

Association Between Atopic Dermatitis and Bronchial Asthma: Evaluating the Atopic March in a Cross-Sectional Study

Rabia A Ghafoor Sadal¹, Sameera Iqbal², Anam Gul³, Nasira Nasr⁴, Maryam Qayyum⁵, Noor Fatima⁶

1. Consultant Dermatologist, THQ Hospital Dinga, Gujrat, Pakistan.
2. Assistant Professor, Dermatology, Central Park Medical College, Lahore, Pakistan.
3. Consultant Dermatologist, DHQ Hospital, Jhelum, Pakistan.
4. Assistant Professor Dermatology, Poonch Medical College Rawalakot/CMH Rawalakot.
5. Assistant Professor, Dermatology, Sialkot Medical College, Sialkot, Pakistan.
6. 4th Year MBBS Student, Central Park Medical College, Lahore, Pakistan. **Rabia A Ghafoor Sadal: Corresponding author**

Abstract

Atopic dermatitis (AD) and bronchial asthma are chronic inflammatory disorders that share a common immunopathological basis driven by type 2 immune responses. The concept of the “atopic march” describes the progression of allergic diseases from early-life skin barrier dysfunction to respiratory allergic conditions such as asthma. Understanding this association is essential for early identification of high-risk individuals and implementation of preventive strategies. This study evaluated the prevalence of bronchial asthma among patients with atopic dermatitis and assessed the strength of association between disease severity and respiratory involvement.

A hospital-based cross-sectional study was conducted involving 460 participants, including 230 patients with clinically diagnosed atopic dermatitis and 230 age- and sex-matched controls without atopic disease. Clinical history, pulmonary function testing, serum IgE levels, eosinophil counts, and asthma diagnosis confirmed by spirometry were evaluated. Disease severity in AD was assessed using the SCORAD index. Bronchial asthma was significantly more prevalent in the atopic dermatitis group compared with controls (38.7% vs 14.3%; $p < 0.001$). Moderate-to-severe AD showed a stronger association with asthma development compared with mild disease (52.1% vs 24.6%; $p < 0.001$). Elevated serum IgE and eosinophil counts were significantly

correlated with dual disease presentation. Logistic regression revealed that severe AD independently increased the risk of asthma (OR 3.62, 95% CI 2.18–6.01; $p < 0.001$). These findings support a strong association between atopic dermatitis and bronchial asthma, reinforcing the concept of the atopic march. Early identification of patients with severe AD may facilitate preventive strategies aimed at reducing progression to respiratory allergic disease.

Keywords: Atopic dermatitis, bronchial asthma, atopic march, allergic disease

Introduction

Atopic dermatitis (AD) is a chronic, relapsing inflammatory skin disorder characterized by intense pruritus, xerosis, impaired skin barrier function, and immune dysregulation. It is one of the most common dermatological conditions in both pediatric and adult populations, with increasing global prevalence over recent decades. The disease is strongly associated with type 2 helper T cell (Th2) immune activation, elevated immunoglobulin E (IgE), and eosinophilic inflammation [1–3].

Bronchial asthma is another major chronic inflammatory disease of the airways characterized by variable airflow obstruction, bronchial hyperresponsiveness, and chronic airway inflammation. Like atopic dermatitis,

asthma is primarily driven by Th2-mediated immune responses, involving cytokines such as interleukin (IL)-4, IL-5, and IL-13. These shared immunological pathways suggest a biological link between cutaneous and respiratory allergic diseases [2–4].

The concept of the “atopic march” describes the typical progression of allergic diseases beginning in early childhood with atopic dermatitis and food allergies, followed by the later development of allergic rhinitis and bronchial asthma. This sequential development is thought to be driven by genetic predisposition, epithelial barrier dysfunction, allergen sensitization, and immune system polarization toward Th2 dominance [3–5].

