Current Pharmaceutical Research (CPR)
Vol 1, Issue 4, October-December 2025
Journal Homepage: https://cpr.org.in/

Review Article

Advances in Asthma Management: From Inflammatory Mechanisms to Clinical Therapies

[1]Ritesh Kapil, [2]Taranvirinderdeep Singh, [2]Achla Sharma*

[1]*Research Scholar, Himachal Pharmacy Colleges, Nalagarh, Himachal Pradesh, India,*
[2]*School of Pharmacy, Desh Bhagat University, Mandi Gobindgarh, Punjab, India*

Keywords

Asthma, Bronchial asthma, Airway inflammation, Immune dysregulation, T helper cells (Th2, Th17), Bronchoprovocation test.

Abstract

Bronchial asthma is chronic inflammatory disorder of the respiratory tract, characterized by increased airway hyperresponsiveness to provocative exposures and episodic airflow obstruction. it caused due to bronchial contraction. some internal and external factor affect asthma.it is non transmissible disease. Many risks factor like smoking, pollutant, depression, allergy affect the asthma disorder. Diagnosis is made with thorough history-taking and physical examination, and the condition is characterised by variable airflow obstruction and airway hyper-responsiveness. Understanding the severity of the disease is important, and treatment is aimed at symptom control and the prevention of future exacerbations. Pharmacologic treatment with beta-agonists for intermittent asthma and inhaled corticosteroids and a combination of inhaled corticosteroids and long-acting beta-2 agonists for persistent asthma are recommended. Additional and alternative treatments with leukotriene modifiers, anticholinergics, biologics, and bronchial thermoplasty are also available. However, understanding an individual's disease phenotype, endotype, and comorbidities is necessary for asthma treatment, with appropriate consultation with asthma specialists required for those with severe asthma.

***Corresponding Author:**
Achla Sharma (achlasharma1997@gmail.com)
ORCID: 0009-0006-5180-2689

Article Info
Received: 22 May 2025; Received in revised form: 16 January 2026; Accepted: 16 January 2026; Available online: 21 January 2026; Volume: 1; Issue: 4; Pages: 458-472.

ISSN: 3049-2955/The authors © 2025, under exclusive license to the Sprout Publication.
DOI: https://doi.org/10.63785/2025.1.4.458472

1. Introduction

Asthma is a chronic inflammatory disorder of the airways characterized by wheezing, shortness of breath, coughing, and chest tightness. Although the precise etiology of asthma remains incompletely understood, both genetic predisposition and environmental exposures are recognized as key contributors to disease development. Allergic and autoimmune inflammatory mechanisms play a central role in the initiation and progression of asthma [1].

Patients with allergic asthma frequently exhibit elevated levels of immunoglobulin E, which plays a pivotal role in allergic responses by binding to specific allergens and triggering the release of inflammatory mediators [2]. These mediators induce bronchoconstriction and promote airway inflammation. Eosinophils are another critical component of the immune response in asthma and are involved in both parasitic defense and allergic inflammation. Increased eosinophil counts are commonly detected in blood, sputum, and bronchial biopsy specimens from individuals with asthma. Activated eosinophils release cytotoxic proteins and proinflammatory mediators that contribute to airway inflammation, epithelial damage, and structural remodeling [3].

During airway inflammation, eosinophils and other inflammatory cells generate nitric oxide, a gaseous signaling molecule associated with inflammatory activity in the respiratory tract [4]. Measurement of fractional exhaled nitric oxide serves as a noninvasive biomarker for assessing airway inflammation, particularly eosinophilic inflammation. Fractional exhaled nitric oxide levels are therefore useful in monitoring disease activity and evaluating therapeutic responses in patients with asthma, as illustrated in Figure 1 [5].

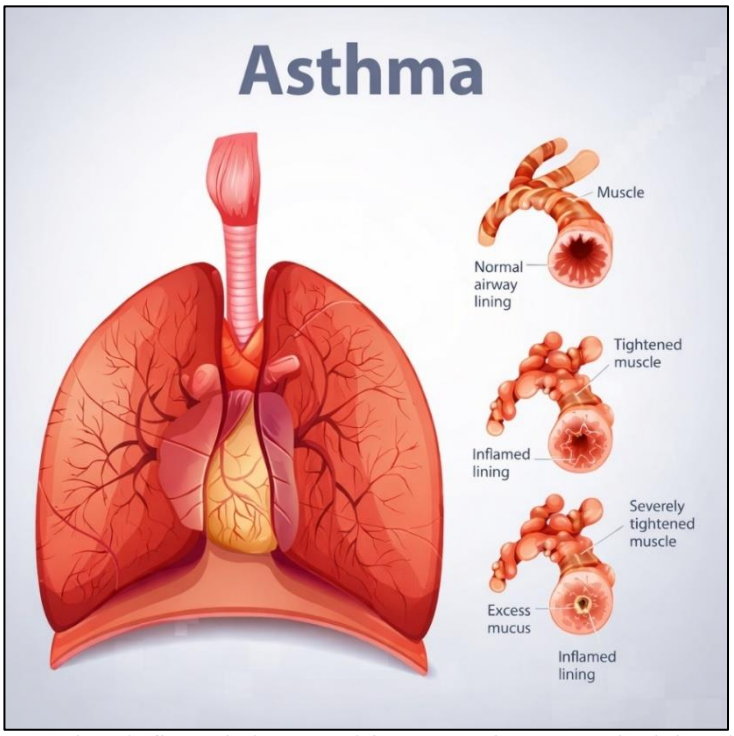

Figure 1: Asthma: Normal vs. inflamed airways with mucus plugs, muscle tightening, and obstruction.

Fractional exhaled nitric oxide is widely used as a noninvasive biomarker of airway inflammation in asthma; however, its clinical application has notable limitations, particularly with respect to false positive results [6]. Elevated fractional exhaled nitric oxide levels are not exclusive to asthma and may also be observed in other conditions such as eosinophilic bronchitis, allergic rhinitis, and atopic disorders. Environmental exposures including smoking, air pollution, and occupational irritants can further influence nitric oxide levels. Consequently, increased fractional exhaled nitric oxide does not invariably indicate asthma, which may lead to diagnostic ambiguity when used in isolation [7].

