Bulletin of Environment, Pharmacology and Life Sciences
Bull. Env. Pharmacol. Life Sci., Vol 15 [9] August 2026. 131-137

Online ISSN 2277-1808
Journal's URL: http://www.bepls.com
CODEN: BEPLAD

ORIGINAL ARTICLE **OPEN ACCESS**

Evaluation of Bronchial Epithelial Histological Changes and Serum IgE Levels in the Pathogenesis of Bronchial Asthma

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ABSTRACT

Bronchial asthma is a chronic inflammatory airway disease characterized by epithelial remodeling, airway hyperresponsiveness, and variable airflow limitation. This prospective observational study evaluated bronchial epithelial histological changes and total serum immunoglobulin E levels in two hundred and eighty adult patients presenting with chronic asthma symptoms compared to sixty healthy controls. Endobronchial mucosal biopsies and serum samples were analyzed to quantify epithelial desquamation, basement membrane thickening, goblet cell hyperplasia, submucosal inflammatory cell infiltration, and total serum immunoglobulin E concentrations across clinical severity grades. Mean serum immunoglobulin E levels were markedly elevated in asthmatic patients compared to healthy controls (412.5 ± 186.2 IU/mL vs. 48.2 ± 19.6 IU/mL, $p < 0.001$). Histopathological assessment demonstrated significant reticular basement membrane thickening (8.4 ± 2.6 µm vs. 3.2 ± 0.8 µm, $p < 0.001$) and extensive epithelial shedding in severe asthmatics. Serum immunoglobulin E levels correlated positively with basement membrane thickness ($r = 0.62$, $p < 0.001$) and goblet cell hyperplasia scores ($r = 0.58$, $p < 0.001$). Multivariable linear regression identified baseline serum immunoglobulin E ($\beta = 0.44$, $p < 0.001$), eosinophilic submucosal infiltration ($\beta = 0.38$, $p < 0.001$), and disease duration ($\beta = 0.22$, $p = 0.002$) as independent predictors of structural epithelial remodeling. Structural bronchial injury and elevated immunoglobulin E serve as central drivers of persistent airway inflammation, offering crucial targets for phenotype-guided biological therapies.

Keywords: *Bronchial asthma, epithelial remodeling, serum immunoglobulin E, basement membrane thickness, goblet cell hyperplasia*

Received 12.07.2026 Revised 30.07.2026 Accepted 24.08.2026

INTRODUCTION

Bronchial asthma is a heterogeneous chronic inflammatory disease of the airways defined by recurrent respiratory symptoms including wheezing, dyspnea, chest tightness, and cough, accompanied by variable expiratory airflow limitation. Clinical and basic research over recent decades has firmly established that chronic airway inflammation induces structural alterations collectively termed airway remodeling [1-3]. These structural changes encompass loss of epithelial junctional integrity, subepithelial fibrosis, goblet cell metaplasia, smooth muscle hypertrophy, and neoangiogenesis. Despite widespread clinical availability of inhaled corticosteroids and long-acting bronchodilators, a substantial subset of asthmatic patients continues to experience persistent symptoms, frequent acute exacerbations, and progressive decline in lung function, highlighting the necessity of clarifying underlying pathogenic mechanisms [4].

The bronchial epithelium functions as the primary physical, immunological, and metabolic barrier against inhaled environmental allergens, pathogens, pollutants, and physical irritants. In genetically susceptible individuals, repeated environmental exposures trigger microvascular permeability, disruption of epithelial tight junction proteins, and extensive desquamation of columnar epithelial cells into the bronchial lumen. Injured bronchial epithelial cells release pro-inflammatory alarmins—including thymic stromal lymphopoietin, interleukin-25, and interleukin-33—which activate innate lymphoid cells and type 2 helper

T lymphocytes. This cascade amplifies downstream production of interleukin-4, interleukin-5, and interleukin-13, driving local tissue eosinophilia, mucous hypersecretion, and persistent submucosal inflammation. Understanding the cellular pathways of epithelial damage is vital for identifying novel therapeutic targets [5-7].

