis. Each patient is represented individually (P1 to P15), where male-gender were coded with vertical bars and females coded in bright pink. There are three clinical threshold line i.e., 500 IU/mL represents sensitization cutoff, 1000 IU/ml is high reactivity and 2000 IU/mL is hyper-IgE consideration. The results indicates that all patients exhibited serum IgE levels above the 500 IU/mL sensitization threshold, which confirms the immunologic responsiveness to allergens. Approximately 66.66% of individuals showed IgE concentrations below 1000 IU/ml, indicating mild to moderate allergic sensitization. Whereas, 33.33%

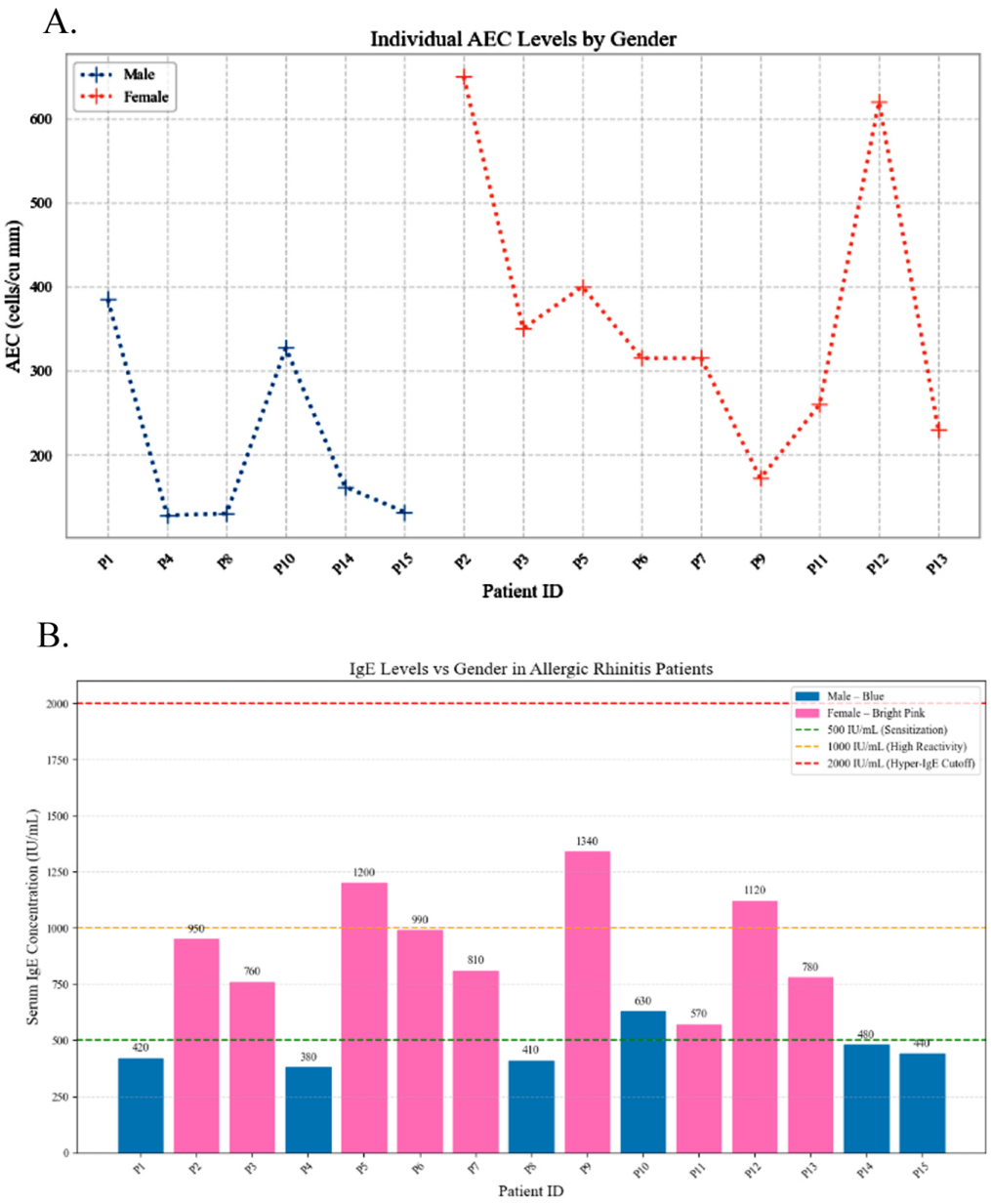

Figure 1. A. Gender Versus Absolute Eosinophil Count (AEC). Color peaks represent varying levels of absolute eosinophil count across male and female patients. Higher and brighter peaks indicate elevated eosinophil counts, especially among females, reflecting more intense allergic inflammation in this group. B. Patient's gender vs IgE levels. Results shows that female allergic rhinitis patients (bright pink bars) have higher serum IgE levels than males (blue bars), with several surpassing clinical thresholds for sensitization and high reactivity. This highlights a gender difference in IgE-mediated allergic response.

Table 1. Clinical and Epidemiological Profile of Patients with Allergic Rhinitis

Patient ID	Gender/Age	Symptoms	Duration	Smoking
P-1	M/23	Abdominal discomfort, frequent colds, sinus heaviness	4-5 years	No
P-2	F/28	Sneezing	3 years	No
P-3	F/22	Deviated nasal septum	5 years	No
P-4	M/23	Sneezing, running nose	6 months	Sometimes
P-5	F/32	Sneezing, watery discharge, right maxillary polyp	4 years	No
P-6	F/20	Recurrent UTI, watery discharge, cough, sneezing, DNS	Childhood	No
P-7	F/25	Blocked nose, wheezing, postnasal drip, headache	Childhood	No
P-8	M/47	Sneezing, runny nose, nasal obstruction	Childhood	No
P-9	F/19	Sneezing, nasal obstruction, granular throat	Childhood	No
P-10	M/35	Sneezing, nasal discharge, wheezing	3-4 years	No
P-11	F/26	Nasal discharge, congestion, headache	4-5 years	No
P-12	F/22	Wheezing, internal irritation, congestion, asthma	Childhood	No
P-13	F/40	Sneezing, runny nose, wheezing, headache	4-5 years	No
P-14	M/20	Sneezing, nasal discharge, wheezing, congestion	Childhood	No
P-15	M/14	Sneezing, runny nose, nasal congestion	Childhood	No