Asthma related immune dysregulation represents a disruption of normal immunological homeostasis, resulting in an imbalance between proinflammatory and regulatory mechanisms. This dysregulation frequently manifests as an exaggerated immune response characterized by excessive activation of airway immune cells, increased release of inflammatory cytokines, and overlap between allergic and autoimmune features [8]. In some cases, immune responses may also be insufficient or inappropriate, impairing the resolution of inflammation and accelerating disease chronicity. The pathophysiology of asthma is therefore driven by a complex interplay of immune mechanisms rather than a single inflammatory pathway [9].

Although asthma is primarily considered a chronic inflammatory airway disease, accumulating evidence suggests that immune system dysregulation contributes significantly to disease initiation and progression. In certain individuals, the immune system may erroneously target components of the respiratory tract [10]. Autoantibodies directed against airway self antigens have been identified in subsets of patients, supporting an autoimmune component in asthma pathogenesis. These immune abnormalities perpetuate chronic inflammation, increase disease severity, and reduce responsiveness to conventional therapies [11].

T helper lymphocytes play a central role in orchestrating immune responses in asthma. A dominant shift toward T helper type two immune activity is well documented and is associated with enhanced allergic inflammation and airway hyperresponsiveness [12]. In parallel, regulatory T cells, which are essential for maintaining immune tolerance and preventing excessive immune activation, are often reduced in number or function in patients with asthma. Impaired regulatory T cell activity allows exaggerated immune responses to otherwise harmless environmental antigens, thereby contributing to persistent airway inflammation [13].

Cytokine signaling networks are critical mediators of immune dysfunction in asthma. Interleukin four promotes immunoglobulin E synthesis and amplifies T helper type two immune responses. Elevated interleukin four levels contribute to increased allergen sensitivity, eosinophil recruitment, and airway inflammation [14]. Interleukin five is essential for eosinophil maturation, activation, and survival and facilitates eosinophilic infiltration of the airways, leading to epithelial injury and tissue remodeling. Interleukin thirteen shares overlapping biological functions with interleukin four and plays a key role in mucus hypersecretion, airway remodeling, and bronchial hyperresponsiveness [15].

In addition to T helper type two pathways, T helper type seventeen cells and their associated cytokine interleukin seventeen contribute to neutrophilic airway inflammation in certain asthma phenotypes.

Interleukin seventeen promotes neutrophil recruitment and activation, leading to persistent airway inflammation and tissue damage. Tumor necrosis factor alpha is another important proinflammatory cytokine involved in asthma pathogenesis, enhancing mucus production, airway narrowing, and inflammatory cell infiltration [16].

Bronchial asthma is clinically characterized by chronic airway inflammation, reversible airflow obstruction, and airway hyperresponsiveness to various stimuli. Symptoms commonly include wheezing, coughing, chest tightness, and shortness of breath. Unlike chronic obstructive pulmonary disease, in which airflow limitation is largely irreversible, airway obstruction in asthma is typically reversible either spontaneously or with appropriate therapy [17].
Asthma is a multifactorial disorder influenced by both genetic susceptibility and environmental exposures. Despite substantial advances in understanding its immunopathogenesis, the precise triggers initiating airway inflammation remain incompletely defined [18]. Since the late twentieth century, the global prevalence and mortality associated with asthma have increased significantly. Hundreds of millions of individuals worldwide are affected, and the disease continues to impose a substantial public health burden [19].

Environmental pollution is considered a major contributor to the rising incidence of asthma. Occupational exposure to flour dust, cotton fibers, smoke, and various chemical agents has been consistently associated with increased asthma risk. Given the significant global impact of this chronic respiratory disorder, comprehensive evaluation of its immunopathology and pharmacological management remains essential [20].

2. NHLBI 1997 Asthma

In 1997, the National Heart, Lung, and Blood Institute described asthma as a chronic inflammatory disorder of the airways involving multiple cellular and molecular components, including mast cells, eosinophils, T lymphocytes, neutrophils, macrophages, and airway epithelial cells. This persistent inflammation leads to recurrent episodes of coughing, chest tightness, wheezing, and dyspnoea in susceptible individuals, particularly during the night or in the early morning hours [21], [22]. These clinical manifestations are typically associated with widespread but variable airflow obstruction that is often reversible either spontaneously or with appropriate therapy. Furthermore, airway inflammation contributes to enhanced bronchial hyperresponsiveness to a variety of stimuli. In some individuals with asthma, however, airflow limitation may not be fully reversible, reflecting disease heterogeneity and progression [23].

3. Pathogenesis

Asthma has a complex pathophysiology characterized by airway inflammation and structural remodeling. The disease process is classically divided into two phases, namely an immediate phase and a late phase. The immediate phase represents an early allergic response that begins rapidly after exposure to a triggering stimulus, reaches a peak within fifteen to thirty minutes, and gradually resolves over the subsequent twenty four hours [24]. Mast cell activation and degranulation with the release of inflammatory mediators constitute the principal mechanism underlying this phase [25]. In susceptible individuals, inhaled allergens are processed by dendritic cells and presented to CD4 positive T lymphocytes, leading to the differentiation of naive T helper cells into Tho cells and their subsequent polarization into Th2 lymphocytes. Activated Th2 cells secrete key cytokines including interleukin four, interleukin five, and interleukin thirteen, which collectively promote allergic airway inflammation, eosinophil recruitment, immunoglobulin E production, and bronchial hyperresponsiveness, as illustrated in Figure 1 [26].

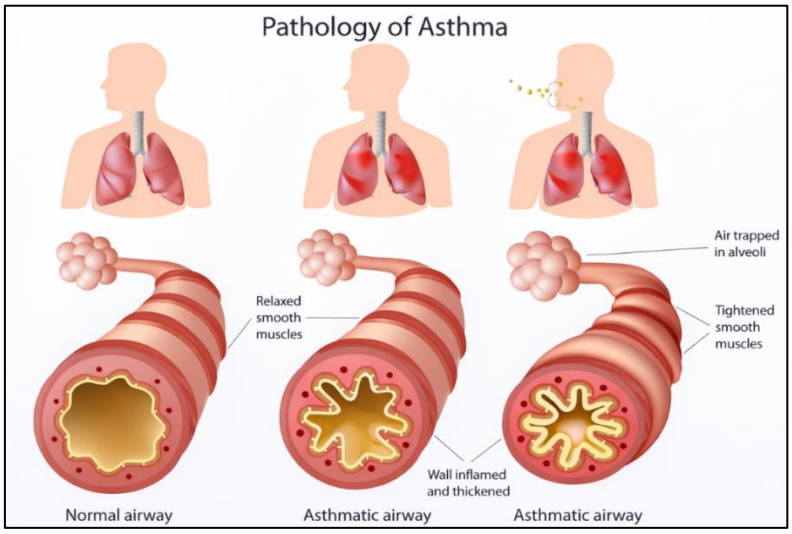

Figure 1: Schematic illustration showing normal airway anatomy compared with asthmatic airway, highlighting smooth muscle constriction, airway wall inflammation and thickening, mucus narrowing, and air trapping in the alveoli during asthma.