Immunoglobulin E plays a central role in mediating type I hypersensitivity reactions and orchestrating chronic allergic airway responses in asthma. Allergen-induced cross-linking of specific immunoglobulin E bound to high-affinity Fc epsilon receptors on surface mast cells and basophils triggers immediate degranulation, releasing histamine, leukotrienes, and prostaglandin D2. Furthermore, sustained systemic elevation of immunoglobulin E promotes persistent inflammatory cell recruitment into the subepithelial bronchial stroma. Although total serum immunoglobulin E levels are routinely measured in clinical practice to confirm allergic endotypes, its quantitative relationship with specific histopathological markers of epithelial structural remodeling remains incompletely defined. Correlating systemic immunoglobulin E titers with direct mucosal biopsy metrics provides critical insights into allergic disease activity [8].

Histopathological evaluation of endobronchial biopsy specimens allows direct visualization and quantitative measurement of mucosal architectural changes in asthmatic patients. Characteristic histological features include thickening and hyalinization of the reticular basement membrane, submucosal eosinophilic and lymphocytic infiltration, loss of ciliated columnar epithelial cells, and prominent enlargement of mucus-secreting goblet cells. Reticular basement membrane thickening, driven by subepithelial collagen deposition by activated myofibroblasts, reflects long-term mechanical stress and chronic tissue injury. Quantifying these microscopic parameters provides an objective basis for assessing disease severity and evaluating response to anti-inflammatory therapies [9-10].

Furthermore, understanding how biological markers like serum immunoglobulin E interact with structural tissue injury can inform precision treatment strategies in modern respiratory medicine. Monoclonal antibody therapies targeting free immunoglobulin E or type 2 inflammatory cytokines have demonstrated clinical efficacy in reducing exacerbation rates and improving forced expiratory volume. However, response rates vary considerably among patient subgroups, underscoring the need for integrated biomarker profiles that reflect both systemic immune activation and localized tissue remodeling. Combining systemic biological markers with endobronchial histopathological assessment offers a robust framework for personalizing asthma management.

To address these critical knowledge gaps, this prospective observational study was designed to evaluate bronchial epithelial histological changes and total serum immunoglobulin E levels in adult patients across varying degrees of asthma severity. By integrating serum biomarker quantification, high-resolution endobronchial histopathology, and physiological pulmonary function metrics, this investigation aims to establish independent correlates of airway remodeling and refine pathogenic risk stratification in bronchial asthma.

MATERIAL AND METHODS

This prospective observational case-control study was conducted in the Department of Anatomy, Continental Medical College, Lahore over a continuous twenty-four-month period. Adult patients aged eighteen to sixty-five years presenting with clinically and spirometrically confirmed bronchial asthma were prospectively evaluated and enrolled alongside a comparison group of healthy, non-atopic adult controls. Sample size estimation was performed prior to study initiation using Epi Info software. Based on an anticipated mean difference in reticular basement membrane thickness of 2.5 µm between asthmatic patients and healthy controls, a statistical power of eighty percent, a confidence level of ninety-five percent, and an anticipated correlation coefficient of at least 0.50 between serum immunoglobulin E and subepithelial collagen deposition, the minimum required sample size was calculated as two hundred and forty asthmatic subjects and fifty controls. To compensate for potential procedure refusal, inadequate tissue biopsy yield, or loss during clinical follow-up, two hundred and eighty asthmatic patients and sixty healthy control subjects were prospectively included in the final analysis cohort. Formal verbal informed consent was documented from all participating subjects prior to enrollment. The study protocol was approved by the institutional ethics review board.

Eligible asthmatic participants met international diagnostic criteria for bronchial asthma, demonstrating recurrent symptoms and spirometric evidence of reversible airway obstruction (increase in forced expiratory volume in one second of at least twelve percent and 200 mL following bronchodilator administration). Asthmatic patients were classified into mild, moderate, and severe clinical categories in accordance with international consensus guidelines. Healthy control subjects had no history of chronic respiratory disease, normal baseline spirometry, and negative skin prick testing. Exclusion criteria for all participants included current or prior cigarette smoking history exceeding five pack-years, active