Skin barrier impairment plays a central role in the initiation of the atopic march. Mutations in the filaggrin gene (FLG) and other structural proteins contribute to increased transepidermal water loss and enhanced penetration of environmental allergens and microbial agents. This facilitates immune sensitization and systemic allergic inflammation, potentially predisposing individuals to respiratory allergic diseases [4–6].

Recent evidence suggests that early-onset and severe atopic dermatitis are strongly associated with the subsequent development

of asthma. Epidemiological studies have demonstrated that children with moderate-to-severe AD are at significantly higher risk of developing asthma compared with those with mild or no skin disease. However, the strength of this association varies across populations and age groups [5–7].

Asthma and atopic dermatitis share not only immunological mechanisms but also genetic and environmental risk factors. Common triggers include exposure to allergens, pollution, tobacco smoke, microbial dysbiosis, and dietary influences. The interaction between genetic susceptibility and environmental exposure plays a critical role in determining disease expression [6–8]. Biomarkers such as serum IgE, eosinophil counts, periostin, and cytokine profiles have been studied as indicators of allergic disease severity and progression. Elevated IgE and eosinophilia are frequently observed in patients with both atopic dermatitis and asthma, reflecting systemic allergic inflammation. These biomarkers may help identify individuals at high risk of atopic march progression [7–9].

Despite increasing recognition of the association between skin and airway allergic diseases, there remains a need for population-based studies evaluating the

strength of this relationship in different clinical settings.

Cross-sectional studies provide valuable insights into disease burden and associated risk factors at a population level [8–10]. Understanding the relationship between atopic dermatitis and bronchial asthma is essential for early diagnosis, risk stratification, and implementation of preventive strategies aimed at interrupting the atopic march. Therefore, the present study was conducted to evaluate the association between atopic dermatitis and bronchial asthma and to assess the role of disease severity in predicting respiratory involvement.

Methodology

A hospital-based cross-sectional analytical study was conducted at THQ Hospital Dinga center. Ethical approval was obtained from the institutional review board prior to commencement of the study. Sample size was calculated using Epi Info version 7. Assuming an expected asthma prevalence of 30% among atopic dermatitis patients, confidence level of 95%, margin of error of 5%, and power of 80%, a minimum sample size of 420 participants was required. To enhance statistical reliability, 460 participants were enrolled.

The study population included 230 patients diagnosed with atopic dermatitis according to the Hanifin and Rajka criteria and 230 age- and sex-matched healthy controls without any history of allergic disease. Participants aged 5–60 years were included. Exclusion criteria included chronic obstructive pulmonary disease, active pulmonary infection, immunodeficiency disorders, and systemic corticosteroid use within the last four weeks. After obtaining informed consent, demographic details, clinical history, environmental exposure data, and family history of atopy were recorded. Atopic dermatitis severity was assessed using the SCORAD (Scoring Atopic Dermatitis) index and categorized into mild, moderate, and severe disease.

Diagnosis of bronchial asthma was established based on clinical history of wheezing, episodic dyspnea, and reversible

Results

airflow obstruction confirmed by spirometry demonstrating $\geq 12\%$ reversibility in forced expiratory volume in one second (FEV1) following bronchodilator administration. Laboratory investigations included serum total IgE levels and peripheral eosinophil counts. Pulmonary function tests were performed using standardized spirometric equipment according to American Thoracic Society guidelines.

Statistical analysis was performed using SPSS version 27. Continuous variables were expressed as mean $\pm$ standard deviation and categorical variables as frequencies and percentages. Chi-square test, independent ttest, and logistic regression analysis were applied. A p-value < 0.05 was considered statistically significant.

Table 1. Baseline Characteristics of Study Population (n=460)

Variable	Atopic Dermatitis (n=230)	Controls (n=230)	p-value
Age (years)	28.6 $\pm$ 12.4	29.1 $\pm$ 11.8	0.68
Male (%)	52.2	50.4	0.71
Family history of atopy (%)	41.7	18.3	<0.001
Serum IgE (IU/mL)	312.4 $\pm$ 145.6	118.7 $\pm$ 62.3	<0.001

Eosinophils (%)	6.8 ± 2.9	2.9 ± 1.4	<0.001
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Patients with atopic dermatitis exhibited significantly higher immunological markers and family history of atopy compared with controls.