Th2 lymphocytes release a spectrum of cytokines that play a pivotal role in amplifying airway inflammation

in asthma. These cytokines promote the recruitment of eosinophils and other inflammatory granulocytes to the airway mucosa [27]. Interleukin five and granulocyte macrophage colony stimulating factor activate eosinophils, leading to the release of cytotoxic granule proteins and the generation of cysteinyl leukotrienes. These mediators damage the airway epithelium and contribute significantly to bronchial hyperresponsiveness [28].

In many atopic individuals with asthma, allergen specific immunoglobulin E is produced and binds to mast cells within the airway wall. Upon inhalation of allergens, cross linking of immunoglobulin E on mast cells induces degranulation and the release of mediators such as prostaglandin D2, histamine, and leukotrienes, which are potent bronchoconstrictors in hyperresponsive airways [29]. This process results in bronchospasm, local vasodilatation, increased capillary permeability, and enhanced chemotaxis, thereby facilitating further infiltration of inflammatory cells into the bronchial wall. Additional mediators released during this early phase include tumor necrosis factor alpha, macrophage inflammatory protein one alpha, and interleukins four, five, and thirteen [30].

The late phase reaction typically begins four to six hours after allergen exposure and may persist for up to twenty four hours, with sustained nonspecific airway hyperresponsiveness. This phase represents the continuation of the inflammatory cascade initiated earlier and is characterized by the accumulation of eosinophils and mononuclear cells driven by chemokines and chemotactic signals [31]. Activated eosinophils release eosinophil cationic protein, major basic protein, and eosinophil derived neurotoxin, along with cysteinyl leukotrienes and interleukins three, five, and eight, leading to persistent epithelial injury and inflammation. Additional mediators such as nitric oxide, adenosine, and neuropeptides further intensify the delayed inflammatory response. Although repair mechanisms may restore normal airway structure following acute inflammation, repeated injury and defective repair in chronic asthma result in airway remodeling, angiogenesis, increased smooth muscle mass, and long term airflow limitation [32].

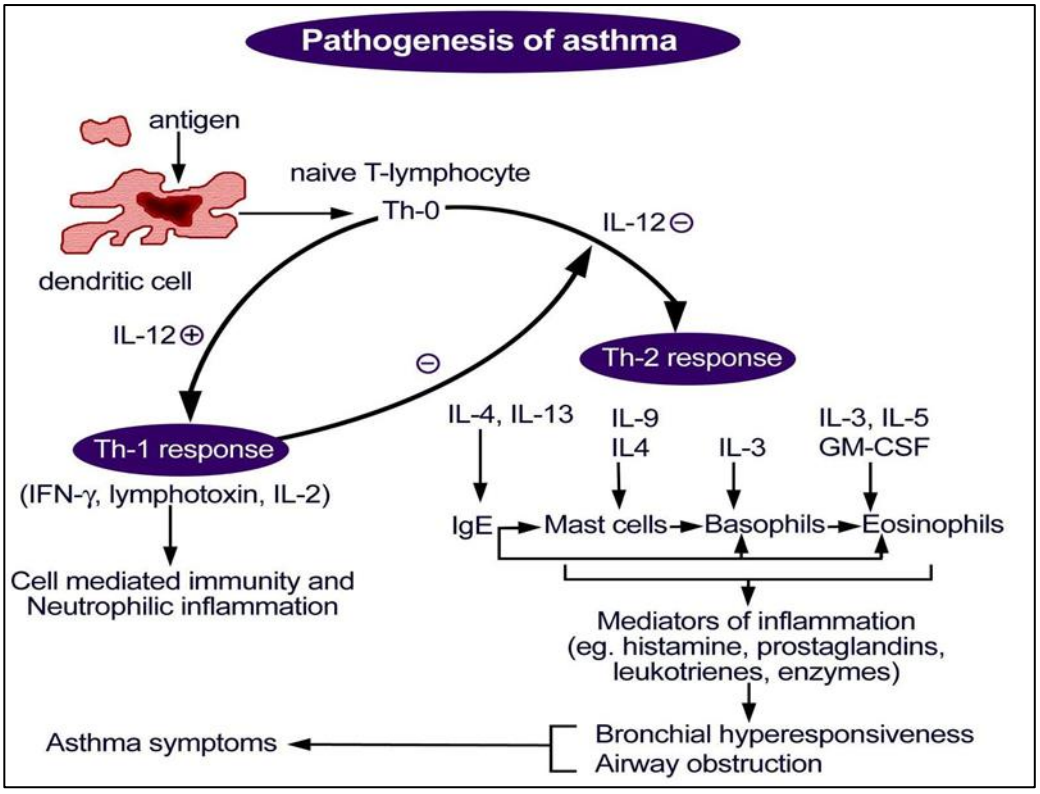

Figure 2: Immunopathogenesis of asthma depicting antigen presentation by dendritic cells, Th0 differentiation toward a Th2-dominant response, cytokine-mediated IgE production, mast cell and eosinophil activation, release of inflammatory mediators, and resultant airway hyperresponsiveness and obstruction.

This remodeling process is characterized by increased smooth muscle growth, subepithelial fibrosis, and progressive thickening of the airway wall, all of which contribute to chronic airflow limitation. A range of growth factors and matrix modifying enzymes play a critical role in driving these structural alterations. Airway smooth muscle cells actively participate in this process by producing proinflammatory mediators and autocrine growth factors, while growth factors released from infiltrating inflammatory cells promote smooth muscle hypertrophy and hyperplasia. Collectively, these changes lead to persistent airway narrowing and reduced reversibility of airflow obstruction, as illustrated in Figure 2 [33].