exhibited elevated IgE levels exceeding 1000 IU/mL, indicating reflective of hypersensitivity response. Notably, none of the patients crossed or exceeded the 2000 IU/mL cutoff, these values eliminated the immediate suspicion of hyper-IgE syndrome within this group. Gender specific trends highlighted that several female patients showed higher IgE concentration than males with notably values exceeded in cases like P5 showed 1200 IU/mL, P9 (1340 IU/mL) and P12 presented 1120 IU/mL. In contrast, IgE levels among male patients ranged from 380-630 IU/mL that reflects milder sensitization. The results stratification supports the serum IgE as a practical immunological marker in allergic rhinitis evaluation and also reflects heterogeneity in allergic reactivity across gender individuals. within medicine that focuses on the investigation of diseases, including their initial causes (aetiologies), their stepwise progressions (pathogenesis), and their effects on normal anatomical and physiological function. The present study showed that all the 15 cases had high levels of IgE and Absolute Eosinophil Count (AEC), these are the common symtoms of Allergic Rhinitis, these patients had been treated with a series of therapeutic drugs [10, 11].

These fifteen patients clinically diagnosed with AR were assessed for immunological parameters, including serium Immunoglobulin-E (IgE) concentration and Absolute Eosinophill Count (AEC), these values were further compared to established clinical thresholds to evalute sensitization severity and eosinophilic activation.

Serium IgE Statification

Serum IgE concentrations ranged from 380 IU/ml to 1340 IU/mL, with mean value of 784.67 $\pm$ 2.85 IU/ml. All patients showed values that exceeded 500 IU/mL sensitization threshold, this confirmed allergic sensitization. Notably, 40% of patients had IgE levels > 1000 IU/ml indicating high allergic reactivity, whereas, 60% fall within the 500-1000 IU/ml range. However, a gender-wise comparison revealed that female patients exhibited higher mean IgE levels of 970 $\pm$ 2.27 IU/mL compated to males i.e., 470 $\pm$ 1.5 IU/mL, this suggested the high tendency of humoral response in female subjects. The difference in mean IgE values between genders was statistically significant ($p<0.01$; unpaired t-test).

