



International Journal of Science, Architecture, Technology and Environment

A REVIEW On Asthma: Current Concepts, Pathaophysiology and Therapeutic Approaches

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DOI: <https://doi.org/10.63680/ijstate092645.38>

Abstract

Asthma is a common chronic respiratory disorder characterized by variable respiratory symptoms and variable expiratory airflow limitation. It is associated with airway inflammation, bronchial hyperresponsiveness, mucus production and structural changes of the airway wall. Symptoms commonly include wheezing, shortness of breath, chest tightness and cough, and may vary in intensity over time. Asthma can affect people of all ages and may be influenced by genetic susceptibility, allergens, respiratory infections, tobacco smoke, air pollution, occupational exposures, exercise and other triggers. Diagnosis is based on a characteristic history together with objective evidence of variable expiratory airflow limitation, when available. Pharmacological management aims to control symptoms, reduce the risk of exacerbation and preserve lung function. Inhaled corticosteroids are the foundation of anti-inflammatory treatment, while bronchodilators such as short-acting and long-acting beta2-agonists and long-acting muscarinic antagonists have important roles in appropriately selected patients. Leukotriene receptor antagonists and biologic therapies may be useful in specific clinical situations. Patient education, inhaler technique, adherence, trigger avoidance and regular assessment are essential components of comprehensive asthma care. This review summarizes the definition, epidemiology, risk factors, pathophysiology, clinical manifestations, diagnosis, classification, pharmacological treatment, non-pharmacological management and prevention of asthma.

Keywords: Asthma; airway inflammation; bronchial hyperresponsiveness; inhaled corticosteroids; bronchodilators; beta2-agonists; Leukotriene antagonists; biologic; exacerbation

1. INTRODUCTION;

Asthma is a heterogeneous chronic airway disease in which respiratory symptoms and expiratory airflow limitation vary over time and in intensity. The clinical picture may differ between individuals, and several phenotype and endotypes have been described. Asthma commonly begins in childhood, although adult-onset

disease is also frequent. Poorly controlled asthma can interfere with sleep, physical activity, education and work and can lead to acute exacerbation requiring urgent medical attention.

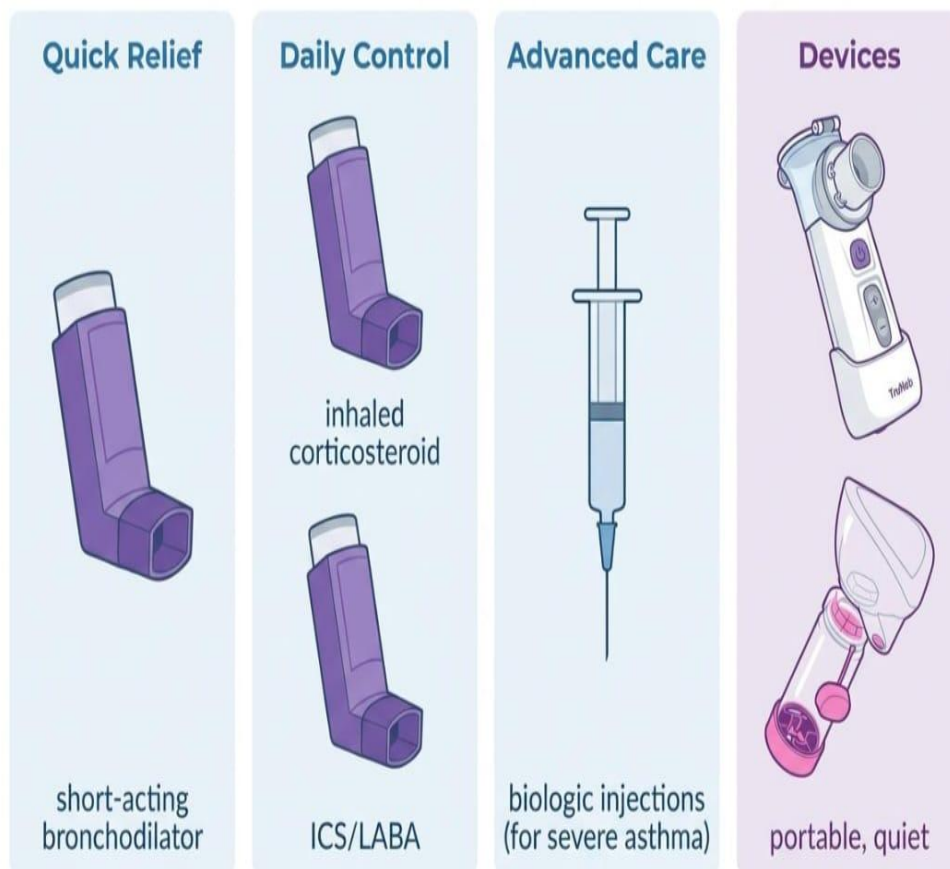
2. EPIDEMIOLOGY;