4. Diagnostic Evaluation of Bronchial Asthma

Bronchial asthma can often be diagnosed based on a

patient's clinical symptoms and detailed medical history. The presence of characteristic features such as wheezing, cough that is worse particularly at night, recurrent episodes of wheeze, repeated difficulty in breathing, and recurrent chest tightness increases the suspicion of bronchial asthma [34]. Symptoms that occur predominantly at night or worsen during sleep, leading to nocturnal awakening, further strengthen the likelihood of the diagnosis. In addition, asthma should be strongly considered when these symptoms are triggered or exacerbated by exposure to specific factors such as animals with fur, aerosolized chemicals, changes in ambient temperature, domestic dust mites, certain medications including aspirin and beta blockers, physical exertion, or exposure to pollen [35].

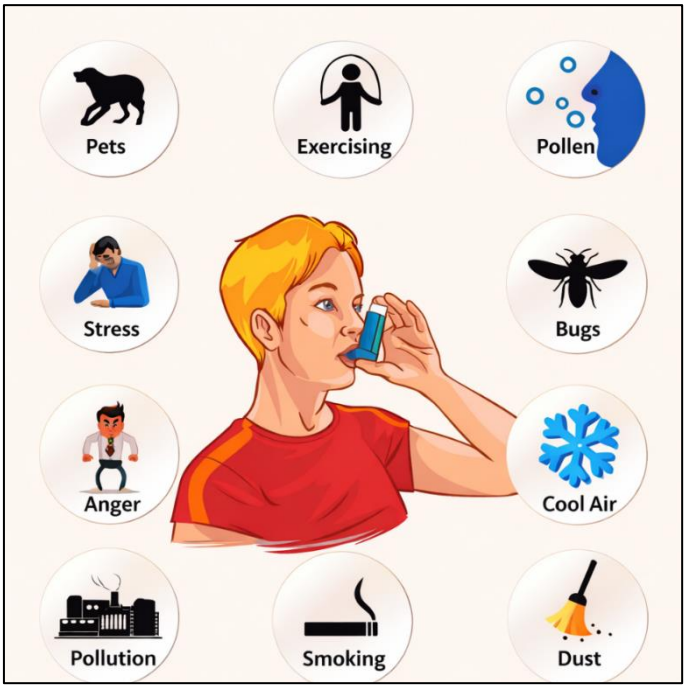

Figure 3: Common triggers of asthma, including allergens, environmental pollutants, climatic factors, physical exertion, emotional stress, and irritants that precipitate bronchoconstriction and acute asthma symptoms.

Respiratory infections, exposure to smoke, and strong emotional expressions are additional factors that can precipitate or exacerbate asthma symptoms. These triggers may intensify airway inflammation and bronchial hyperresponsiveness, leading to worsening respiratory symptoms in susceptible individuals, as illustrated in Figure 3 [36].

Common threat factors responsible for both development and deterioration of the lungs. Conditions and psoriasis include smoking, rotundity and low physical exertion, adulterants. Infection, exposure to allergens, malnutrition, metabolic pattern, connective towel. Diseases, depression, and use of certain medicines [37].

4.1. Smoking

Epidemiological studies indicate that individuals with psoriasis are more likely to be active or passive smokers compared with the general population. Smoking has been shown to correlate significantly with both the frequency and severity of psoriatic lesions [38]. Concurrently, smoking is recognized as the most important etiological factor for chronic obstructive pulmonary disease. Other smoking related respiratory disorders include asthma and idiopathic pulmonary fibrosis. Therefore, smoking may represent a shared risk factor contributing to the increased prevalence of pulmonary disease observed in patients with psoriasis [39].

4.2. Obesity

Obesity and physical inactivity have been identified as important risk factors for both the development and severity of psoriasis, with a higher prevalence of obesity consistently reported among psoriatic patients. Obesity is also a major risk factor for several respiratory disorders, including asthma, pulmonary hypertension, sleep apnea, and chronic obstructive pulmonary disease. Consequently, obesity may partially explain the increased burden of lung disease in individuals with psoriasiss [40].

4.3. Pollutants

Exposure to environmental pollutants has been implicated in the pathogenesis of both psoriasis and pulmonary diseases. Cadmium, a heavy metal used in batteries, dental materials, and various industrial applications, has been proposed as a potential air pollutant associated with psoriasis [41]. Elevated blood cadmium levels have been reported in patients with psoriasis. Similarly, increased cadmium exposure has been associated with chronic obstructive pulmonary disease, even among nonsmokers. These findings suggest that cadmium and other airborne pollutants may simultaneously influence the development of psoriasis and lung disease in susceptible individuals [42].

4.4. Infection

Infectious agents may play a significant role in the development and progression of both psoriasis and

respiratory disorders. Alterations in the lower respiratory tract microbiome, which may increase susceptibility to psoriasis, interact with the mucosal innate immune system and have been linked to the development of asthma. In addition, human immunodeficiency virus infection is a recognized risk factor for psoriasis as well as lung diseases such as asthma, chronic obstructive pulmonary disease, and pulmonary arterial hypertension. HIV infection may therefore contribute to the onset or exacerbation of both dermatological and pulmonary pathology [43].

4.5. Allergy

Patients with psoriasis have demonstrated mild hypersensitivity to various inhaled, food, and contact allergens, including birch, mugwort, thyme, rye pollen, house dust mites, and molds. The severity of allergic sensitization has been shown to correlate with the Psoriasis Area and Severity Index [44]. Allergic sensitization also plays a central role in the onset, severity, and management of asthma. Sensitivity to allergens such as house dust mites, animal dander, cockroaches, and molds has been identified as a significant risk factor for asthma, suggesting overlapping immunological pathways between the two conditions [45].

4.6. Depression

Psoriasis is a chronic inflammatory skin disorder that can substantially impair occupational performance, social interactions, family life, leisure activities, and sexual health. These limitations often result in psychological stress, which is known to exacerbate psoriasis. Conversely, depression is a common and persistent comorbidity in patients with interstitial lung diseases. The coexistence of psychological distress, psoriasis, and pulmonary disease highlights the importance of addressing mental health as an integral component of comprehensive disease management [46].

5. Diagnosis

Asthma is a chronic disorder of the lower respiratory tract that affects individuals of all ages and both sexes. The diagnosis of asthma is primarily clinical, as no single gold standard diagnostic test is available [47]. Considerable heterogeneity exists in both the pathophysiology and clinical presentation of asthma, and overdiagnosis may occur, particularly in the absence of objective confirmation of variable airflow limitation. Therefore, accurate diagnosis requires a comprehensive medical history, detailed physical examination, and objective assessment of lung function, most commonly through spirometry [48].

