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Etiology and Pathogenesis of Bronchial Asthma

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Abstract: Asthma is a long-term lung condition where your airways get inflamed, tighten up, and sometimes block airflow. It's not one-size-fits-all—different people get it for different reasons, but the end result is usually the same: wheezing, coughing, and shortness of breath. The Global Initiative for Asthma (GINA) helps us make sense of it all. Asthma and COPD (the lung disease heavy smokers often get) are two of the biggest respiratory problems worldwide. In kids, asthma is the #1 reason for hospital stays because of breathing trouble. In adults, COPD is a top killer. Both are tied to things in our environment that stress out lung cells and invite immune cells to overreact.

Keywords: Asthma, Causes, Mechanisms, GINA, Oxidative Stress, Th2, Airway Remodeling, Inflammation, Phenotypes, Endotypes

Bronchial asthma is one of the most common chronic diseases in the world. The number of patients diagnosed with this condition already exceeds 300 million, and this figure is growing annually. Asthma can appear at any time and completely change a person's life. Every 10 seconds, someone worldwide experiences an asthma attack, which can be fatal.Classification of asthma

Seven parameters were selected to classify the endotypes in patients with BA after the cluster analysis: clinical characteristics, biomarkers, lung physiology, genetics, histopathology, epidemiology, and response to treatment.

Based on **etiology** and **clinical features**, asthma is usually divided into the following major types:

a) Extrinsic (Allergic) Asthma

- Triggered by exposure to allergens such as dust mites, pollen, animal dander, or molds.
- Commonly occurs in childhood or early adulthood.
- Associated with elevated **IgE** levels and positive **skin prick tests**.
- Often accompanied by other atopic diseases (eczema, allergic rhinitis).

b) Intrinsic (Non-allergic) Asthma

- Occurs in adults, usually after respiratory infections.
- No evident allergen sensitization and normal IgE levels.
- Triggers include cold air, exercise, stress, or irritant fumes.

c) Mixed Asthma

- Features of both allergic and non-allergic types.

d) Occupational Asthma

- Caused by workplace exposure to irritants such as chemicals, dust, or fumes.
- Symptoms improve when away from work.

3. Classification Based on Severity (According to GINA – Global Initiative for Asthma)

Severity	Symptoms	Night Symptoms	PEF (Peak Expiratory Flow)	Treatment Need
Intermittent	≤2 times/week	≤2 times/month	>80% predicted	Short-acting β_2-agonist as needed
Mild Persistent	>2 times/week	>2 times/month	>80% predicted	Low-dose inhaled corticosteroid
Moderate Persistent	Daily symptoms	>1 time/week	60–80% predicted	Low/medium-dose ICS + LABA
Severe Persistent	Continuous	Frequent	<60% predicted	High-dose ICS + LABA ± systemic steroids

4. Classification Based on Pathophysiology (Phenotypes and Endotypes)

a) Phenotypic Classification

- **Allergic asthma**
- **Non-allergic asthma**
- **Late-onset (adult) asthma**
- **Obesity-related asthma**
- **Exercise-induced asthma**

b) Endotypic Classification

Defined by underlying **immunological mechanisms**:

- **Type 2 (T2-high) asthma** – eosinophilic inflammation, IL-4, IL-5, IL-13 cytokines, good response to corticosteroids.
- **Non-Type 2 (T2-low) asthma** – neutrophilic or paucigranulocytic inflammation, poor steroid response.

5. Other Classifications

- **By Control:** well-controlled, partly controlled, uncontrolled.
- **By Trigger:** drug-induced (e.g., aspirin-sensitive asthma), infection-induced, exercise-induced.
- **By Age of Onset:** childhood-onset vs. adult-onset asthma.

Role of T-lymphocytes in the development of inflammation in the respiratory tract

In allergic BA, the predominant immune response involves the second type of T-helper cells (Th2). This immune profile is commonly observed in both children and adults with mild to moderate asthma [18]. It is characterized by elevated levels of Th2 effector cytokines, specifically interleukins (IL) -4, -5, and -13. These cytokines are produced by CD4+ Th2 cells and innate immune cells of the second group known as ILC 2. IL-5 primarily contributes to the recruitment of eosinophils, while IL-4 and IL-13 play roles in mucosal cell metaplasia, respiratory hyperreactivity, and airway remodeling [18]. The sensitivity to corticosteroid treatment varies significantly among patients with Th2-type inflammation, with some showing high sensitivity and others having moderate or low sensitivity, particularly in cases of more severe disease.