Asthma occurs worldwide and represents an important public-health burden. Its prevalence varies between countries and populations. Differences in genetics, environmental exposures, urbanization, air quality, occupational conditions, healthcare access and diagnostic practices contribute to this variation. Asthma can occur at any age and is frequently associated with other allergic or respiratory conditions.

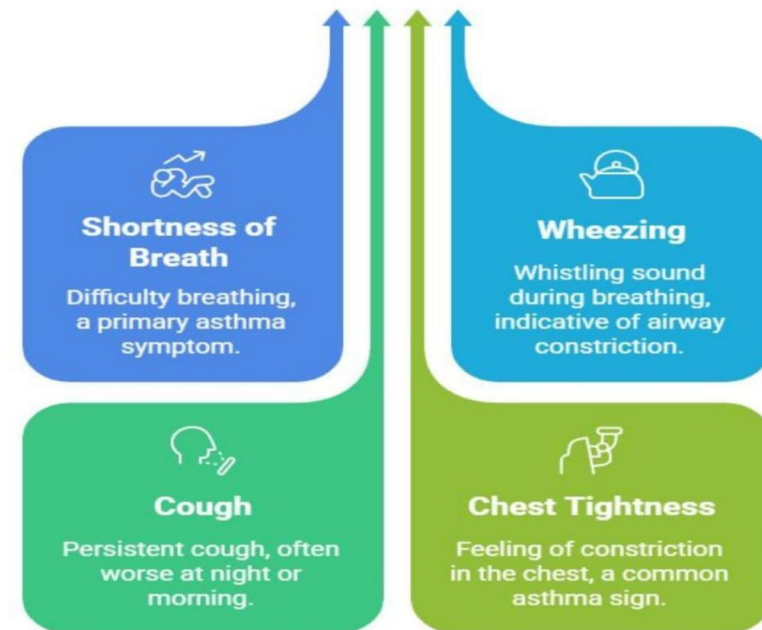
3. ETIOLOGY AND RISK FACTORS;

Asthma results from interactions between host susceptibility and environmental exposures. Important risk factors include family history of asthma or allergy, allergic sensitization, exposure to house-dust mites, pollen, animal allergens and molds, tobacco smoke, outdoor and indoor air pollution, occupational sensitizers, respiratory viral infections, obesity and certain medications in susceptible individuals. Exercise, cold air, strong odors and emotional.

ASTHMA TREATMENTS



Common Asthma Symptoms



stress may precipitate symptoms in some patients. Triggers vary considerably among individuals.

4. PATHOPHYSIOLOGY;

The characteristic physiological abnormality in asthma is variable airflow obstruction. Airway inflammation can involve eosinophils, mast cells, T lymphocytes, macrophages and airway epithelial cells. In some patients, type 2 inflammatory pathways involving interleukin such as IL-4, IL-5 and IL-13 contribute to IgE production, eosinophilic inflammation and airway hyperresponsiveness. Other patients have non-type-2 inflammatory patterns. Bronchoconstriction, airway wall edema and increased mucus secretion narrow the airway lumen. Repeated inflammation may contribute to airway remodeling, including epithelial changes, increased smooth-muscle mass and sub epithelial fibrosis.

5. CLINICAL MANIFESTATIONS;

Typical symptoms are wheezing, shortness of breath, chest tightness and cough. Symptoms often vary in frequency and severity and may be worse at night or early in the morning. Some patients experience symptoms mainly with exercise or after exposure to specific triggers. During an acute exacerbation, breathlessness, cough and wheezing may increase and expiratory airflow may become substantially reduced. Severe attacks can be life-threatening and require urgent assessment and treatment.

