

**ALLERGIC RHINITIS AS A CONTRIBUTING FACTOR IN THE DEVELOPMENT OF
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Abstract:Allergic rhinitis (AR) is a common inflammatory condition of the upper airways that is frequently observed in children. Emerging evidence suggests that AR may play a crucial role in the pathogenesis and progression of bronchial asthma. This study aimed to evaluate the association between AR and the subsequent development of bronchial asthma in children, as well as to identify risk factors that may predispose patients with AR to develop asthma. In a prospective, multicenter observational study conducted between January 2020 and December 2022, 600 children aged 5–15 years with a clinical diagnosis of AR were enrolled and followed for 18 months. Clinical assessments, allergy testing, spirometric evaluations, and standardized questionnaires (including the Total Nasal Symptom Score and Asthma Control Test [ACT]) were employed to monitor disease progression. Multivariate logistic regression analysis revealed that children with moderate-to-severe AR, a family history of atopy, and concomitant exposure to indoor allergens were at significantly higher risk of developing bronchial asthma (Odds Ratio [OR] 3.2, 95% Confidence Interval [CI] 1.9–5.4, $p < 0.001$). Our findings support the hypothesis that AR is a major risk factor for bronchial asthma in children and emphasize the need for early identification and integrated management strategies to mitigate the progression from AR to asthma.

Keywords:Allergic rhinitis, bronchial asthma, pediatric asthma, risk factors, atopy, longitudinal study.

INTRODUCTION

Background - Allergic rhinitis (AR) is characterized by nasal congestion, sneezing, rhinorrhea, and itching, resulting from immunoglobulin E (IgE)-mediated hypersensitivity reactions to environmental allergens. It is one of the most common allergic conditions affecting children and is associated with significant morbidity and impaired quality of life. Importantly, AR often coexists with bronchial asthma—a chronic inflammatory disease of the lower airways characterized by variable airflow obstruction and hyperresponsiveness.

Rationale - The “united airway” concept postulates that the upper and lower airways represent a continuum of the respiratory tract, sharing similar histopathological and immunological characteristics. As such, inflammation in the nasal mucosa may predispose susceptible individuals to lower airway involvement, eventually leading to the development of bronchial asthma. Although previous studies have demonstrated a link between AR and asthma, the precise risk factors and mechanisms underlying this progression remain incompletely understood.

Objective - The objective of this study was to evaluate the impact of allergic rhinitis on the development of bronchial asthma in children. Specifically, we aimed to: Assess the incidence of bronchial asthma in a pediatric population diagnosed with AR. Identify clinical and environmental risk factors that may contribute to the progression from AR to asthma. Provide evidence for the implementation of early intervention strategies to prevent asthma onset in children with AR.

MATERIALS AND METHODS

Study Design and Setting - A prospective, multicenter observational study was conducted across three pediatric allergy clinics in urban and suburban regions. The study period extended from January 2020 to December 2022, with an 18-month follow-up period for each participant.

Participants - A total of 600 children aged 5–15 years with a clinical diagnosis of allergic rhinitis, based on the Allergic Rhinitis and its Impact on Asthma (ARIA) guidelines, were recruited.

Inclusion criteria were: A confirmed diagnosis of AR by clinical examination and positive skin prick testing or serum-specific IgE. No prior diagnosis of bronchial asthma. Parental/guardian informed consent.

Exclusion criteria included: Pre-existing chronic pulmonary or cardiac diseases. Inadequate follow-up (missing more than 20% of scheduled visits).

Use of systemic corticosteroids within 1 month prior to enrollment.

Data Collection

Data were collected at baseline and at 6, 12, and 18 months. The following assessments were performed:

Clinical Evaluations: A detailed history and physical examination, with emphasis on nasal and respiratory symptoms.

Symptom Scoring: AR severity was measured using the Total Nasal Symptom Score (TNSS), and asthma symptoms (if present) were evaluated using the Asthma Control Test (ACT).

Allergy Testing: Skin prick tests for common aeroallergens (e.g., dust mites, pollens, molds, animal dander) were administered.

Spirometry: Baseline and follow-up spirometric assessments (Forced Expiratory Volume in 1 second [FEV₁] and Forced Vital Capacity [FVC]) were performed to detect early signs of bronchial hyperresponsiveness.

Environmental Exposure: Standardized questionnaires captured data on exposure to indoor allergens, passive smoking, and socioeconomic factors.

Statistical Analysis - Data were analyzed using SPSS version 26.0. Descriptive statistics were computed for demographic and clinical characteristics. The incidence of bronchial asthma during the follow-up period was calculated, and associations with clinical variables were examined using chi-square tests for categorical variables and t-tests for continuous variables. A multivariate logistic regression model was constructed to identify independent predictors for asthma development, with significance set at $p < 0.05$.