respiratory tract infection within the preceding six weeks, prior diagnosis of chronic obstructive pulmonary disease, bronchiectasis, interstitial lung disease, active malignancy, severe systemic autoimmune disorders, or systemic immunosuppressive therapy within thirty days of baseline evaluation. All enrolled subjects underwent baseline clinical history taking, physical examination, pre- and post-bronchodilator spirometry, and peripheral blood sampling for total serum immunoglobulin E quantification and absolute eosinophil counts. Total serum immunoglobulin E levels were measured using a standardized automated chemiluminescent immunoassay protocol. Flexible fiberoptic bronchoscopy was performed under local anesthesia and conscious sedation in accordance with standard clinical safety guidelines. Endobronchial mucosal biopsy specimens (3 to 5 biopsies per subject) were obtained from the carinae of the segmental and subsegmental bronchi of the right lower lobe.

Biopsy tissue specimens were immediately fixed in ten percent neutral buffered formalin, processed, paraffin-embedded, and cut into 4-µm serial sections. Sections were stained with hematoxylin-eosin, Periodic acid-Schiff for mucin identification, and Masson's trichrome for collagen visualization. Digital microscopic image analysis was conducted by two independent pathologists blinded to clinical data. Measured histopathological parameters included: reticular basement membrane thickness measured at ten uniform points per section using calibrated software; proportion of epithelial shedding quantified as the percentage of denuded basement membrane; submucosal inflammatory cell density (eosinophils and lymphocytes per high-power field); and goblet cell hyperplasia score calculated as the percentage of Periodic acid-Schiff positive mucin-containing cells within the surface epithelium.

Statistical analysis was conducted using SPSS version 27.0. Continuous variables conforming to normal distribution were expressed as mean ± standard deviation, whereas categorical variables were reported as absolute frequencies and percentages. Differences across control and asthma severity groups were evaluated using one-way analysis of variance (ANOVA) with Tukey post-hoc tests or Kruskal-Wallis tests where appropriate. Categorical variables were compared using Pearson's chi-square test. Pearson or Spearman correlation coefficients were calculated to assess relationships between serum immunoglobulin E levels, lung function metrics, and histopathological scores. Multivariable linear regression analysis was performed to identify independent biological and cellular predictors of reticular basement membrane thickness, reporting adjusted regression coefficients (β) and corresponding p-values. Statistical significance was defined as a two-sided p-value of less than 0.05.

RESULTS

Table 1. Baseline Clinical, Spirometric, and Immunological Parameters in Healthy Controls and Asthmatic Severity Subgroups (n = 340)

Parameter	Healthy Controls (n = 60)	Mild Asthma (n = 95)	Moderate Asthma (n = 110)	Severe Asthma (n = 75)	p-value
Age (years)	42.1 ± 10.4	41.5 ± 11.2	43.8 ± 11.8	45.2 ± 12.1	0.284
Body Mass Index (kg/m^2)	24.2 ± 3.1	24.8 ± 3.5	25.4 ± 3.9	26.1 ± 4.2	0.042
Post-bronchodilator FEV1 (% predicted)	98.4 ± 6.2	86.2 ± 7.1	71.5 ± 8.4	52.8 ± 9.6	<0.001
FEV1/FVC Ratio (%)	82.5 ± 4.1	76.8 ± 5.2	68.4 ± 6.1	58.2 ± 7.5	<0.001
Total Serum IgE (IU/mL)	48.2 ± 19.6	185.4 ± 72.1	386.2 ± 124.5	682.1 ± 210.8	<0.001
Absolute Blood Eosinophil Count (cells/µL)	142 ± 52	285 ± 94	462 ± 145	698 ± 215	<0.001

Table 1 details clinical and physiological measures, demonstrating progressive declines in spirometric parameters alongside marked elevations in serum immunoglobulin E and blood eosinophil counts with increasing asthma severity ($p < 0.001$).