6. DIAGNOSIS;

Diagnosis requires recognition of a characteristic pattern of variable respiratory symptoms together with evidence of variable expiratory airflow limitation. Spirometry is commonly used to demonstrate airflow obstruction and its variability, including improvement after a bronchodilators when appropriate. Peak expiratory flow monitoring can also demonstrate variability in selected situations. Bronchial provocation testing may be considered when the diagnosis remains uncertain. Differential diagnoses depend on age and presentation and may include chronic obstructive pulmonary disease, vocal cord dysfunction, cardiac disease, foreign body aspiration and other respiratory disorders.

7. ASSESSMENT OF ASTHMA CONTROL AND SEVERITY;

Assessment should consider both current symptom control and future risk. Important risk factors for poor outcomes include previous severe exacerbation, poor adherence to controller therapy, incorrect inhaler technique, smoking or other exposures, low lung function and certain comorbidities. Asthma severity is generally assessed retrospectively according to the level of treatment required to maintain control rather than solely by symptoms at the time of diagnosis.

8. PHARMACOLOGICAL MANAGEMENT;

Modern asthma management emphasizes anti-inflammatory treatment and avoidance of reliance on bronchodilators-only therapy. Inhaled corticosteroids (ICS) reduce airway inflammation and the risk of exacerbation. Depending on age, symptom pattern and severity, ICS may be used alone or in combination with other agents. Long-acting beta2-agonists (LABAs) provide sustained bronchodilators and are used in combination with ICS rather than as mono therapy in asthma. Long-acting muscarinic antagonists (LAMAs) may be added for selected patients whose asthma remains uncontrolled. Leukotriene receptor antagonists may be useful in some patients, particularly when allergic rhinitis or aspirin-exacerbated respiratory disease is relevant. For severe asthma with specific inflammatory phenotype, biologic therapies targeting IgE or type-2 inflammatory pathways may be considered by specialist clinicians. Systemic corticosteroids have an

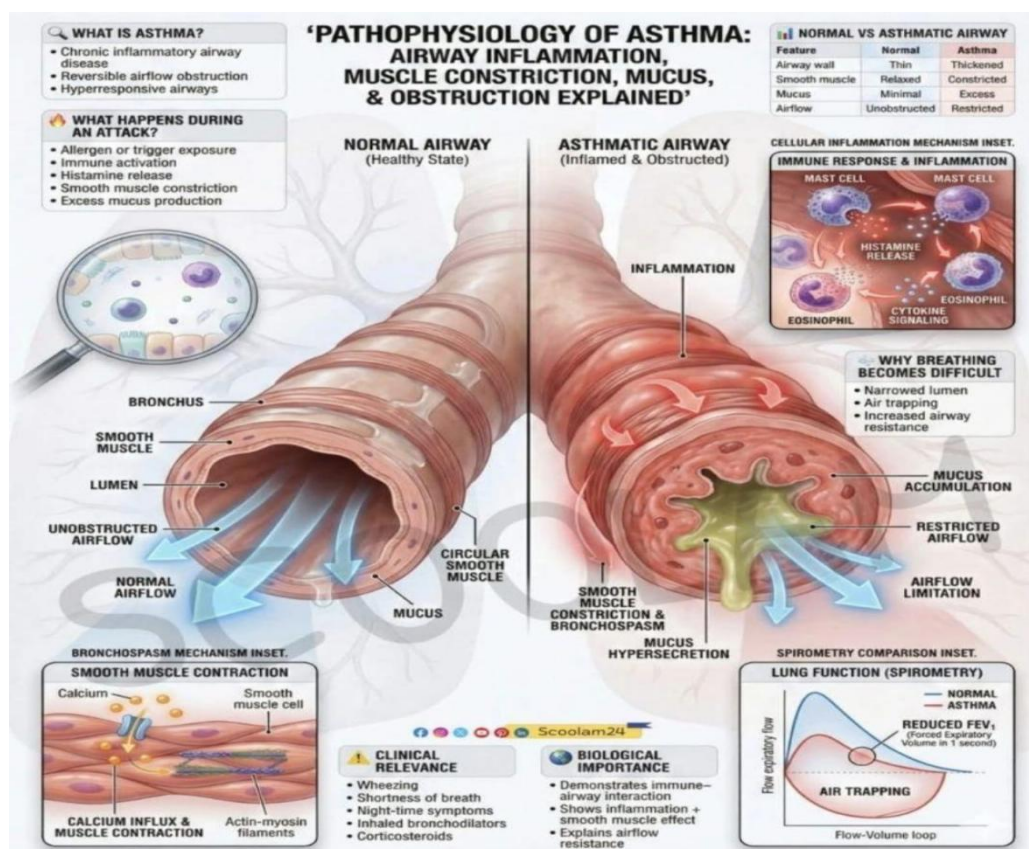
important role in treating significant acute exacerbation but repeated or long-term exposure is associated with substantial adverse effects.

