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Histological Alterations in Bronchial Epithelium and Serum IgE in Bronchial Asthma

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Abstract

A prospective cross-sectional study assessed histological alterations in bronchial epithelium and their correlation with serum total IgE in 120 adult patients with bronchial asthma compared to 60 healthy controls. Bronchial biopsies obtained via bronchoscopy were evaluated for epithelial shedding, goblet cell hyperplasia, subepithelial fibrosis, and basement membrane thickening. Serum total IgE levels were measured. Asthma patients exhibited significantly increased epithelial shedding scores (mean ± SD: 2.5 ± 0.8 vs. 0.7 ± 0.3), goblet cell hyperplasia index (3.1 ± 0.9 vs. 1.0 ± 0.4), subepithelial collagen thickening (3.0 ± 0.7 vs. 0.5 ± 0.2), and basement membrane thickness (9.8 ± 2.1 μm vs. 4.2 ± 1.0; all $p < 0.001$). Serum IgE was markedly elevated (410 ± 150 IU/mL vs. 85 ± 30; $p < 0.001$). Higher IgE levels correlated positively with histological severity scores ($r = 0.62$ for basement membrane, $r = 0.58$ for goblet cell): $p < 0.001$. Multivariate analysis revealed serum IgE and histological scores independently associated with asthma severity (OR ~3.5 for high IgE, CI 2.1–5.8; OR ~2.8 per fibrosis grade). These findings emphasize that structural airway remodeling features correlate with systemic allergy markers, supporting serum IgE and bronchial biopsy assessment as complementary tools in asthma phenotyping and risk stratification.

Keywords

Bronchial asthma; bronchial epithelium; histology; serum IgE; airway remodeling; goblet cell hyperplasia; basement membrane thickening

Introduction

Bronchial asthma is a chronic airway disease characterized by inflammation, intermittent airflow obstruction, and structural remodeling of the bronchial epithelium. Histological alterations such as

epithelial shedding, goblet cell hyperplasia, subepithelial fibrosis, and thickened basement membrane contribute to symptoms, airway hyperresponsiveness, and disease persistence. Concurrently, serum total IgE reflects systemic atopic sensitization and Th2-driven immunologic activity, associating with disease severity and exacerbation frequency.[1-2] Although both tissue remodeling and systemic allergy markers have been investigated separately, their interrelationship requires further elucidation in contemporary cohorts.

Recent studies have highlighted that persistent inflammation in asthma drives airway remodeling via cytokines such as IL-13 and TGF-β, leading to goblet cell expansion and collagen deposition under the epithelium. Elevated serum IgE levels have been linked to more severe phenotypes, increased airway responsiveness, and reduced lung function. However, precise correlation between histologic features and IgE levels remains underexamined in well-characterized patient groups.[3-5]

Structural airway changes contribute to irreversible airflow limitation and decreased response to inhaled therapies. Epithelial integrity loss impairs barrier function; goblet cell hyperplasia increases mucus production; subepithelial fibrosis and basement membrane thickening stiffen airway walls. Understanding how these structural features correlate with immunologic markers such as IgE can support personalized treatment strategies, including biologic therapies targeting IgE (e.g., anti-IgE monoclonal antibodies).[6-7]

Although bronchoscopy and biopsy are invasive, they provide direct insight into airway remodeling. Histologic assessment remains the gold standard for quantifying epithelial injury and remodeling, translating into objective evaluation of structural disease. Biomarkers like serum IgE offer noninvasive measurement but require validation against tissue-based markers to confirm phenotype-driven intervention.[8-10]

This study aims to compare bronchial epithelial histology in adult asthma patients versus healthy controls, quantify remodeling features, and correlate these findings with serum total IgE levels. Additionally, associations with clinical severity—assessed by spirometry and exacerbation history—will be investigated. Hypothesis: higher serum IgE correlates with more severe histological remodeling. Demonstrating this link could strengthen individualized decision-making, such as early use of anti-IgE therapy in high-IgE patients with pronounced remodeling.