Table 2. Quantitative Endobronchial Histopathological Metrics Across Study Subgroups

Histopathological Metric	Healthy Controls (n = 60)	Mild Asthma (n = 95)	Moderate Asthma (n = 110)	Severe Asthma (n = 75)	p-value
Reticular Basement Membrane Thickness (μm)	3.2 ± 0.8	5.4 ± 1.2	7.8 ± 1.8	11.2 ± 2.9	<0.001
Epithelial Shedding Area (%)	4.1 ± 1.5	14.2 ± 4.8	28.5 ± 7.6	48.6 ± 11.2	<0.001
Submucosal Eosinophils (cells/HPF)	1.8 ± 0.9	12.4 ± 4.2	26.8 ± 7.9	48.2 ± 12.4	<0.001
Submucosal Lymphocytes (cells/HPF)	8.2 ± 2.5	18.5 ± 5.1	31.4 ± 8.2	45.1 ± 10.8	<0.001
Goblet Cell Hyperplasia (%)	8.5 ± 2.8	22.4 ± 6.1	38.2 ± 9.4	58.4 ± 12.6	<0.001

Table 2 summarizes quantitative mucosal biopsy findings, revealing that basement membrane thickness, epithelial desquamation, inflammatory cell density, and goblet cell hyperplasia increase significantly across disease severity stages ($p < 0.001$).

Table 3. Multivariable Linear Regression Model for Predictors of Reticular Basement Membrane Thickness in Asthmatic Patients (n = 280)

Independent Variable	Standardized Beta (β)	95% Confidence Interval	p-value
Log Total Serum IgE Level	0.44	0.32 – 0.56	<0.001
Submucosal Eosinophil Density	0.38	0.26 – 0.50	<0.001
Duration of Asthma Symptoms (years)	0.22	0.11 – 0.33	0.002
Goblet Cell Hyperplasia Percentage	0.18	0.06 – 0.30	0.012
Baseline Post-bronchodilator FEV1 (% predicted)	-0.26	-0.38 – -0.14	<0.001

Table 3 outlines the multivariable regression analysis, establishing that elevated serum immunoglobulin E, submucosal eosinophilia, longer disease duration, and goblet cell hyperplasia independently predict reticular basement membrane thickening, while lung function demonstrates a negative association.