9. COMMON ANTI-ASTHMATIC DRUG CLASSES;

Inhaled corticosteroids: Examples include budesonide, beclometasone and fluticasone. They suppress airway inflammation and are the principal controller medicines.

Short-acting beta2-agonists: Salbutamol is a common example and provides rapid bronchodilators for symptom relief; current treatment strategies generally emphasize use with appropriate anti-inflammatory therapy.

Long-acting beta2-agonists: Formoterol and salmeterol provide prolonged bronchodilators. LABAs should be used with ICS in asthma.



Leukotriene receptor antagonists: Montelukast is an example and blocks Leukotriene-mediated effects. Anticholinergic bronchodilators: Ipratropium is useful in selected acute settings, while tiotropium may be used as an add-on controller in appropriate patients.

Biologic medicines: Examples include omalizumab, mepolizumab, benralizumab, dupilumab and tezepelumab; selection depends on disease phenotype, biomarkers and clinical eligibility.

ASTHMA DRUG THERAPY

RELIEVERS

1. Short-acting β_2 -agonists

Asthavent[®] MDI /
DP-Haler[®] /
Revolizer[®] *
(Salbutamol)



Berotec[®] MDI
(Fenoterol)



Venteze[®] MDI
(Salbutamol)



Ventolin[®] MDI /
Accuhaler[®] *
(Salbutamol)



2. Anticholinergics

Atrovent[®] MDI
(Ipratropium Bromide)



Ipvent-40[®] MDI
(Ipratropium Bromide)



Spiriva Handihaler[®]
(Tiotropium)



CONTROLLERS

Long-acting β_2 -agonists

Foratec DP-Haler[®] /
Revolizer[®] *
(Formoterol)



Oxis Turbuhaler[®]
(Formoterol)



Serevent[®] MDI /
Accuhaler[®] *
(Salmeterol)



COMBINATIONS

DP-Haler[®] / Revolizer[®] *
(Budesonide + Formoterol)



Seretide[®] MDI /
Accuhaler[®] *
(Fluticasone + Salmeterol)



Symbicort Turbuhaler[®]
(Budesonide + Formoterol)



PREVENTERS

1. Inhaled Corticosteroids

Alvesco[®] MDI
(Ciclesonide)



Beclate HFA[®] MDI
(Beclomethasone)



Budeflam DP-Haler[®] /
Revolizer[®] *
(Budesonide)



Budeflam HFA Gentle-Haler[®]
(Budesonide)



Flixotide[®] MDI /
Accuhaler[®] *
(Fluticasone)



Inflammid[®] MDI /
Novolizer[®] *
(Budesonide)



Pulmicort Turbuhaler[®]
(Budesonide)



QVAR[®] MDI
(Beclomethasone)



2. Leukotriene receptor antagonist

Singulair[®] tablets
(Montelukast)



10.MECHANISMS OF ACTION;

ICS enter airway cells and regulate gene transcription through glucocorticoid receptors, reducing the expression of multiple inflammatory mediators. Beta2-agonists activate beta2-adrenergic receptors on airway smooth muscle, increasing intracellular cyclic AMP and producing relaxation. Muscarinic antagonists block Anticholinergic-mediated Bronchoconstriction at muscarinic receptors. Leukotriene receptor

antagonists inhibit Leukotriene D4/E4 signaling through the cysteinyl Leukotriene receptor. Biologic agents act on selected immune pathways, such as IgE, IL-5, the IL-4 receptor pathway or thymic stromal lymphopoietin, thereby reducing specific components of airway inflammation.