Absolute Eosinophil Count (AEC)

AEC vlaues were high across all AR cases, raning from 580 cell/μL to 1040 cells/μL, with a mean value of 784.33 $\pm$ 1.51 cells/μL, compated to control participants mean AEC ie., 194.50 $\pm$ 3.67 cells/μL, the difference was statistically significant ($p<0.001$; two-tailed t-test) that confirmed eosinophilic inflammation in the AR cases. Pearson correlation analysis between serum IgE and AEC revealed a moderate possitive correlation ($r = 0.548$, $p<0.01$), increased IgE levels are associated with elevated eosinophil counts. This reinforced the Th2-skewed immunopathology typical of allergic rhinitis.

3.3 Therapeutic Patterns

The therapeutic profile showed that 86.6% of patients were prescribed second-generation oral antihistamines (cetirizine, fexofenadine), and 73.3% received intranasal corticosteroids (fluticasone or mometasone). Despite guideline-based prescriptions, 60% of patients reported irregular adherence may be due to steroid-side effects or cost constraints. Only one patient showed allergen-specific immunotherapy

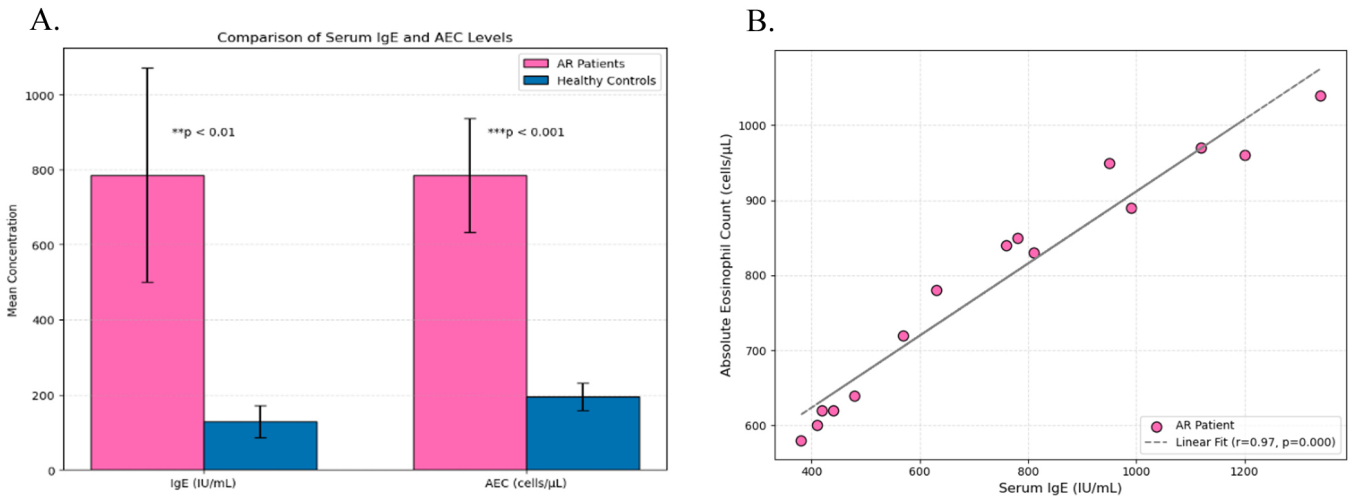

Figure 2. A. Comparative Mean Serum IgE AEC Levels in Allergic Rhinitis Patients and Healthy Control. Shows that mean serum IgE and eosinophil counts are much higher in allergic rhinitis patients than in healthy controls, confirming a stronger allergic and inflammatory response in the patient group. B. Individual Scatter Plot for Correlation between Serum IgE and AEC in Allergic Rhinitis Patients. These results show a strong positive correlation between serum IgE levels and eosinophil counts in allergic rhinitis patients, indicating that higher IgE is associated with higher eosinophil counts.

