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Review

Evolving Perspectives in Asthma: From Inflammation to Precision Therapy

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Abstract

Asthma is a chronic inflammatory disorder of the airways characterized by variable and recurring symptoms, reversible airflow obstruction, and bronchial hyperresponsiveness. It affects individuals of all age groups and represents a significant global health burden. The pathophysiology of asthma involves complex interactions among airway inflammation, genetic predisposition, environmental triggers, and immune dysregulation. This review provides an overview of asthma including its introduction, epidemiology, etiology, pathophysiology, clinical features, classification, diagnosis, Treatment, and recent advances in therapy. Understanding these aspects is essential for effective disease control and improvement of patient quality life.

Keywords: Asthma; airway inflammation; bronchoconstriction; inhaled corticosteroids

INTRODUCTION:

Asthma is a disease affecting the airways that carry air to and from lungs.[1] People who suffer from this chronic condition (long-lasting or recurrent) are said to be asthmatic. The inside walls of an asthmatic's airways are swollen or inflamed. This swelling or inflammation makes the airways extremely sensitive to irritations and increases your susceptibility to an allergic reaction. As inflammation causes the airways to become narrower, less air can pass through them, both to and from the lungs. Symptoms of the narrowing include wheezing (a hissing sound while breathing), chest tightness, breathing problems, and coughing.[2] Asthmatics usually experience these symptoms most frequently during the night and the early morning[3]

EPIDEMIOLOGY:

Asthma affects more than 300 million people globally and its prevalence continues to rise, particularly in low- and middle-income countries.[4] The disease is more common in children, with a male predominance during childhood and female predominance in adulthood.[5] Urbanization, environmental pollution, lifestyle changes, and increased exposure to allergens are major contributors to the increasing incidence of asthma.[6]

ETIOLOGY AND RISK FACTORS

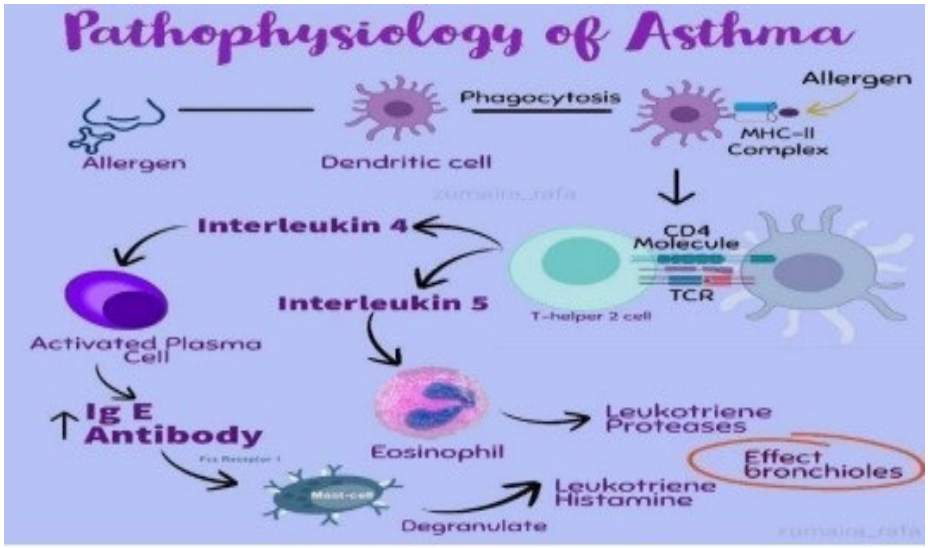
Asthma is a multifactorial disease influenced by both genetic susceptibility and environmental exposure.[7]

- Genetic Factors
  - Family history of asthma or atopic disorders
  - Gene polymorphisms affecting IgE production and immune regulation

- Environmental Factors
  - Allergens such as house dust mites, pollen, molds, and animal dander
  - Air pollutants and tobacco smoke
  - Occupational exposure to sensitizing chemicals
  - Viral respiratory infections, especially in early childhood.[8]

