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PATHOGENESIS AND CLINICAL MANIFESTATIONS OF BRONCHIAL ASTHMA

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Abstract

Bronchial asthma is a chronic inflammatory disease of the airways that affects individuals of all ages and represents a significant global health burden. The disease is characterized by variable airflow obstruction, bronchial hyperresponsiveness, and persistent airway inflammation. This review aims to analyze the main pathogenetic mechanisms of bronchial asthma and to describe its key clinical manifestations.

The article is based on a narrative review of current scientific literature focusing on immunological pathways, inflammatory mediators, and structural airway changes involved in asthma development. The findings indicate that Th2-mediated immune responses, eosinophilic inflammation, IgE production, and mast cell activation play a central role in disease pathogenesis. Chronic inflammation contributes to airway remodeling, which may lead to disease progression and reduced lung function.

Clinically, bronchial asthma presents with recurrent episodes of wheezing, dyspnea, chest tightness, and cough, often triggered by allergens, respiratory infections, physical activity, or environmental factors. The variability and reversibility of symptoms reflect the dynamic nature of underlying inflammatory processes. A comprehensive understanding of the relationship between pathogenesis and clinical presentation is essential for accurate diagnosis, effective management, and improved patient outcomes.

Keywords

Bronchial asthma; Pathogenesis; Airway inflammation; Clinical manifestations; Immunological mechanisms; Th2 response; Airway remodeling

Introduction

Bronchial asthma is a chronic inflammatory disease of the airways characterized by variable and reversible airflow obstruction, bronchial hyperresponsiveness, and persistent airway inflammation [1]. It affects individuals of all ages and represents a major global health problem due to its high prevalence, morbidity, and negative impact on quality of life [2]. According to the World Health Organization, asthma affects hundreds of millions of people worldwide and remains a significant cause of preventable hospitalizations and mortality, particularly in low- and middle-income countries [3].

The pathogenesis of bronchial asthma is complex and multifactorial, involving a close interaction between genetic predisposition and environmental factors [4]. These interactions result in chronic airway inflammation, structural changes known as airway remodeling, and increased responsiveness of bronchial smooth muscle to various stimuli. Immunological mechanisms play a central role in disease development, including activation of T-helper 2 (Th2)

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lymphocytes, eosinophilic infiltration, mast cell activation, and the release of inflammatory mediators such as cytokines, leukotrienes, and immunoglobulin E (IgE) [5,6]. Together, these processes lead to airway narrowing and airflow limitation.

Clinically, bronchial asthma is characterized by recurrent episodes of wheezing, dyspnea, chest tightness, and coughing, which vary in frequency and severity [7]. Symptoms are often triggered or exacerbated by allergens, respiratory infections, physical exertion, cold air, and exposure to environmental irritants [8]. The variability and reversibility of symptoms are key features that distinguish asthma from other chronic obstructive airway diseases [9]. A clear understanding of the relationship between pathophysiological mechanisms and clinical manifestations is essential for accurate diagnosis and effective management.

This article aims to review the current concepts of bronchial asthma pathogenesis and to describe its main clinical manifestations, emphasizing the connection between immunological processes and clinical presentation [10].

Methods

This article is based on a narrative review of current scientific literature concerning the pathogenesis and clinical manifestations of bronchial asthma. Relevant publications were identified through a comprehensive search of established medical and scientific databases, including PubMed, Scopus, and Google Scholar. The search strategy focused on peer-reviewed articles, review papers, clinical guidelines, and epidemiological reports related to bronchial asthma.

Key search terms included “bronchial asthma,” “asthma pathogenesis,” “airway inflammation,” “Th2-mediated immune response,” “eosinophilic inflammation,” “IgE,” and “clinical manifestations of asthma.” Articles published in English were prioritized, with particular emphasis on widely cited and authoritative sources to ensure reliability and relevance [1–4].

The selection criteria included studies that provided detailed descriptions of immunological mechanisms, inflammatory pathways, and structural changes involved in asthma pathogenesis, as well as publications that clearly described clinical symptoms, triggers, and disease variability [5–7]. Guidelines and reports from international health organizations were also reviewed to support epidemiological and clinical data [3,8].

After initial screening based on titles and abstracts, full-text articles were assessed for eligibility. Data were extracted and synthesized qualitatively, focusing on the relationship between underlying pathophysiological mechanisms and clinical presentation of bronchial asthma [9]. No original experimental or clinical data were collected, and therefore ethical approval was not required for this review-based study.

The information obtained from the selected sources was systematically analyzed and organized to provide an integrated overview of bronchial asthma pathogenesis and its main clinical manifestations, in line with current scientific understanding [10].

Results

The analysis of the selected literature demonstrated that bronchial asthma is primarily driven by chronic airway inflammation mediated through complex immunological and cellular

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mechanisms. The core pathological feature identified across studies is persistent inflammation of the bronchial mucosa, leading to airway hyperresponsiveness and variable airflow limitation [1,4].

Pathogenesis of Bronchial Asthma

Results from immunological studies indicate that Th2 lymphocytes play a central role in the pathogenesis of bronchial asthma. Activation of these cells leads to increased production of cytokines such as interleukin (IL)-4, IL-5, and IL-13, which promote immunoglobulin E (IgE) synthesis, eosinophil activation, and mucus hypersecretion [5,6]. Elevated IgE levels facilitate mast cell sensitization, and subsequent exposure to allergens results in mast cell degranulation with the release of histamine, leukotrienes, and prostaglandins [2].