11. INHALER DEVICES AND PATIENT EDUCATION;

The effectiveness of inhaled medicines depends strongly on correct device use. Patients should receive practical instruction and their technique should be checked periodically. Device choice should consider inspiratory ability, coordination, age, dexterity, preference and availability. Spacers can improve delivery from pressurized metered-dose inhalers in suitable patients. Education should also cover adherence, written action plans, recognition of worsening symptoms and appropriate response to exacerbation.

12. NON-PHARMACOLOGICAL MANAGEMENT;

Non-pharmacological care includes identification and reduction of relevant triggers, avoidance of tobacco smoke, management of occupational exposures, physical activity appropriate to disease control, weight management when indicated, vaccination according to national recommendations, and treatment of relevant comorbidities such as allergic rhinitis, gastroesophageal reflux or obstructive sleep apnoea. Environmental interventions should be individualized rather than broadly applied without evidence of relevance to the patient.

13. ACUTE ASTHMA EXACERBATION;

An acute exacerbation is a worsening of asthma symptoms and lung function compared with the patient's usual status. Assessment includes symptom severity, respiratory rate, oxygen saturation, ability to speak and objective measures of airflow when feasible. Treatment commonly includes repeated inhaled short-acting bronchodilators therapy, oxygen when indicated, and systemic corticosteroids for significant exacerbation. Ipratropium may be added in severe attacks. Patients with severe or life-threatening features require urgent emergency management.

14. ADVERSE EFFECTS AND SAFETY;

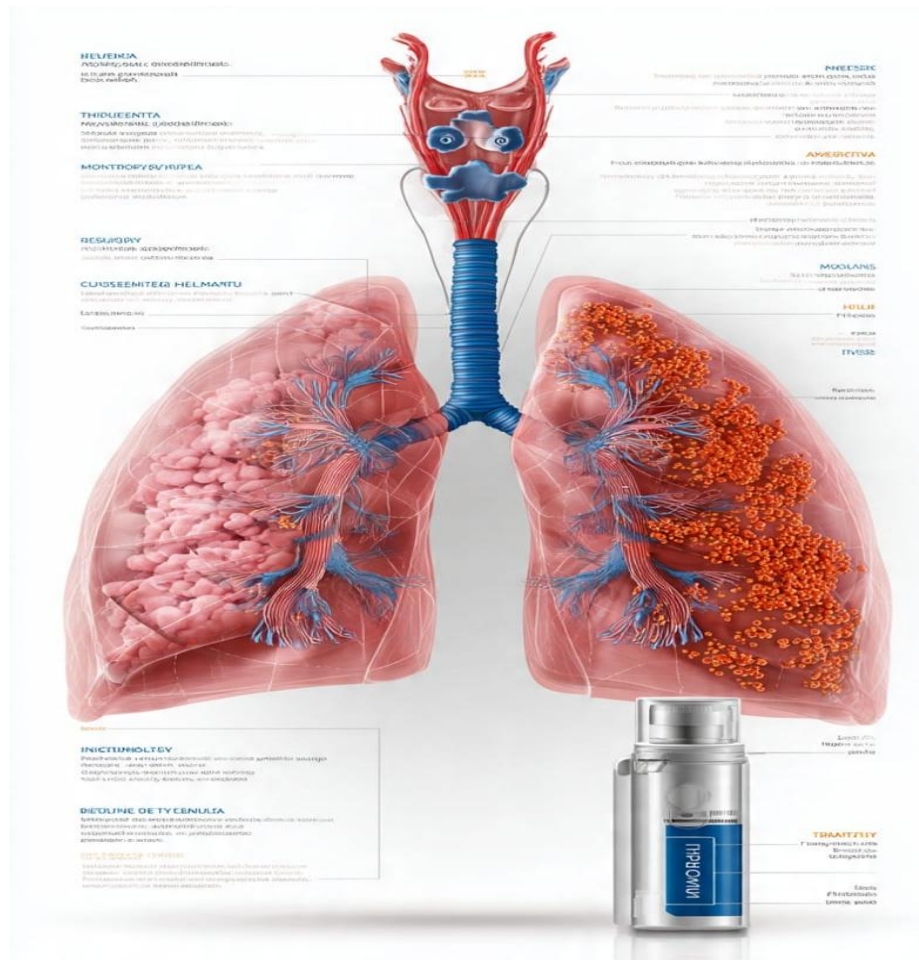
Inhaled corticosteroids may cause oral candidiasis and dysphonia; rinsing the mouth after use can reduce local effects. Beta2-agonists may cause tremor, palpitations and transient changes in serum potassium. Leukotriene receptor antagonists have generally good tolerability but can rarely be associated with neuropsychiatric adverse effects. Systemic corticosteroids can cause metabolic, cardiovascular, bone and infectious complications with repeated or prolonged use. Biologic therapies can cause injection-site reactions and, rarely, hypersensitivity reactions. Medication selection should balance expected benefits and risks.