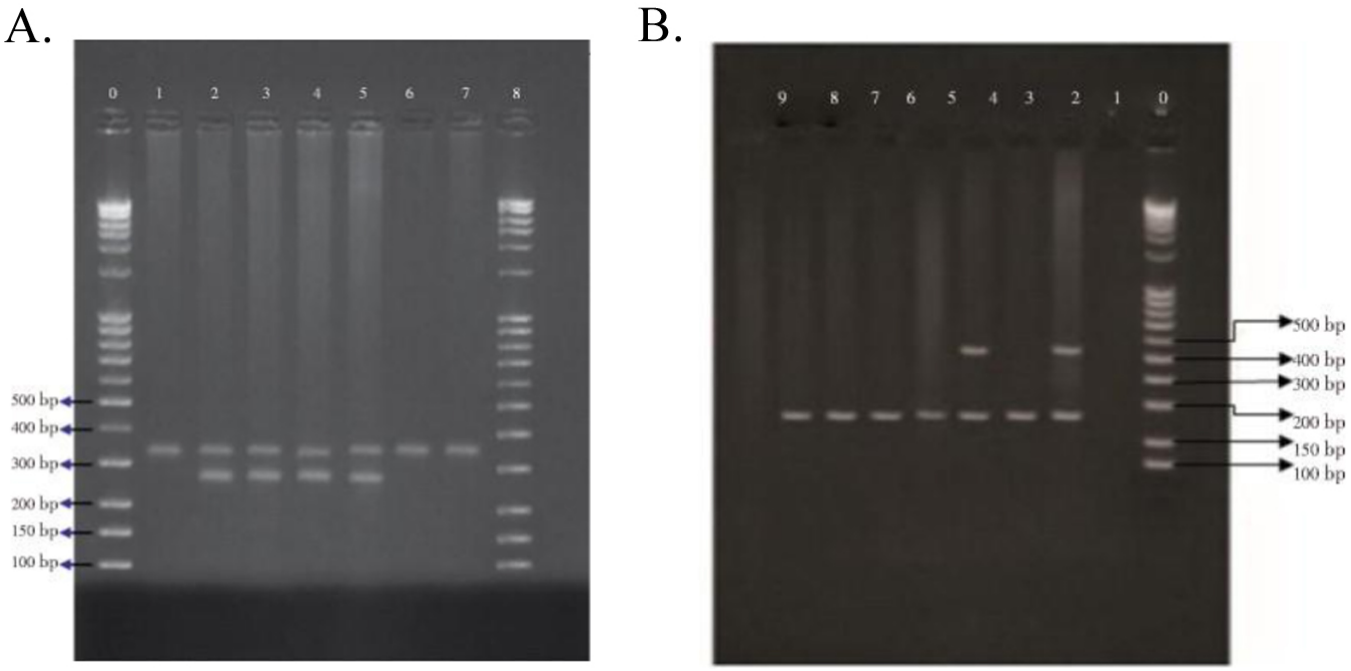

Figure 3. A. PCR assay for GSTM1 gene polymorphism. Lanes 2, 3, 4, and 5 display the 273 bp band indicating GSTM1-positive genotypes, while lanes 1, 6, and 7 lack this band, confirming GSTM1 null genotypes. The internal control band is visible in all lanes, validating the PCR results. B. PCR assay for GSTT1 gene polymorphism. Lanes 2 and 4 display a visible GSTT1 amplicon band, indicating GSTT1-positive genotypes. In contrast, lanes 3, 5, 6, 7, and 8 lack the specific GSTT1 band, which identifies these samples as GSTT1 null genotypes. The presence of the internal control gene CYP3A53 band in every lane confirms successful DNA amplification and assay reliability.