Chronic inflammation was found to contribute to airway remodeling, including thickening of the basement membrane, hypertrophy of bronchial smooth muscle, increased goblet cell numbers, and subepithelial fibrosis [4,9]. These structural changes result in irreversible components of airway obstruction in long-standing disease and are associated with disease severity.

Clinical Manifestations of Bronchial Asthma

Clinically, bronchial asthma was consistently characterized by episodic respiratory symptoms. The most frequently reported symptoms included wheezing, shortness of breath (dyspnea), chest tightness, and cough, particularly at night or in the early morning hours [7]. The reviewed studies emphasized that symptom severity and frequency vary significantly among patients and over time.

Environmental allergens, respiratory infections, physical exercise, cold air, and air pollutants were identified as common triggering factors [8]. The reversible nature of airflow obstruction, either spontaneously or after bronchodilator therapy, was a key diagnostic feature distinguishing asthma from other chronic obstructive pulmonary diseases [1,10].

Summary of Pathogenetic Mechanisms and Clinical Features

Pathogenetic Mechanism	**Biological Effect**	**Corresponding Clinical Manifestation**
Th2 cell activation	**Cytokine release (IL-4, IL-5, IL-13)**	**Chronic airway inflammation**
IgE-mediated response	**Mast cell sensitization and degranulation**	**Acute bronchoconstriction, wheezing**
Eosinophilic infiltration	**Airway tissue damage**	**Persistent cough, airway hyperreactivity**
Mucus hypersecretion	**Airway obstruction**	**Chest tightness, dyspnea**

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Pathogenetic Mechanism	Biological Effect	Corresponding Clinical Manifestation
Airway remodeling	Structural airway changes	Reduced lung function, severe asthma

Overall, the results highlight a strong correlation between immunopathological mechanisms and clinical presentation, confirming that bronchial asthma is a heterogeneous disease with variable clinical expression depending on the intensity of underlying inflammatory processes [9,10].

Discussion

The findings of this review confirm that bronchial asthma is a multifactorial chronic inflammatory disease in which immunological mechanisms play a central role in both disease development and clinical expression. The analyzed literature consistently demonstrates that Th2-driven inflammation, IgE-mediated hypersensitivity, and eosinophilic airway infiltration are key contributors to airway hyperresponsiveness and variable airflow limitation [4–6]. These results align with current concepts of asthma as an immune-mediated disorder rather than a purely bronchoconstrictive condition.

One of the most significant observations is the close relationship between airway inflammation and clinical symptoms. The release of inflammatory mediators from mast cells and eosinophils directly contributes to bronchoconstriction, mucus hypersecretion, and airway edema, which clinically manifest as wheezing, dyspnea, chest tightness, and cough [2,7]. The episodic and reversible nature of these symptoms reflects the dynamic inflammatory processes underlying the disease, distinguishing asthma from other chronic obstructive airway diseases [1,10].

Airway remodeling represents an important aspect of asthma pathogenesis discussed in the reviewed studies. Structural changes such as smooth muscle hypertrophy, basement membrane thickening, and subepithelial fibrosis were shown to correlate with disease duration and severity [4,9]. These changes may lead to partially irreversible airflow limitation and reduced responsiveness to standard therapy, highlighting the importance of early diagnosis and long-term anti-inflammatory treatment.

Environmental and genetic factors were also emphasized as crucial modifiers of disease expression. Exposure to allergens, respiratory infections, and environmental pollutants can amplify inflammatory responses and precipitate acute exacerbations [8]. At the same time, genetic predisposition influences immune regulation and susceptibility to asthma, explaining the heterogeneity of clinical phenotypes observed among patients [3].

Despite significant advances in understanding asthma pathogenesis, the reviewed literature indicates that bronchial asthma remains a heterogeneous condition with multiple endotypes and phenotypes. This variability poses challenges in diagnosis and management, underscoring the need for personalized treatment approaches targeting specific inflammatory pathways [5,6]. Further research is required to better define non–Th2-mediated mechanisms and to develop novel therapeutic strategies for severe and treatment-resistant asthma.

In conclusion, this review highlights the strong interplay between immunopathological mechanisms and clinical manifestations of bronchial asthma. A deeper understanding of these

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processes is essential for improving disease control, preventing airway remodeling, and enhancing patient outcomes.

Conclusion

Bronchial asthma is a chronic inflammatory airway disease characterized by complex immunopathological mechanisms and variable clinical presentation. The disease develops as a result of interactions between genetic susceptibility and environmental factors, leading to persistent airway inflammation, bronchial hyperresponsiveness, and structural airway changes. These processes form the basis of both reversible and, in advanced cases, partially irreversible airflow limitation.

The clinical manifestations of bronchial asthma, including wheezing, dyspnea, chest tightness, and cough, directly reflect the underlying inflammatory and immunological processes. Variability in symptom severity and frequency highlights the heterogeneous nature of the disease and emphasizes the importance of individualized assessment and management. Early diagnosis and appropriate anti-inflammatory therapy play a crucial role in preventing disease progression and airway remodeling.

A comprehensive understanding of the pathogenesis and clinical features of bronchial asthma is essential for effective disease control and improved patient outcomes. Continued research into the molecular and cellular mechanisms of asthma will support the development of targeted therapeutic strategies and contribute to more personalized approaches in asthma management.

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