PATHOPHYSIOLOGY

Asthma is associated with T helper cell type-2 (Th2) immune responses, [9]which are typical of other atopic conditions. Various allergic (e.g., dust mites, cockroach residue, furred animals, moulds, pollens) and nonallergic (e.g., infections, tobacco smoke, cold air, exercise) triggers produce a cascade of immune-mediated events leading to chronic airway inflammation. Elevated levels of Th2 cells in the airways release specific cytokines, including interleukin (IL)-4, IL-5, IL-9 and IL-13,[9] that promote eosinophilic inflammation and immunoglobulinE (IgE) production by mast cells. IgE production, in turn, triggers the release of inflammatory mediators, such as histamine and cysteinyl leukotrienes, that cause bronchospasm (contraction of the smooth muscle in the airways), edema (swelling) and increased mucous secretion (mucous hypersecretion), which lead to the characteristic symptoms of asthma .[10]The mediators and cytokines released during the early phase of an immune response to an inciting allergen, trigger a further inflammatory response (late-phase asthmatic response) that leads to further airway inflammation and bronchial hyperreactivity. Evidence suggests that there may be a genetic predisposition for the development of asthma. A number of chromosomal regions associated with asthma susceptibility have been identified, such as those related to the production of IgE antibodies, expression of airway hyperresponsiveness, and the production of inflammatory mediators. However, further study is required to determine specific genes involved in asthma as well as the geneenvironment interactions that may lead to expression of the disease.



CLASSIFICATION OF ASTHMA

- Based on Severity
  - Intermittent asthma
  - Mild persistent asthma
  - Moderate persistent asthma
  - Severe persistent asthma[11]
- Based on Etiology
  - Allergic (extrinsic) asthma
  - Non-allergic (intrinsic) asthma
  - Occupational asthma
  - Exercise-induced asthma .[11]

DIAGNOSIS

Asthma is a disease of the lower respiratory tract that affects men and women of all ages. It is diagnosed clinically, but no single gold standard test is available; there is significant heterogeneity to asthma's pathophysiology and clinical overdiagnosis can occur, especially in those without spirometric confirmation. [12] Therefore, a thorough history and physical examination along with spirometry are important for the diagnosis of asthma.

Differential Diagnosis

Asthma can mimic other diseases and, therefore, it is important to consider various differential diagnoses in patients presenting with asthma like symptoms. Differential diagnoses of asthma include diseases of the upper and lower respiratory tracks, pathologies of the cardiovascular and gastrointestinal systems, and psychiatric conditions .[13] For example, congestive heart failure can cause wheezing and airflow obstruction from pulmonary oedema and pulmonary vascular congestion, mimicking asthma.[14] This condition has been termed 'cardiac asthma' and treatment of the underlying heart failure often leads to the improvement of the symptoms.[15] Vocal cord dysfunction (VCD) is another common differential diagnosis of asthma. These patients often present with recurrent asthma exacerbations that are refractory to corticosteroids or bronchodilator treatment. Chronic obstructive pulmonary disease (COPD) is a progressive, obstructive lung disease that presents similarly to asthma. Both diseases affect the small airways and have airflow obstruction seen on spirometry; however, COPD patients have limited airway hyper-responsiveness (<12% improvement in forced expiratory flow in 1 second [FEV1] after bronchodilator inhalation on pulmonary function test [PFT]) and often have a significant smoking history. Asthma and COPD can exist as a spectrum of obstructive diseases and can sometimes be difficult to distinguish from one another, especially in patients with chronic, poorly controlled asthma that leads to fixed airflow obstruction due to chronic inflammation and airway remodelling, as this can make the distinction between the two diseases more difficult.[16] Some of these patients can have chronic persistent airflow obstruction with aspects of asthma and meet the diagnosis for asthma–COPD overlap syndrome.

Understanding and recognising these two disease processes are important.

TREATMENT

The goal of asthma treatment is symptom control and prevention of future exacerbations. It involves an understanding of the heterogeneous pathophysiology and phenotypes of asthma and an individualised treatment plan. Patient education and a written asthma action plan can raise awareness of worsening symptoms, impending exacerbations, and the need for titration of therapy for better symptom control. [16,17]

Selfmanagement and a shared care approach have also been shown to improve asthma outcomes .In addition, education about proper inhaler techniques, medication compliance, and avoidance of allergens and irritants is crucial to all asthma patience. Currently, it is recommended that all patients with asthma have SABA inhalers for rescue therapy. In those with persistent asthma,[18] addition of low-dose ICS in titrating doses is recommended. For those with moderate-to-severe persistent asthma, long-acting beta-2 agonists (LABA) or leukotriene inhibitors are often added to the ICS regimen. Select use of biologic agents can be considered for those patients with more severe, difficult-to-control forms of asthma.