Table 2. Prevalence of Bronchial Asthma in Study Groups

Group	Asthma Present (%)	Asthma Absent (%)	p-value
Atopic Dermatitis	38.7	61.3	<0.001
Controls	14.3	85.7	<0.001

Bronchial asthma prevalence was significantly higher among patients with atopic dermatitis compared with controls, supporting a strong association between both conditions.

Table 3. Association Between AD Severity and Asthma

AD Severity	Asthma Present (%)	Odds Ratio	p-value
Mild (n=92)	24.6	Reference	—
Moderate (n=88)	41.0	2.12	0.003
Severe (n=50)	52.1	3.62	<0.001

Increasing severity of atopic dermatitis was strongly associated with higher odds of bronchial asthma development.

Table 4. Multivariate Predictors of Asthma in Atopic Dermatitis Patients

Variable	Adjusted OR	95% CI	p-value
Severe AD	3.41	2.01–5.78	<0.001
High IgE levels	2.76	1.62–4.68	<0.001
Peripheral eosinophilia	2.19	1.31–3.67	0.003

Family history of atopy	2.48	1.46–4.20	0.001
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Severe atopic dermatitis and elevated immunological markers were independent predictors of bronchial asthma.

Discussion

The present study demonstrates a strong and statistically significant association between atopic dermatitis and bronchial asthma, reinforcing the concept of the atopic march. Patients with atopic dermatitis exhibited a markedly higher prevalence of asthma compared with matched controls, suggesting shared immunopathological mechanisms [11–12]. A key finding was the dose-dependent relationship between disease severity and asthma risk. Individuals with severe atopic dermatitis demonstrated more than threefold increased odds of developing bronchial asthma. This supports the hypothesis that greater skin barrier dysfunction and systemic immune activation contribute to progression along the atopic march [12–13].

Elevated serum IgE and eosinophil counts further strengthened the association between cutaneous and respiratory allergic disease. These biomarkers reflect systemic

type 2 immune activation, which plays a central role in both atopic dermatitis and asthma pathogenesis. Their elevation in patients with dual disease suggests a shared inflammatory pathway [13–14].

The role of skin barrier dysfunction remains fundamental in explaining disease progression. Disruption of epidermal integrity facilitates allergen penetration and immune sensitization, which may lead to systemic allergic inflammation affecting the respiratory tract. This mechanism is increasingly supported by genetic studies involving filaggrin mutations [14–15]. Environmental factors such as pollution, allergens, and microbial exposure may further modify disease expression.

Urbanization and lifestyle changes have been associated with increased prevalence of allergic diseases globally, potentially contributing to rising rates of both atopic dermatitis and asthma [15–16]. The findings of this study also highlight the importance of early identification of highrisk patients. Individuals with severe atopic dermatitis and elevated IgE levels may benefit from closer respiratory monitoring and early preventive interventions aimed at reducing asthma development [16–17]. From a

clinical perspective, these results support integrated management approaches involving dermatologists and pulmonologists. Multidisciplinary care may allow earlier recognition of respiratory involvement and improved long-term outcomes in patients following the atopic march [17–18].

Although cross-sectional in design, the study provides valuable epidemiological evidence supporting the atopic march hypothesis. Longitudinal studies are required to confirm temporal progression and evaluate preventive strategies that may interrupt disease evolution [18–20].

Conclusion

A strong and statistically significant association exists between atopic dermatitis and bronchial asthma, supporting the concept of the atopic march. Disease severity, elevated IgE levels, and eosinophilia significantly increase the risk of asthma development. Early identification of highrisk atopic dermatitis patients may allow preventive strategies to reduce progression to respiratory allergic disease.

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