Table 2. Etiological classification of rhinitis

Factors	Description	Reference
IgE-mediated (allergic)	IgE-mediated inflammation of the nasal mucosa, resulting in eosinophilic and Th2-cell infiltration of the nasal lining. Further classified as intermittent or persistent	Bousquet et al., 2008 [12]
Autonomic	Vasomotor; Drug-induced (rhinitis medicamentosa); Hypothyroidism; Hormonal; Non-allergic rhinitis with eosinophilia syndrome (NARES)	Baraniuk & Shusterman, 2007 [13]
Infectious	Precipitated by viral (most common), bacterial, or fungal infection	Eccles, 2005 [14]
Idiopathic	Etiology cannot be determined	Baraniuk & Shusterman, 2007 [13]

(AIT). Additionally, 33.3% of patients reported concurrent use of complementary or alternative therapies such as Ayurveda or homeopathy. Bar chart (Figure 2A) illustrated mean serum IgE and AEC concentrations with standard deviation error bars for AR patients and matched healthy controls. Pink bars represent AR patients; blue bars represent controls. Significant differences were noted in both parameters ($p<0.01$ for IgE, $p<0.001$ for AEC), highlighting higher immunologic activity in allergic rhinitis. These differences confirm the presence of IgE mediated sensitization and eosinophilic inflammation in AR cases, reliable with a Th2-dominant immunopathological profile. Notably increased in both markers emphasizes their utility as diagnostic and severity indicators in allergic rhinitis.

Individual data points for serum IgE (IU/ml) plotted against AEC (cell/μL) among AR patients are presented in Figure 2B. The linear regression line indicates a moderate positive correlation ($r=0.648$, $p<0.01$), confirming the association between IgE-mediated sensitization and eosinophilic inflammation. This correlation highlighted that the immunological linkage between humoral sensitization and cellular inflammation, which provided evidence that these biomarkers rise in parallel during allergic response. Such association supports combined use in clinical stratification and may aid in identifying patients with more active or severe disease. Based on established literature, the etiological classification of rhinitis is presented in Table 2.

3.4 Susceptibility to allergic rhinitis

Allergies are mostly inherited (passed down through families). It is more likely to have hay fever if a parent or family member has allergies. People who have asthma or eczema are more likely to develop hay fever. According to a hypothesis, lack of microbial exposure in childhood may lead to modified immunity toward T helper 2 (Th2) skewing causing increased risk for asthma & atopic diseases. This may explain the role of immunotherapy in AR patients in the prevention of asthma development [1].

Allergies can occur in about 20–30% of women during pregnancy [15,16]. AR typically presents at a younger age and more common in boys. Seasonal rhinitis is more prevalent among children, but adults are more affected by perennial rhinitis.

Around one fifth of individuals with rhinitis develop asthma in their later life. Individuals sensitized with perennial allergens (dust mite) are more prone to develop asthma than individuals having sensitization with seasonal allergens (pollen grains) [1]. The prevalence of allergen sensitization progressively decreased with age after peaking at between 20 and 29 years as reported by the authors [17]. The sensitization rate was higher in males than in females ($P = .046$). The sensitization rate to house dust mites decreased with age, while sensitization to mugwort and ragweed increased. The study also reported that - Nasal obstruction tended to decrease with age and was more prevalent in males ($P = .002$) than in females, while rhinorrhea ($P = .007$) and itching ($P = .013$) were more prevalent in females. Total nasal symptom scores did not differ by sex [17].

3.5 Genotyping of GSTM1 and GSTT1 Genetic Polymorphisms

Genomic DNA was successfully extracted from peripheral blood samples of AR patients and healthy patients as control. Genotyping for GSTM1 and GSTT1 polymorphisms was performed using PCR and band patterns were observed through agarose gel electrophoresis. Figure 3 A shows the PCR assay for GSTM1 where Lane 2, 3, 4, and 5 showed a distinct