Beta-2 Agonists

Beta-2 agonists are bronchodilators that play an important role in asthma control and treatment of acute exacerbations .They bind to the beta-2 adrenergic receptors on the bronchial smooth muscle cells, causing smooth muscle relaxation and bronchodilation.SABA are often used to treat mild

intermittent asthma and acute exacerbations but should not be considered a controller medication; increased use of SABA has been associated with worse asthma control and ICS can sometimes be added to the treatment of those with mild intermittent asthma to limit SABA use.SABA are most effective in treating acute bronchoconstriction and have a rapid onset of action of 1–5 minutes, with peak effects at 2 hours and median duration of action of 3 hours.[19,20] Examples of SABA include albuterol, levalbuterol, terbutaline, metaproterenol,and pirbuterol.

LABA include salmeterol and formoterol and can have bronchodilatory effects lasting >12 hours.44 However, LABA should only be prescribed in conjunction with ICS in asthma patients. The researchers found that there were more respiratory and asthma-related deaths and life-threatening experiences in those treated with LABA than those receiving placebo.[22]

Corticosteroids

Corticosteroids are integral to the management of acute asthma exacerbations and chronic disease control because a significant portion of asthmatic patients have an inflammatory phenotype. ICS have also been shown to reduce the rates of exacerbations and improve lung function.[23] Examples of currently available ICS include beclomethasone, triamcinolone, flunisolide, ciclesonide, budesonide, fluticasone, and mometasone.

In patients with moderate-to-severe persistent asthma, the addition of LABA to ICS has been found to be beneficial. Studies by Kavuru et al.53 and Shapiro et al.54 showed that a combination of salmeterol and fluticasone resulted in improvements in PEFR, reduced symptom scores, nocturnal symptoms, and albuterol use compared to fluticasone alone. A study by O'Byrne et al.47 showed that ICS alone reduced the risk of severe exacerbations and poorly controlled symptom days, and that the addition of LABA to ICS further improved overall lung function.[24]

Leukotriene Receptor Antagonists and Synthesis Inhibitor

Leukotrienes are lipid mediators involved in bronchoconstriction and airway inflammation. Leukotrienemodifying drugs, including zafirlukast, montelukast, and zileuton, work by inhibiting leukotriene synthesis or as competitive antagonists of the leukotriene receptors.[25]

Antimuscarinics

The parasympathetic system, controlled by acetylcholine and the activation of muscarinic receptors, contributes to airway smooth muscle constriction and mucous secretion.[26] Currently available shortacting muscarinic antagonists (SAMA) include ipratropium and long-acting muscarinic antagonists (LAMA) include tiotropium, aclidinium, umeclidinium, and glycopyrronium. Both SAMA and LAMA can be used to treat severe, poorly controlled asthma exacerbations and as an add-on maintenance therapy to LABA/ICS therapy.[27]

Biological Therapy

For those with severe asthma, the use of biologic agents should be considered carefully. Targeted use of biologic therapy allows these patients to achieve control while limiting their oral corticosteroid exposure (Table 1).[26] Omalizumab is the first approved biologic for asthma and works by binding to IgE and downregulating activation of airway inflammation. In clinical trials, omalizumab has been shown to reduce overall asthma exacerbation rates by 25% and severe exacerbations by 50%, as well as improving asthma quality of life in those with uncontrolled moderate-to-severe asthma with perennial aeroallergen sensitivity. Newer biologic agents targeting IL-5 pathways are also available. IL-5 is a major cytokine responsible for the growth, differentiation, and survival of eosinophils, which play a large role in airway inflammation. Mepolizumab is a humanised monoclonal antibody against IL-5, hence it blocks the IL-5 pathway.[27]

Table 1: Recent Advances in Asthma Therapy

Biologic	Mechanism of action	Indication	Dose	Evidence
Omalizumab	Monoclonal antibody against IgE	Poor control on ICS or LABA, positive perenniall aeroallerge testing,total.serum IgElevel ≥30 IU/mL.	Subcutaneously onc every 2–4 weeks based on IgE level and weight.	Reduced all exacerbations by 25% and severe exacerbations by 50%.
Mepolizumab	Monoclonal antibody against IL-5.	Poor control on ICS c LABA,>2 exacerbationsper yea eosinophili >150 cells/µl	100mg subcutaneously once every 4 weeks.	>50% reduction in overall exacerbation rate and a >60% reduction in hospitalisation/ emergency department visits.
Reslizumab	Monoclonal antibody against IL-5.	Poor control on ICS or LABA, multiple exacerbations, peripheral eosinophilia >400 cells/µL	Intravenous infusion once every 4 weeks, based on weight.	>50% reduction in overall exacerbation rate and a >60% reduction in hospitalisation/ emergency department visits.

Recent advances include the development of targeted biologic agents, phenotype-based treatment strategies, smart inhaler technology, and improved drug delivery systems.[27] These innovations have significantly improved outcomes in patients with severe and refractory asthma.