5.1. Clinical Presentation

The clinical presentation of asthma is variable and often nonspecific, which may complicate diagnosis. Patients typically present with wheezing, shortness of breath, and cough that occur intermittently and are more pronounced during the night or early morning hours. Symptoms are usually episodic and may be triggered by factors such as environmental irritants, specific allergens, or physical exertion. Wheezing and nocturnal dyspnoea show a strong association with asthma, with wheezing being the most sensitive and frequently reported symptom [49]. Respiratory symptoms that vary in intensity over time, worsen at night or in the early morning, and are associated with identifiable triggers increase the likelihood of an asthma diagnosis, as illustrated in Figure 4. In contrast, features such as persistent sputum production, chest pain, or isolated chronic cough without additional respiratory symptoms reduce the probability of asthma. A thorough clinical history is essential, and the diagnosis should be supported by evidence of variable airflow limitation on lung function testing [50].

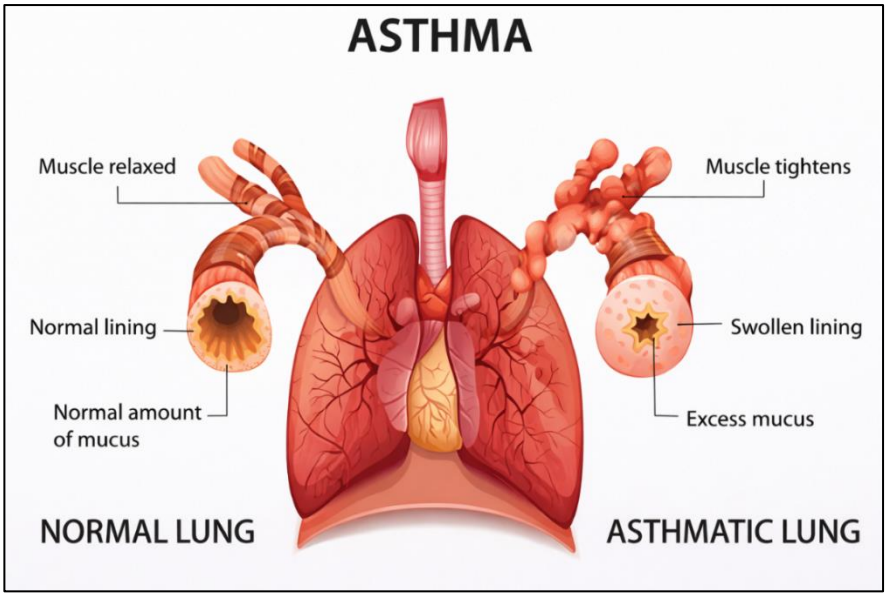

Figure 4: Comparative illustration of normal and asthmatic lungs showing airway smooth muscle constriction, mucosal swelling, excess mucus secretion, and resultant narrowing of the bronchial lumen in asthma.

5.2. Differential Diagnosis

Asthma shares clinical features with several other conditions, and a broad range of differential diagnoses should be considered in patients presenting with asthma like symptoms. These include disorders of the

upper and lower respiratory tract, cardiovascular diseases, gastrointestinal conditions, and certain psychological disorders. Congestive heart failure may mimic asthma by producing wheeze and airflow limitation due to pulmonary oedema and vascular congestion, a condition often referred to as cardiac asthma. Symptoms in such cases typically improve with appropriate treatment of the underlying cardiac disease [51].

Vocal cord dysfunction is another important differential diagnosis. Patients with this condition often experience recurrent episodes of breathlessness and wheeze that do not respond adequately to bronchodilators or corticosteroids. Vocal cord dysfunction is frequently associated with anxiety, depression, laryngopharyngeal reflux, and gastro oesophageal reflux, and results from paradoxical vocal cord movement causing episodic upper airway obstruction. Early recognition of this condition is essential to avoid unnecessary corticosteroid exposure and excessive healthcare utilization [52].

Chronic obstructive pulmonary disease is a progressive obstructive lung disorder that may present with symptoms similar to asthma. Although both conditions involve airflow limitation on spirometry, chronic obstructive pulmonary disease is typically characterized by limited reversibility of airflow obstruction and a significant history of tobacco smoking. Distinguishing between asthma and chronic obstructive pulmonary disease may be challenging, particularly in individuals with long standing poorly controlled asthma who develop fixed airflow obstruction due to persistent inflammation and airway remodeling. Some patients exhibit features of both conditions and may be classified as having asthma chronic obstructive pulmonary disease overlap, emphasizing the importance of careful clinical evaluation and accurate diagnosis [53].

Table 1 summarizes the principal differential diagnoses of asthma and highlights the clinical, functional, and therapeutic features that aid in distinguishing asthma from other conditions presenting with similar respiratory symptoms. The table compares characteristic symptom patterns, spirometric findings, and key diagnostic clues across fifteen common respiratory and non-respiratory disorders [54]. Asthma is characterized by episodic symptoms, nocturnal worsening, and reversible airflow obstruction, whereas conditions such as chronic obstructive pulmonary disease and interstitial lung disease typically exhibit persistent symptoms with fixed or restrictive ventilatory defects. Cardiovascular, upper airway, gastrointestinal, and psychological disorders may mimic asthma clinically but can be differentiated based on specific clinical features, response to treatment, and objective lung function testing. By systematically outlining these distinctions, Table 1 underscores the importance of comprehensive clinical evaluation and objective assessment in reducing misdiagnosis and guiding appropriate management strategies [55].

Table 1: Differential diagnosis of asthma and distinguishing clinical features.