15. ROLE OF PHARMACISTS;

Pharmacists contribute to asthma care through medication counseling, inhaler-technique assessment, adherence support, identification of drug-related problems, monitoring of adverse effects and reinforcement of asthma action plans. They can also help patients understand the difference between controller and reliever medicines and encourage appropriate follow-up when control is poor.

16. PREVENTION AND LONG-TERM FOLLOW-UP;

Prevention focuses on reducing avoidable exposures, maintaining appropriate controller therapy, improving adherence and inhaler technique, and recognizing early deterioration. Regular follow-up allows assessment of symptom control, exacerbation risk, lung function when indicated, treatment response and opportunities to step treatment up or down. Treatment should be individualized and periodically reviewed.



17. RECENT DEVELOPMENTS AND FUTURE PERSPECTIVES;

Asthma research increasingly focuses on precision medicine, biomarkers and biologically defined disease mechanisms. Improved phenotyping may help identify patients who are likely to respond to targeted biologic therapies. Digital inhalers, remote monitoring, artificial-intelligence-assisted analysis and newer anti-inflammatory approaches are being studied to improve adherence, monitoring and individualized care. Future management is likely to combine clinical assessment with biomarkers and patient-specific treatment pathways.

18. CONCLUSION;

Asthma is a chronic and heterogeneous respiratory disease characterized by variable symptoms, variable expiratory airflow limitation and airway inflammation. Effective management requires accurate diagnosis,

assessment of control and risk, appropriate pharmacotherapy, correct inhaler technique, adherence and patient education. Inhaled corticosteroids remain central to anti-inflammatory treatment, while bronchodilators, Leukotriene modifiers and biologic therapies have roles according to clinical need and disease phenotype. Early recognition of exacerbation and regular follow-up can reduce morbidity and improve quality of life. Continued research into disease mechanisms and precision therapies is expected to further improve asthma care.

19.TABLES;

Drug class	Examples	Main role
Inhaled corticosteroids	Budesonide, Beclometasone, Fluticasone	Control airway inflammation
SABA	Salbutamol	Rapid bronchodilators / symptom relief
LABA	Formoterol, Salmeterol	Long-acting bronchodilators with ICS
LAMA	Tiotropium	Add-on bronchodilators in selected patients
Leukotriene receptor antagonists	Montelukast	Anti-Leukotriene therapy
Biologics	Omalizumab, Mepolizumab, Dupilumab, Tezepelumab	Targeted therapy for selected severe asthma

Table 2. Selected clinical features and assessment;

Feature	Examples / significance
Symptoms	Wheeze, dyspnea, chest tightness, cough; variable over time
Objective assessment	Spirometry, bronchodilators response, peak-flow variability when appropriate
Control assessment	Daytime symptoms, night waking, reliever use, activity limitation
Risk assessment	Previous exacerbations, low lung function, adherence, exposures, comorbidities

Declaration of Conflicting Interests

The authors declare no potential conflicts of interest with respect to the research, authorship and publication of this article.

Funding

The author received no financial support for the research, authorship and publication of this article.

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