Factors contributing to OS development

Reactive Oxygen and Nitrogen Species (RONS) are vital in regulating lung function. When triggered by pro-oxidant and inflammatory environmental factors, they accumulate, leading to cell and tissue damage, and even cell death. Oxidative reactions primarily affect proteins and lipids, causing molecular structure changes and cell function impairments. In Bronchial Asthma (BA) and Chronic

Obstructive Pulmonary Disease (COPD), increased RONS levels such as superoxide, hydrogen peroxide, and others are observed in various samples, evidenced by higher oxidative stress markers like malondialdehyde and nitric oxide. In severe asthma and COPD cases, these oxidative stress levels are significantly higher, correlating with worsening patient conditions, symptom severity, and reduced corticosteroid efficacy.

Conclusion

Asthma incidence is highest in childhood and thereafter decreases and remains stable at ~1–3 new cases per 1000 per year throughout late adolescence and adulthood. In adult populations, the prevalence of active cases of childhood-onset asthma (COA) and adult-onset asthma (AOA) are approximately equal, or favor AOA. Reasons for this counterintuitive prevalence ratio include (1) the propensity for COA to remit more frequently than AOA and (2) the greater number of years of adulthood in which to accrue new cases. Of relevance to clinical management and population disease burden is the wide range of asthma severities, from mild intermittent to severe persistent; the most severe 20% of cases account for 80% of health care utilization and morbidity. Robust population-based data indicate that around half of adults with asthma remain sub-optimally controlled, even when treated with currently available anti-inflammatory medications, and ~15% of adults with active asthma are severely uncontrolled. These data indicate the need for novel therapies that are effective in the most severe and treatment-resistant cases of asthma that account for the majority of morbidity, mortality and health care utilization. The emerging evidence that a wide variety of microbes are present in the lower airway and may play a role in asthma pathogenesis suggests that manipulating the airway microbiome may be a novel approach towards this goal. Among the various infections associated with asthma, the obligate intracellular respiratory pathogen CP is of particular interest, as it is associated with both asthma severity and treatment resistance. The etiology of asthma remains unknown and is almost certainly multifactorial

Literatures:

1. Ray A., Cohn L. Th2 cells and GATA-3 in asthma: new insights into the regulation of airway inflammation. J. Clin. Invest. 1999, 104 (8): 985-993.
2. Rivera J., Fierro N.A., Olivera A., Suzuki R. New insights on mast cell activation via the high affinity receptor for IgE. Adv. Immunol. 2008, 98: 85-120.
3. Roche WR., Beasley R., Williams J.H., Holgate S.T. Subepithelial fibrosis in the bronchi of asthmatics. Lancet. 1989, 1: 520-524.
4. Savelkoul H.F, Seymour B.W, Sullivan L., Coffman R.L. IL-4 can correct defective IgE production in SJA/9 mice. J. Immunol. 1991, 146 (6): 1801-1805.
5. Shimoda K., van Deursen J., Sangster M.Y. et
6. Holt P.G., Macaubas C., Stumbles P. A., Sly P.D. The role of allergy in the development of asthma. Nature. 1999, 402 (6760): 12-17.
7. Holt R.J., Zhang Y., Binia A. et al. Allele-specific transcription of the asthma-associated PHD finger protein 11 gene (PHF11) modulated by octamer-binding transcription factor 1 (Oct-1). J. Allergy Clin. Immunol. 2011, 127 (4): 1054-1062.
8. Howard T.D., Koppelman G.H., Xu J. et al. Genegene interaction in asthma: Il4ra and il13 in a dutch population with asthma. Am.J.Hum.Genet. 2002, 70 (1): 230-236.
9. Iwamoto I., Nakajima H., Endo H., Yoshida S. Interferon gamma regulates antigen-induced eosinophil recruitment into the mouse airways by inhibiting the infiltration of CD4+ T cells. J. Exp. Med. 1993, 177 (2): 573-576.
10. Iwata A., Kawashima S., Kobayashi M. et al. Th2-type inflammation instructs inflammatory dendritic cells to induce airway hyperreactivity. Int. Immunol. 2013.

11. Fedoseev GB, Korovina OV, Tenigina NG. The comprehensive diagnosis of different clinical and pathogenetic types of bronchial asthma. *Therapy Archive*. 1977;6:51–55.