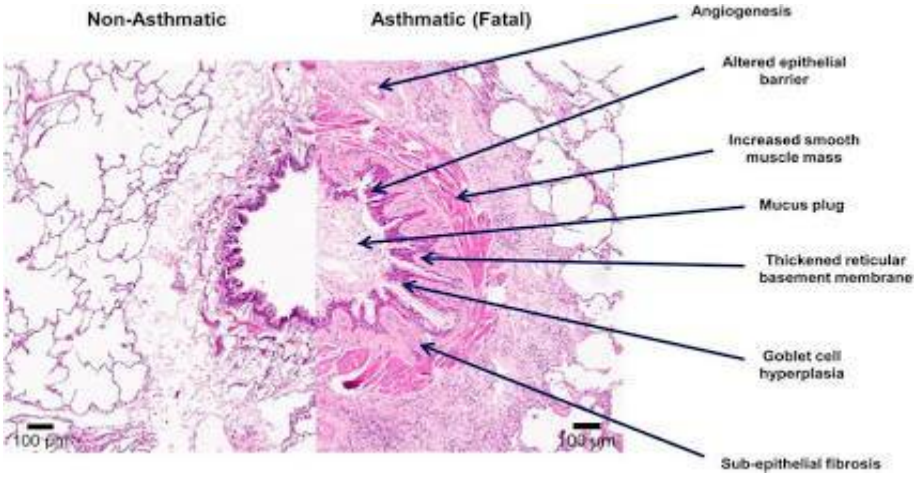


Figure 1: Comparative Overview of Asthmatic Airway Remodeling

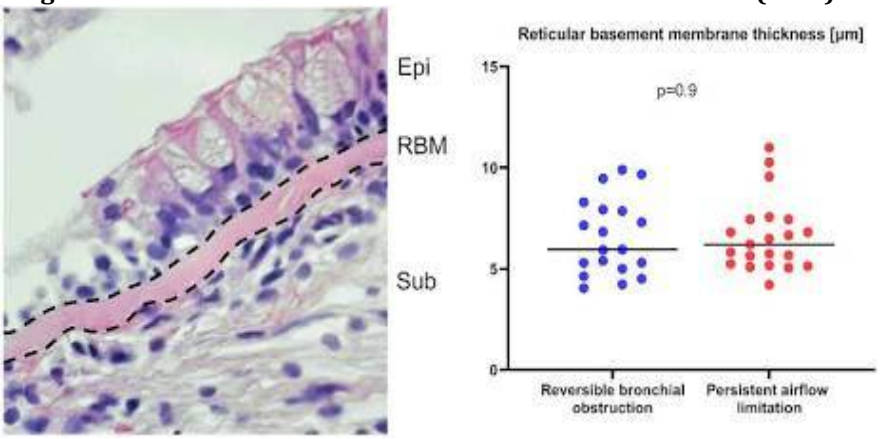
Thickened Reticular Basement Membrane (RBM): Demonstrates excessive subepithelial collagen layer widening (corresponds to $11.2 \pm 2.9\ \mu\text{m}$ in Severe Asthma vs. $3.2 \pm 0.8\ \mu\text{m}$ in Healthy Controls, $p < 0.001$).

Goblet Cell Hyperplasia: Prominent expansion of mucin-secreting goblet cells along the luminal surface (corresponds to 58.4% in severe asthmatics vs. 8.5% in controls).

Mucus Plug & Altered Epithelial Barrier: Luminal occlusion by dense mucin secretion alongside loss of pseudostratified ciliated architecture.

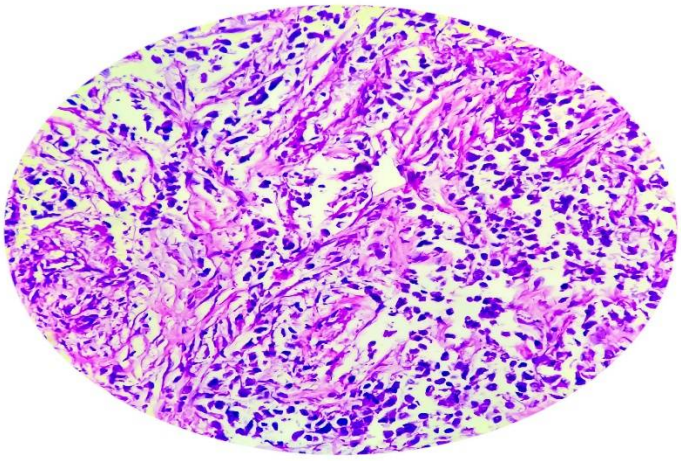
Sub-epithelial Fibrosis & Smooth Muscle Mass: Increased extracellular matrix deposition driven by persistent IgE and type-2 cytokine signaling.

Figure 2. High-Power Measurement of Reticular Basement Membrane (RBM) Thickening



Epi (Epithelium): Luminal columnar cell layer displaying patchy desquamation and junctional disruption.
RBM (Reticular Basement Membrane): Delineated subepithelial band exhibiting hyalinization and progressive collagen thickening. As established in the regression analysis (**Table 3**), log total serum IgE level was the single strongest independent predictor of this widening ($\beta = 0.44$, $p < 0.001$).
Sub (Submucosa): Underlying stroma harboring dense inflammatory cell populations.

Figure 3. Submucosal Inflammatory Infiltration & Eosinophilic Density



Submucosal Eosinophils: Bilobed granulocytes abundantly recruited to the subepithelial stroma
Perivascular Lymphocytes: Accumulation of T2-polarized mononuclear cells driving IL-4, IL-5, and IL-13 secretion.
Matrix Remodeling: Matrix metalloproteinase deposition and stromal edema resulting from degranulation products.

DISCUSSION

The principal findings of this prospective observational investigation demonstrate that bronchial epithelial structural remodeling and total serum immunoglobulin E elevation are closely intertwined in the pathogenesis and progression of bronchial asthma. In this study of two hundred and eighty asthmatic subjects and sixty healthy controls, quantitative endobronchial histopathology revealed progressive structural tissue alterations—including reticular basement membrane thickening, epithelial shedding, submucosal inflammatory cell accumulation, and goblet cell metaplasia—that correlated directly with clinical disease severity and systemic immunoglobulin E levels. These findings confirm that structural airway changes reflect ongoing allergic airway inflammation [11, 12].