S. No.	Condition	Symptoms	Symptom pattern	Spirometry findings	Distinguishing features	Clinical response	References
1.	Bronchial asthma	Wheeze, cough, dyspnoea, chest tightness	Episodic, nocturnal, trigger related	Variable airflow obstruction with reversibility	Atopy, airway hyperresponsiveness	Good response to bronchodilators and inhaled corticosteroids	[56], [57]
2.	Chronic obstructive pulmonary disease	Dyspnoea, chronic cough, sputum	Persistent and progressive	Fixed airflow limitation	Smoking history, older age	Partial response to bronchodilators	[58]
3.	Asthma COPD overlap	Wheeze, dyspnoea, cough	Persistent with variability	Partially reversible obstruction	Features of asthma and COPD	Requires combined therapy	[59], [60]
4.	Congestive heart failure	Wheeze, orthopnoea, dyspnoea	Worse on exertion or supine	Restrictive or mixed	Pulmonary oedema, cardiomegaly	Improves with cardiac treatment	[61], [62]
5.	Vocal cord dysfunction	Dyspnoea, throat tightness	Sudden onset and resolution	Normal between attacks	Inspiratory wheeze, poor asthma response	Improves with speech therapy	[63]
6.	Gastro oesophageal	Cough, wheeze,	Postprandial or	Usually normal	Heartburn, regurgitation	Improves with acid	[64]

	reflux disease	chest discomfort	nocturnal			suppression	
7.	Upper airway cough syndrome	Chronic cough	Persistent	Normal	Postnasal drip, rhinitis	Responds to nasal therapy	[65], [66]
8.	Bronchiectasis	Chronic cough, sputum	Persistent	Obstructive pattern	Recurrent infections	Responds to antibiotics and airway clearance	[67]
9.	Pulmonary embolism	Acute dyspnoea, chest pain	Sudden onset	Usually normal	Risk factors for thrombosis	Responds to anticoagulation	[68], [69]
10.	Interstitial lung disease	Progressive dyspnoea	Gradual onset	Restrictive pattern	Crackles, fibrosis on imaging	Limited bronchodilator response	[70]
11.	Foreign body aspiration	Cough, wheeze	Sudden onset	Localized obstruction	History of choking	Bronchoscopy required	[71]
12.	Obstructive sleep apnea	Nocturnal dyspnoea, fatigue	Sleep related	Normal	Snoring, obesity	Improves with CPAP	[72], [73]
13.	Anxiety disorder	Dyspnoea, chest tightness	Episodic	Normal	Associated panic symptoms	Responds to psychotherapy	[74], [75]
14.	Lung cancer	Cough, wheeze, weight loss	Progressive	Variable	Hemoptysis, mass on imaging	Depends on tumor treatment	[76]
15.	Tracheomalacia	Wheeze, cough	Persistent	Variable obstruction	Airway collapse on imaging	Limited response to bronchodilators	[77], [78]

5.3. Spirometry and Bronchoprovocation Testing

Current guidelines from the Global Initiative for Asthma and the National Asthma Education and Prevention Program recommend spirometry as a key diagnostic tool in patients with suspected asthma. Asthma is characterized by variable airflow obstruction and airway hyperresponsiveness. Objective evidence of airflow limitation includes a reduced ratio of forced expiratory volume in one second to forced vital capacity below seventy percent or below the lower limit of normal, along with demonstrable reversibility following inhalation of a short acting beta two agonist. Reversibility is defined as an improvement in forced expiratory volume in one second of at least twelve percent and two hundred milliliters [79].

Due to the variable nature of asthma, spirometry may be normal in some patients at the time of testing. In such cases, bronchoprovocation testing using methacholine or mannitol is useful to support or exclude the diagnosis. A reduction in forced expiratory volume in one second exceeding twenty percent at a provocation concentration below sixteen milligrams per milliliter or a provocation dose below four hundred micrograms is considered diagnostic [80]. Methacholine acts as a direct airway smooth muscle stimulant through acetylcholine receptors and is highly sensitive for asthma detection. Mannitol is an indirect bronchoconstrictor and is more specific but less sensitive. Both agents demonstrate comparable diagnostic performance, particularly in patients without active symptoms. Bronchoprovocation testing is therefore especially useful for ruling out asthma in patients not receiving inhaled corticosteroids [81].

5.4. Fractional Exhaled Nitric Oxide

Nitric oxide produced by the airway epithelium serves as an indirect marker of airway inflammation. Measurement of fractional exhaled nitric oxide is noninvasive and has been used to identify eosinophilic airway inflammation in patients with suspected or confirmed asthma [82]. Fractional exhaled nitric oxide is less informative in non eosinophilic asthma phenotypes and primarily reflects type two mediated inflammation. According to recommendations from the American Thoracic Society, fractional exhaled nitric oxide values below twenty five parts per billion in adults suggest a lower likelihood of eosinophilic inflammation and corticosteroid responsiveness [83]. The role of fractional exhaled nitric oxide in asthma monitoring remains debated. Some studies demonstrate that elevated levels correlate with disease severity and that monitoring with fractional exhaled nitric oxide and sputum eosinophil counts may reduce overall exposure to inhaled corticosteroids [84]. However, other investigations report no significant

reduction in exacerbation rates or corticosteroid use, and some patients monitored with fractional exhaled nitric oxide receive higher corticosteroid doses without symptomatic improvement. Despite these mixed findings, fractional exhaled nitric oxide measurement is still recommended as an adjunct tool for assessing disease activity and guiding treatment decisions in selected patients [85].

5.5. Exercise Challenge Testing

Exercise challenge testing is used to diagnose exercise induced bronchoconstriction in patients with exertional respiratory symptoms. During the test, patients undergo progressively increasing exercise intensity on a treadmill or stationary bicycle to achieve a high level of ventilation relative to baseline lung function [86]. A fall in forced expiratory volume in one second greater than ten percent following exercise confirms exercise induced bronchoconstriction. A reduction exceeding twenty five percent indicates moderate severity, while a decrease greater than fifty percent reflects severe disease. Exercise challenge testing should be considered in individuals with suggestive symptoms and a negative baseline diagnostic evaluation [87].

6. Treatment

The primary goals of asthma management are optimal symptom control and prevention of future exacerbations. Effective treatment requires an individualized approach based on an understanding of the heterogeneous pathophysiology and variable clinical presentation of asthma. Patient education and the use of a written asthma action plan are essential, as they improve recognition of worsening symptoms, early identification of exacerbations, and appropriate adjustment of therapy. Self management strategies and collaborative care models have been shown to improve asthma related outcomes. Education regarding correct inhaler technique, adherence to prescribed medications, and avoidance of known allergens and irritants is fundamental for all patients [88].

Pharmacologic therapy is recommended in a stepwise manner. Initial treatment selection is guided by asthma severity classification, while subsequent adjustments follow a step up or step down approach based on symptom control. Short acting beta two agonists are recommended as rescue therapy for all patients [89]. In individuals with persistent asthma, low dose inhaled corticosteroids are advised and titrated according to disease severity. For patients with moderate to severe persistent asthma, leukotriene modifiers or long acting beta two agonists are commonly added to inhaled corticosteroid therapy. In patients with severe or difficult to control asthma, selective use of biologic agents may be considered [90].