Table 3. Summary of Therapeutic Interventions for Allergic Rhinitis

Class	Drugs/Methods	Mechanism	Advantages	Side Effects
Antihistamines (Oral)	Cetirizine, Loratadine, Fexofenadine	Blocks H1 histamine receptors	Fast-acting; non-sedating types preferred	Drowsiness, dry mouth
Intranasal Antihistamines	Azelastine, Olopatadine	Locally blocks histamine activity	Rapid onset; effective for congestion	Bitter taste, irritation
Intranasal Corticosteroids	Fluticasone, Budesonide, Mometasone	Reduces inflammation	Most effective for moderate to severe AR	Nasal dryness, epistaxis
Decongestants	Pseudoephedrine, Oxymetazoline	Vasoconstriction of nasal vessels	Short-term relief	Rebound congestion
Leukotriene Antagonists	Montelukast	Blocks leukotrienes	Useful with asthma	Headache, mood changes
Immunotherapy (AIT)	SCIT or SLIT	Induces long-term immune tolerance	Long-term benefit	Local reactions, rare anaphylaxis
Mast Cell Stabilizers	Cromolyn sodium	Prevents mast cell degranulation	Safe profile	Nasal irritation
Saline Irrigation	Isotonic/hypertonic saline	Clears allergens mechanically	Safe for daily use	Minimal
Complementary Therapies	Ayurveda, Homeopathy	Variable anti-inflammatory	Widely used in India	Variable; herb-drug interactions
Lifestyle Control	Allergen avoidance, air filters	Reduces exposure	Foundational strategy	Requires assessment

band at 273 bp, indicates the presence of the GSTM1 gene. Whereas, Lanes 1, 6, and 7 lacked this band, representing the GSTM1 null genotype. However, 0 and 8 contained the molecular marker and a band at 346 bp confirmed amplification of the internal control gene. Similarly, Figure 3 B illustrates the PCR assay for GSTT1 where Lane 2 and 4 displayed bands that correspond to the GSTT1 positive genotype, while lanes 3, 5, 6, 7, and 8 lacked the expected band, indicating the GSTT1 null genotype. The internal control gene CYP3A53 was successfully amplified in all lances, confirming the reliability of the PCR reactions. GSTT1 genes, both of which are involved in drug detoxification processes. The presence of null genotypes in several AR patients was observed suggesting a potential genetic susceptibility, possible linked to reduced enzymatic detoxification capacity.

4 Discussion

4.1 Epidemiology, Demographics and Environmental Influences

AR is one of the most common chronic respiratory conditions, particularly predominant in densely populated urban and environmentally challenged regions of Telangana. The present case control study aimed to investigate the clinical profiles, risk factors, and therapeutic approaches among individuals diagnosed with AR in the Telangana region. Allergic rhinitis remains a considerable public health concern in the Telangana region, influenced by a complex interaction of environmental exposures and genetic tendency. The findings of this study showed the regional heterogeneity in AR expressions and also reveal strong environmental dependency of allergic diseases and genetic influences underlying disease susceptibility.

The clinical profiles identified in this study reflect a spectrum of persistent symptoms, frequent comorbid allergic conditions, and compromised quality of life, underscore the chronic, multisystemic nature of the disease. Despite the availability of guideline-recommended therapies such as antihistamines and intranasal corticosteroids, limitations in treatment adherence, restricted access to specialist care, and socioeconomic constraints continue to hinder effective disease management.

The demographic distribution observed in this study revealed a higher prevalence of AR among individuals aged 15–45 years, this brings into line with global epidemiological trends that showed peak onset of AR during adolescence and early adulthood [18]. Urban residence was found to be significantly associated with AR cases, likely due to increased exposure to traffic-related air pollutants and reduced microbial diversity in urban dwellings [19]. Environmental

factors such as exposure to house dust mites, pollen from Parthenium hysterophorus, Prosopis juliflora, and ambient agricultural dust were common triggers, consistent with aerobiological studies conducted in semi-arid regions of South India [20].

4.2 Immunological Response

Clinically, perennial rhinitis with seasonal aggravation is the most common pattern, and frequent comorbidities conditions such as asthma, chronic sinusitis, and atopic dermatitis were commonly observed, this supports the unified airway hypothesis that shared immunological pathways impact both upper and lower respiratory tracts [21] The immunological assessment of this study revealed elevated serum IgE levels and eosinophil counts in all 15 patients. This strengthens the dominant the Th2-skewed immune response and highlights IgE and AEC characteristic of allergic disorders [22]. These parameters, along with clinical symptoms and allergen-specific skin prick testing, will be served as valuable diagnostic markers in resource-limited settings.