Reticular basement membrane thickening represents a hallmark feature of chronic airway remodeling in bronchial asthma. In our cohort, severe asthmatic patients exhibited a mean basement membrane thickness of 11.2 µm, more than three-fold greater than healthy controls. This structural alteraction results from excessive deposition of extracellular matrix proteins, including collagen types I, III, and V, beneath the true basal lamina. Subepithelial collagen deposition is driven by persistently activated bronchial myofibroblasts, which are stimulated by transforming growth factor-beta released by damaged epithelial

cells and infiltrating mucosal eosinophils. These histopathological observations reinforce clinical literature demonstrating that subepithelial fibrosis develops early in the disease course and contributes to irreversible airflow limitation [13-14].

Total serum immunoglobulin E levels demonstrated a strong positive correlation with both basement membrane thickness and mucosal goblet cell hyperplasia. In multivariable linear regression modeling, log serum immunoglobulin E emerged as the single strongest independent biological predictor of basement membrane thickness, followed by submucosal eosinophil density and symptom duration. Immunoglobulin E cross-linking on mucosal mast cells promotes the sustained release of pro-fibrotic cytokines, including interleukin-4 and interleukin-13. Interleukin-13 acts directly on bronchial epithelial cells to induce MUC5AC gene expression, driving mucous cell metaplasia, while simultaneously stimulating subepithelial fibroblast proliferation and collagen synthesis. These molecular pathways explain the strong link between systemic immunoglobulin E titers and tissue-level remodeling [15-17].

Epithelial shedding was particularly prominent in patients with moderate to severe asthma, with severe subjects exhibiting shedding across nearly half of the analyzed mucosal surface area. Loss of structural epithelial integrity compromises the physical barrier against inhaled environmental antigens, exposing sensory nerve endings and subepithelial dendritic cells to ambient irritants. This epithelial disruption enhances mucosal permeability, amplifies localized airway hyperresponsiveness, and perpetuates chronic inflammation. Furthermore, extensive epithelial desquamation triggers continuous repair responses that favor fibrotic matrix deposition over normal tissue regeneration, creating a self-sustaining cycle of tissue damage and remodeling [18, 19].

The therapeutic implications of these findings are substantial. Demonstrating that serum immunoglobulin E correlates with tissue remodeling supports the early utilization of phenotype-targeted biological therapies in allergic asthma. Monoclonal anti-immunoglobulin E antibodies, such as omalizumab, neutralize circulating immunoglobulin E and downregulate high-affinity receptors on effector cells, blunting downstream cytokine cascades. Suppressing immunoglobulin E-mediated mucosal signaling can reduce submucosal inflammatory cell recruitment, attenuate goblet cell metaplasia, and potentially halt or partially reverse reticular basement membrane deposition.

Strengths of this study include its prospective design, direct quantification of mucosal histopathology via endobronchial biopsy, standardized automated biomarker profiling, and inclusion of a well-characterized control cohort. Limitations include the single-center setting and the cross-sectional nature of the biopsy protocol, which precluded serial tissue sampling post-biologic therapy. Long-term multi-center prospective studies with repeat endobronchial biopsies are warranted to determine whether therapeutic targeting of immunoglobulin E reverses established structural remodeling over extended follow-up periods.

CONCLUSION

Bronchial epithelial shedding, reticular basement membrane thickening, and goblet cell hyperplasia correlate directly with clinical disease severity and total serum immunoglobulin E levels in bronchial asthma. Elevated serum immunoglobulin E and submucosal eosinophilia serve as major independent drivers of subepithelial airway remodeling. Integrating serum immunoglobulin E profiling with structural tissue markers enhances pathogenic understanding, supporting early targeted anti-immunoglobulin E interventions to mitigate progressive airway damage.

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CITATION OF THIS ARTICLE

Kanwal S, Sumya G, Ahmad Farzad Q, Saba S, Rabia L, Kaneez F. Evaluation of Bronchial Epithelial Histological Changes and Serum IgE Levels in the Pathogenesis of Bronchial Asthma. Bull. Env. Pharmacol. Life Sci., Vol 15 [9] August 2026. 131-137