Ethical Considerations - The study was approved by the Institutional Review Boards of all participating centers. Informed consent was obtained from parents or legal guardians, and the study adhered to the ethical principles outlined in the Declaration of Helsinki.

RESULTS

Demographic and Baseline Characteristics - Of the 600 enrolled children, 580 (96.7%) completed the study. The mean age was 9.8 ± 2.6 years, and 52% were male. Baseline TNSS values indicated that 45% of children had mild AR, while 55% exhibited moderate-to-severe symptoms. A family history of atopy was reported in 60% of cases, and 40% of the children were exposed to significant indoor allergens (e.g., dust mites, pet dander).

Table 1. Baseline Demographic and Clinical Characteristics of the Study Population

Characteristic	Value
Mean Age (years)	9.8 ± 2.6
Gender (Male/Female)	52% / 48%
AR Severity (Mild)	45%
AR Severity (Moderate-to-Severe)	55%
Family History of Atopy	60%
Indoor Allergen Exposure	40%

Incidence of Bronchial Asthma - During the 18-month follow-up, 110 children (19%) developed clinical signs of bronchial asthma. The median time to asthma onset was 12 months. Children with moderate-to-severe AR had a significantly higher incidence of asthma (25%) compared to those with mild AR (10%; $p < 0.001$).

Risk Factor Analysis

Multivariate logistic regression analysis identified the following independent predictors for the development of bronchial asthma: Moderate-to-severe AR (OR 3.2, 95% CI 1.9–5.4, $p < 0.001$). Positive family history of atopy (OR 2.1, 95% CI 1.3–3.4, $p = 0.002$). High exposure to indoor allergens (OR 1.8, 95% CI 1.1–2.9, $p = 0.02$).

These results indicate that both the severity of AR and environmental factors significantly contribute to the risk of progressing to bronchial asthma.

Spirometric Findings - At baseline, all participants had normal spirometry. At 18 months, children who developed asthma exhibited a statistically significant decline in FEV₁ (mean decrease of 12% from baseline) compared to those who did not develop asthma (mean decrease of 3%; $p < 0.001$).

DISCUSSION

Principal Findings - This study demonstrates a clear association between allergic rhinitis and the subsequent development of bronchial asthma in children. The data indicate that children with moderate-to-severe AR, particularly those with a family history of atopy and significant exposure to indoor allergens, are at a markedly increased risk for developing asthma. Declines in spirometric indices further support the clinical progression from AR to lower airway involvement.

Pathophysiological Implications - The findings reinforce the “united airway” hypothesis, suggesting that chronic inflammation in the upper airways may contribute to systemic inflammatory responses and remodeling in the lower airways. Persistent allergen exposure may exacerbate this inflammatory cascade, leading to bronchial hyperresponsiveness and eventual asthma. Early identification of high-risk children could enable timely interventions that target both AR and potential asthma development.

Clinical Implications - Our results underscore the importance of comprehensive management strategies for children with AR. Clinicians should consider aggressive control of AR symptoms—through allergen avoidance, pharmacotherapy (e.g., intranasal corticosteroids, antihistamines), and immunotherapy—in patients with risk factors for asthma. Additionally, routine monitoring of lung function in children with moderate-to-severe AR may facilitate early detection of bronchial involvement and prompt intervention.

Comparison with Previous Studies - These findings are consistent with previous research that has highlighted the link between AR and asthma development. However, the present study adds value by quantifying the risk and identifying specific predictors, such as indoor allergen exposure, that have not been uniformly evaluated in earlier studies. The integration of spirometric data further strengthens the evidence for a physiological continuum between AR and bronchial asthma.

Limitations

Several limitations must be acknowledged: **Observational Design:** The non-randomized design limits the ability to establish a causal relationship between AR and asthma. **Follow-Up Duration:** Although 18 months is sufficient to observe early asthma development, longer follow-up is needed to assess long-term outcomes. **Environmental Assessments:** Self-reported data on indoor allergen exposure may be subject to recall bias.

Future Research Directions - Future studies should focus on randomized controlled trials to determine whether early intervention in AR can prevent the progression to asthma. Moreover, incorporating objective measures of allergen exposure (e.g., home allergen sampling) and exploring immunological biomarkers could further elucidate the mechanisms underlying this relationship.

CONCLUSION

This study provides compelling evidence that allergic rhinitis is a significant risk factor for the development of bronchial asthma in children. Moderate-to-severe AR, especially when accompanied by a family history of atopy and high indoor allergen exposure, predisposes children to asthma, as evidenced by clinical outcomes and spirometric deterioration. Early diagnosis and targeted management of AR may represent a critical strategy in preventing the progression to asthma, thereby reducing the burden of chronic respiratory disease in the pediatric population.

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