6.1. Beta Two Agonists

Beta two agonists are central to asthma management due to their bronchodilatory effects and role in relieving acute symptoms. These agents induce smooth muscle relaxation by binding to beta two adrenergic receptors on bronchial smooth muscle cells. Short acting beta two agonists are frequently used for mild intermittent asthma and acute exacerbations but should not be considered controller medications [91]. Excessive reliance on short acting beta two agonists is associated with poorer asthma control. These agents have a rapid onset of action, achieve peak effect within two hours, and have a short duration of action, making them well suited for acute bronchoconstriction. Common examples include albuterol, levalbuterol, terbutaline, metaproterenol, and pirbuterol [92].

Long acting beta two agonists such as formoterol and salmeterol provide prolonged bronchodilation lasting more than twelve hours. However, in patients with asthma, long acting beta two agonists should only be used in combination with inhaled corticosteroids [93]. Large clinical trials demonstrated an increased risk of severe asthma related events when long acting beta two agonists were used without inhaled corticosteroids. In contrast, multiple studies have shown that combination therapy with inhaled corticosteroids and long acting beta two agonists improves lung function and reduces exacerbation risk compared with inhaled corticosteroids alone. Consequently, combination inhalers containing both agents are considered safe and effective step up therapy in appropriately selected patients [94].

6.2. Corticosteroids

Corticosteroids play a critical role in both acute and chronic asthma management, particularly in patients with an inflammatory and eosinophilic phenotype [95]. Inhaled corticosteroids are the cornerstone of long term asthma control and act by suppressing airway inflammation through inhibition of eosinophil and mast cell activation. Their use is associated with reduced airway hyperresponsiveness, improved lung function, and decreased exacerbation frequency. Clinical studies have consistently demonstrated superior outcomes in patients treated with inhaled corticosteroids compared with beta agonist therapy alone [96].

In patients with moderate to severe persistent asthma, the addition of a long acting beta two agonist to inhaled corticosteroid therapy has been shown to further improve peak expiratory flow rate, reduce nocturnal symptoms, decrease rescue medication use, and enhance overall symptom control. Commonly used inhaled corticosteroids include beclomethasone, triamcinolone, flunisolide, ciclesonide, budesonide, fluticasone, and mometasone [97].

Systemic corticosteroids remain essential for the management of acute asthma exacerbations and uncontrolled disease. Short term use effectively reduces airway inflammation and bronchial constriction [98]. However, long term systemic corticosteroid therapy is discouraged due to significant adverse effects such as weight gain, osteoporosis, hypertension, adrenal suppression, gastritis, and psychiatric disturbances. Patients who require prolonged systemic corticosteroid therapy should be referred to an asthma specialist for further evaluation and consideration of alternative treatment strategies, including biologic therapies [99].

6.3. Leukotriene Receptor Antagonists and Synthesis Inhibitors

Leukotrienes are lipid mediators that contribute to airway inflammation, mucus secretion, and bronchial smooth muscle contraction. Leukotriene modifying agents such as zileuton, montelukast, and zafirlukast act by inhibiting leukotriene synthesis or blocking leukotriene receptors. Cysteinyl leukotrienes released from mast cells and eosinophils play a significant role in asthma pathophysiology [100].

These agents reduce airway inflammation, improve lung function, and alleviate asthma symptoms. Leukotriene receptor antagonists are generally used as add on therapy to inhaled corticosteroids or as an alternative option in patients with mild persistent asthma who are unable to tolerate inhaled corticosteroids [101].

6.4. Antimuscarinics

Antimuscarinic agents have a long history of use in the treatment of bronchoconstriction. Parasympathetic activation mediated by acetylcholine contributes to airway smooth muscle contraction and mucus secretion through muscarinic receptors. Antimuscarinic drugs inhibit this pathway, resulting in bronchodilation [102].

Short acting muscarinic antagonists such as ipratropium and long acting muscarinic antagonists including tiotropium, aclidinium, umeclidinium, and glycopyrronium are available for clinical use. These agents may be used as add on maintenance therapy in patients receiving inhaled corticosteroid and long acting beta two agonist combinations, as well as during severe exacerbations [103]. Clinical trials have demonstrated that tiotropium improves lung function, symptom control, and reduces exacerbation risk in patients with poorly controlled asthma. Long acting muscarinic antagonists therefore remain a valuable therapeutic option for individuals with persistent symptoms despite optimized standard therapy [104].

7. Bronchial Thermoplasty

Bronchial thermoplasty is a nonpharmacological treatment option for patients with severe asthma that remains uncontrolled despite optimized therapy with inhaled corticosteroids and bronchodilators. This procedure involves the bronchoscopic delivery of controlled thermal energy to the airway wall, resulting in targeted reduction of airway smooth muscle mass and subsequent attenuation of bronchoconstriction and airway hyperplasia [105]. Clinical trials have demonstrated that bronchial thermoplasty can improve lung function, reduce asthma symptoms, and increase symptom free days in patients with moderate to severe asthma. Although initial studies reported transient worsening of respiratory symptoms shortly after the procedure, longer term follow up showed improvements in forced expiratory volume, symptom scores, and reduction in exacerbation frequency [106]. Despite these benefits, bronchial thermoplasty is associated with potential risks, including severe asthma exacerbations and procedure related complications. Therefore, careful patient selection and management in specialized centers are essential when considering bronchial thermoplasty as a therapeutic option for refractory asthma [107].

8. Comorbid Conditions

Identification and management of comorbid conditions are integral components of comprehensive asthma care. Several conditions, including obesity, gastro oesophageal reflux disease, anxiety and depression, allergic rhinitis, sinusitis, and both seasonal and perennial allergies, have been shown to exacerbate asthma symptoms and impair disease control [108]. Addressing these comorbidities through targeted interventions can lead to significant improvements in symptom burden, quality of life, and overall asthma control. In patients with severe or difficult to treat asthma, effective management of associated comorbid conditions is particularly important and may reduce the need for escalation of asthma specific pharmacotherapy [109].