Comorbid conditions

Treatment of comorbid conditions and avoidance of environmental and allergic triggers are important in asthma management. For example, obesity, gastro-oesophageal reflux disease, anxiety and depression, rhinitis and sinusitis, and seasonal and perennial allergies have all been associated with worsening asthma symptoms. [12]

Additional treatments targeting these comorbidities can significantly improve asthma control, especially in those with severe asthma.

CONCLUSION

Asthma is a heterogeneous disease affecting millions of people worldwide. It is characterised by airway hyperresponsiveness and airway inflammation with variable airflow obstruction. Understanding the various phenotypes and pathophysiologies and providing individualised treatment that is suited to the patient' scomorbidities and lifestyle is important in the management of asthma.

REFERENCES

1. Global Initiative for Asthma (GINA). Global Strategy for Asthma Management and Prevention, 2023.
2. Barnes PJ. Nat Rev Dis Primers. 2018.
3. FitzGerald JM et al. Can Respir J. 2006.
4. WHO. Global Surveillance of Chronic Respiratory Diseases, 2019.
5. Kovesi T et al. CMAJ. 2010. [12] Bousquet J et al. N Engl J Med. 1990.
6. World Health Organization. Asthma Fact Sheet, 2023.
7. National Heart, Lung, and Blood Institute. EPR-3 Guidelines, 2007.
8. Martinez FD. Pediatrics. 2002.
9. Robinson DS et al. N Engl J Med. 1992.

10. Peters-Golden M et al. J Allergy Clin Immunol. 2006.
11. Bateman ED et al. Eur Respir J. 2008.
13. McCracken JL et al. Diagnosis and management of asthma in adults: A review. JAMA. 2017;318(3):279- 90.
14. Jorge S et al. Cardiac asthma in elderly patients: Incidence, clinical presentation and outcome. BMC Cardiovasc Disord. 2007;7(1):16.
15. Kenn K, Balkissoon R. Vocal cord dysfunction: What do we know? Eur Respir J. 2011;37(1):194-200.
16. National Heart, Lung, and Blood Institute, National Asthma Education and Prevention Program. Expert Panel Report 3: Guidelines for the Diagnosis and Management of Asthma 2007. Available at: https://www.nhlbi.nih.gov/files/docs/guidelines/asthgdln.pdf. Last accessed: 12 September 2018.
17. Wilson SR et al. Shared treatment decision making improves adherence and outcomes in poorly controlled asthma. Am J Respir Crit Care Med. 2010;181(6):566-77.
18. Rosen JP et al. Duration of action of oral albuterol in an asthmatic population. Ann Allergy. 1986;56(1):28-33.
19. U.S. Food and Drug Administration. PROAIR HFA (albuterol sulfate) inhalation aerosol indications and usage.2008. Available at: https://www.accessdata.fda.gov/drugsatfdadocs/label/2008/021457s013lbl.p df. Published Last accessed: 11 October 2018.
20. Barnes PJ, Pedersen S. Efficacy and safety of inhaled corticosteroids in asthma. Am Rev
12. Aaron SD et al. Reevaluation of diagnosis in adults with physiciandiagnosed asthma. JAMA. 2017;317(3):269-79.
Respir Dis. 1993;148(4 Pt 2):S1-26.
21. Shapiro G et al. Combined salmeterol 50 microg and fluticasone propionate 250 microg in the diskus device for the treatment of asthma. Am J Respir
22. Nelson HS et al. The salmeterol multicenter asthma research trial: A comparison of usual pharmacotherapy for asthma or usual pharmacotherapy plus salmeterol. Chest. 2006;129(1):15-26.
23. Nelson HS et al. The salmeterol multicenter asthma research trial: A comparison of usual pharmacotherapy for asthma or usual pharmacotherapy plus salmeterol. Chest. 2006;129(1):15-26.
24. Barnes PJ. Muscarinic receptor subtypes in airways. Life Sci. 1993;52(5-6):521-7.
25. Guyer AC, Long AA. Long-acting anticholinergics in the treatment of asthma. Curr Opin Allergy Clin Immunol. 2013;13(4):392-8.
26. Holgate ST. Clin Exp Allergy. 2008.
27. 14. Grembiale RD, Camporota L, Naty S, Tranfa CM, Djukanovic R, Marsico SA:Effects of specific immunotherapy in allergic rhinitic individuals with bronchial hyperresponsiveness. Am J Respir Crit Care Med 2000,162:2048-2052.