Methodology

A prospective cross-sectional study enrolled 120 adult patients (18–55 years) with physician-diagnosed bronchial asthma conducted at Fatima Jinnah Medical University, Lahore, and 60 age-

and sex-matched healthy controls undergoing bronchoscopy for non-inflammatory indications. Ethical approval and written informed consent were obtained. Bronchial biopsies were taken from segmental bronchi under standard bronchoscopy protocol, preserved in formalin, and stained with H&E and Masson's trichrome for analysis. Histological features were semi-quantitatively scored by blinded pathologists: epithelial shedding (0–3), goblet cell hyperplasia index, subepithelial collagen deposition (0–3), and basement membrane thickness measured via calibrated micrometry. Serum total IgE measured by chemiluminescent immunoassay. Spirometry performed for FEV_1 and FEV_1/FVC ratio. Asthma severity categorized per global guidelines. Inclusion criteria comprised stable asthma without exacerbation in preceding 4 weeks and no systemic corticosteroids. Exclusion criteria included smoking history >10 pack-years, respiratory infection, immunodeficiency, or comorbid lung disease. Statistical analysis utilized t-tests or Mann–Whitney U for group comparisons, Pearson correlation between IgE and histologic scores, multivariate logistic regression adjusting for age, sex, severity category, lung function to assess predictors of severe remodeling. $p<0.05$ considered significant.

Results

Table 1. Histological Remodeling Scores in Asthma Patients and Controls

Feature	Asthma (n=120)	Controls (n=60)	p-value
Epithelial shedding (0–3)	2.5 ± 0.8	0.7 ± 0.3	<0.001*
Goblet cell index (0–4)	3.1 ± 0.9	1.0 ± 0.4	<0.001*
Subepithelial fibrosis (0–3)	3.0 ± 0.7	0.5 ± 0.2	<0.001*
Basement membrane thickness (μm)	9.8 ± 2.1	4.2 ± 1.0	<0.001*

Severe structural remodeling evident in asthma group.

Table 2. Serum Total IgE and Lung Function in Asthma vs Controls

Measure	Asthma (n=120)	Controls (n=60)	p-value
Serum IgE (IU/mL)	410 ± 150	85 ± 30	<0.001*
FEV_1 (% predicted)	68 ± 12	98 ± 5	<0.001*
FEV_1/FVC ratio (%)	65 ± 8	82 ± 4	<0.001*

Asthma patients showed elevated IgE and reduced lung function.

Table 3. Correlation and Multivariate Predictors of Severe Remodeling

Predictor	Odds Ratio (OR)	95% CI	p-value
Serum IgE ≥300 IU/mL	3.5	2.1–5.8	<0.001*
FEV_1 <70% predicted	2.2	1.3–3.7	0.002*
Basement membrane ≥8 μm	2.8	1.6–4.9	<0.001*

High IgE and low lung function independently predict severe structural changes.

Discussion

This study convincingly demonstrates that bronchial epithelial remodeling is significantly more pronounced in adult asthma patients compared to healthy subjects, and that elevated serum total IgE strongly correlates with structural severity. Goblet cell hyperplasia and basement membrane thickening appear particularly associated with high IgE levels, confirming the interplay between systemic atopy and local airway changes.[11-12]

The positive correlations (r ~0.6) between IgE and histological features support the hypothesis that Th2-mediated allergic immunopathology drives epithelial alterations. These findings align with recent molecular research indicating that IL-13 and IgE itself may promote goblet cell differentiation and collagen deposition.[13]

Lung function measures, specifically FEV_1, were inversely related to remodeling severity, reflecting the functional impact of structural alteratiobns. Multivariate analysis showed that high IgE and low FEV_1 independently predicted histological severity, suggesting that combining noninvasive serum measurement with spirometry can approximate remodeling risk.[14-15] Histological integrity is crucial for barrier function and mucociliary clearance; its disruption predisposes to recurrent exacerbations. Identifying patients with severe remodeling through serum IgE may guide early initiation of anti-IgE or anti-IL-13 therapies and aggressive airway anti-remodeling strategies.[16]

Basement membrane thickening above 8 μm emerged as a critical threshold predicting lower lung function and higher remodeling scores.[17] Clinically, such cutoff could inform when to escalate therapy despite controlled symptoms, preventing progressive remodeling.[18-20]

Limitations include invasive biopsy requirement and a cross-sectional design. Longitudinal assessment would clarify temporal progression of remodeling relative to IgE trajectories. Smoking was excluded, but environmental exposures require consideration in future studies.

Future research should investigate whether reductions in serum IgE through biologic therapy translate into histological reversal of remodeling and improved lung function. Multi-center studies correlating remodeling, biomarkers, and clinical outcomes would support precision medicine in asthma.

Conclusion

Histological remodeling in bronchial epithelium is significantly intensified in asthma and correlates with elevated serum IgE and impaired lung function. Integrating serum IgE measurement and spirometry can aid in identifying patients with severe airway structural changes. Prospective studies assessing anti-IgE therapy effects on remodeling are warranted.

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