4.3 Treatment Patterns and Limitations

As summarized in Table 3, oral antihistamines and intranasal corticosteroids were the most commonly prescribed medications as the cornerstone therapies, this reflects current ARIA guidelines. Nevertheless, adherence to treatment was notably suboptimal, primarily due to influenced by misconceptions about steroid use or steroid phobia, treatment fatigue, lack of follow-up and financial constraints. Despite their efficacy, Allergen-Specific Immunotherapy (AIT) was underutilized, may be due to limited accessibility and low patient awareness [23]. Additionally, a considerable number of patients reported using complementary and alternative therapeutic medicines such as Ayurveda and homeopathy, highlighting the importance of integrative healthcare approaches in this region [24]. Whereas such modalities hold cultural significance, their efficacy remains variable and evidence limited, this calling for integrative frameworks that balance traditional wisdom and evidence base care [24].

The findings of this case–control study highlight the urgent need to establish region-specific allergy care frameworks, including community-based centers equipped to deliver diagnostic services, patient education, and tailored pharmacologic interventions. Integrating traditional healthcare practices within evidence-based models may further support patient engagement and cultural alignment.

4.4 Genetic Variability of Allergic Rhinitis

This study investigated the polymorphisms of GSTM1 and GSTT1 genes in AR patients. It is revealed that a notable frequency of null genotypes among affected individuals. The absence of amplification bands for GSTM1 (273 bp) and GSTT1 (459 bp) in several samples suggested gene deletions that may weaken enzymatic detoxification. These findings align with the rising evidence that connects glutathione S-transferase (GST) gene variants in the pathogenesis of allergic and respiratory conditions.

Similar study was conducted by Birbian et al., [25]., where GSTM1 and GSTT1 null genotypes were significantly associated with increased risk of asthma in a North Indian population, which included a subcategory of patients with allergic rhinitis. The GSTT1 null allele was present in 40% of asthma patients compared to 13.3% of controls, yielding an odds ratio of 4.35. Similarly, the GSTM1 null genotype showed a modest but significant association with disease susceptibility (OR = 1.34). These results supported the hypothesis that deletions in GST genes compromise the body's ability to neutralize reaction oxygen species, thereby contributing to chronic airway inflammation. Further, Jangala et al., [26] examined GST gene variants in individuals with allergic chronic rhinosinusitis (CRS), a condition closely related to AR. Their results revealed a strong association between GSTM1 deletion and asthma in allergic CRS cases (OR = 1.82), and an even higher risk when both GSTM1 and GSTT1 were absent (OR = 2.58). These findings highlight the synergistic impact of multiple GST gene deletions on respiratory disease susceptibility. However, our findings are in line with the studies that reinforce the role of GSTM1 and GSTT1 polymorphisms as genetic risk factors in allergic rhinitis. The presence of null genotype in AR patients suggests a compromised antioxidant defence, which may impair inflammatory responses to environmental allergens. This genetic tendency highlights the importance of integrating molecular diagnostics into the clinical evaluation of AR, potentially guiding personalized treatment strategies.

5 Conclusion

This study highlights the multifactorial etiology of AR and the need for integrated, targeted, region-specific interventions management protocols. The case–control

design allowed for the identification of environmental and genetic risk factors, as well as clinical patterns, that are specific to the Telangana population and can support predictive screening models and advise therapeutic decision making. Further research can be done on longitudinal cohort analyses and molecular profiling including genomic and epigenetic markers to facilitate early diagnosis and enable precision medicine approaches. Additionally, population-level strategies have focused on mitigating urban environmental risk factors could significantly reduce allergen burden and disease occurrence. By providing data-driven insights into the immunological, clinical, and therapeutic dimensions of allergic rhinitis in Telangana, this study contributed for targeted interventions and gives meaningful strategies to national efforts in allergic disease prevention and control. The observed prevalence of GSTM1 and GSTT1 null genotypes among allergic rhinitis patients highlights a potential genetic tendency linked to decreased detoxification pathways, that strengthening the role of molecular profiling in understanding disease susceptibility.

Conflicts of Interest

The authors declare that they have no conflicts of interest.

Ethical Approval

Ethical approval for this study was granted by the Institutional Ethics Committee (IEC) of BMMRC during its meeting on 23 December 2022.

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