Conclusion

Asthma is a heterogeneous chronic inflammatory disorder of the airways characterized by variable airflow limitation, airway hyperresponsiveness, and structural remodeling. Its pathophysiology involves complex interactions among genetic susceptibility, environmental exposures, immune dysregulation, and inflammatory mediators, resulting in diverse clinical phenotypes and treatment responses. Accurate diagnosis relies on careful clinical assessment supported by objective measures such as spirometry, bronchoprovocation testing, and selected biomarkers, while recognition of differential diagnoses and comorbid conditions remains essential to avoid misclassification and inappropriate therapy. Current asthma management emphasizes individualized, stepwise treatment strategies that combine pharmacologic and nonpharmacologic interventions to achieve optimal symptom control and prevent exacerbations. Inhaled corticosteroids remain the cornerstone of long term therapy, with additional agents including long acting bronchodilators, leukotriene modifiers, antimuscarinics, and biologic therapies reserved for patients with more severe or refractory disease. Adjunctive approaches such as bronchial thermoplasty may provide benefit in carefully selected patients with severe asthma who remain uncontrolled despite optimized medical treatment. Equally important is the management of comorbid conditions and environmental triggers, which can significantly influence disease severity and treatment outcomes. Ongoing advances in the understanding of asthma immunopathogenesis and phenotypic variability are driving the development of more targeted and personalized therapeutic approaches. Future research should focus on refining diagnostic tools, identifying reliable biomarkers, and optimizing treatment strategies to improve long term outcomes, reduce disease burden, and enhance quality of life for individuals living with asthma.

Acknowledgment

The authors sincerely acknowledge their respective institutions for providing the necessary facilities and

academic support to carry out this review work. The authors are also grateful to colleagues and peers for their constructive suggestions during the preparation of the manuscript.

Author Contribution
RK; Literature survey, **TS;** Manuscript drafting, **AS;** Supervision and final approval.

Conflict of Interest
The authors declare no conflict of interest.

Source of Funding
There is no funding available to conduct this study.

Declarations
The authors declare that they used AI language tools (ChatGPT and Grammarly Premium) to enhance this manuscript's linguistic clarity and readability. They carefully reviewed and edited all generated text to ensure accuracy and alignment with the research's intended meaning.

References

1. D. A. Pleşca, M. Ionescu, and A. C. Dragănescu, “Asthma prevention and recent advances in management,” Glob. Pediatr., 2024, doi: 10.1016/j.gpeds.2024.100209.
2. M. R. Bhise, V. Trivedi, S. Devi, A. Kumar, T. Jayendra, and K. Sunand, “Graphene Quantum Dots in Cancer Diagnostics and Therapeutics : Advances in Biosensing , Imaging , and Treatment Applications,” Curr. Med. Sci., no. 0123456789, 2025, doi: 10.1007/s11596-025-00155-8.
3. D. Westhölter, S. Reuter, and C. Taube, “Advances in the management of asthma,” 2024. doi: 10.1055/a-2249-2207.
4. P. Vishvakarma et al., “Nanocarrier Mediated Delivery of Gepotidacin : A Novel Triazaacenaph- thylene Antibiotic Targeting Drug-Resistant Bacterial Infections,” Anti-Infective Agents, pp. 1–18, 2026, doi: 10.2174/0122113525430497251211145738.
5. I. Agache et al., “Advances and highlights in asthma in 2021,” 2021. doi: 10.1111/all.15054.
6. R. Ahmad et al., “Mechanistic insights into the neuroprotective effects of Radix Astragali (Huang Qi): Bridging Traditional Chinese Medicine and modern pharmacology,” Pharmacol. Res. - Mod. Chinese Med., vol. 17, no. July, p. 100711, 2025, doi: 10.1016/j.prmcm.2025.100711.
7. V. H. Patel et al., “Current Limitations and Recent Advances in the Management of Asthma,” Disease-a-Month, 2023, doi: 10.1016/j.disamonth.2022.101483.
8. S. Ali, S. A. Ali, S. Shamim, and N. A. Farooqui, “Emerging Trends in the Pathogenesis, Diagnosis, and Management of Human Metapneumovirus-Associated Respiratory Infections,” Anti-Infective Agents, vol. 24, 2025, doi: 10.2174/0122113525396349251011213223.
9. D. Westhölter, S. Reuter, and C. Taube, “Advances in the management of asthma,” Dtsch. Medizinische Wochenschrift, 2023, doi: 10.1055/a-1990-2341.
10. S. A. Ali, S. Ali, and S. Shamim, “Role of Artificial Intelligence in Transforming Diagnosis, Treatment, and Patient Care: A Review,” Curr. Pharmacogenomics Person. Med., vol. 23, 2025, doi:
11. Y. Gautam, E. Johansson, and T. B. Mersha, “Multi-Omics Profiling Approach to Asthma: An Evolving
12. I. Jahan, S. Ali, J. Hak, S. Shamim, M. Kumar, and T. Ali, “Emerging Trends in Magnetic Nanoparticle Delivery, Synthesis and Applications in Biomedicine,” Drug Deliv. Lett., vol. 16, pp. 1–17, 2025, doi: 10.2174/0122103031400035251014115919.
13. R. Castagnoli, I. Brambilla, M. Giovannini, G. L. Marseglia, and A. Licari, “New approaches in childhood asthma treatment,” 2023. doi: 10.1097/ACI.0000000000000922.
14. K. Singh et al., “Emerging Applications and Innovations in Emulgel Technology for Enhanced Topical Drug Delivery,” Drug Deliv. Lett., vol. 15, pp. 1–16, 2025, doi: 10.2174/0122103031384904250930153320.
15. K. R. Jat and A. Gupta, “Recent Advances in Long-Term Management of Asthma,” 2022. doi: 10.1007/s12098-021-04060-5.
16. M. C. Amado and J. M. Portnoy, “Recent advances in asthma management.,” 2006. doi: 10.17925/erpd.2018.4.1.15.
17. S. Siddiqui, S. Gonem, and A. Wardlaw, “Advances in the management of severe asthma,” Semin. Respir. Crit. Care Med., 2012, doi: 10.1055/s-0032-1326964.
18. R. Singh, S. Shamim, S. Ali, R. Kumar, and T. Ali, “A Global Public Health Review of the Mumps Virus: Epidemiology, Pathogenesis, and Advances in Vaccination,” Anti-Infective Agents, vol. 24, 2025, doi: 10.2174/0122113525412555250922164155.
19. D. J. Jackson, A. Sykes, P. Mallia, and S. L. Johnston, “Asthma exacerbations: Origin, effect, and prevention,” J. Allergy Clin. Immunol., 2011, doi: 10.1016/j.jaci.2011.10.024.
20. B. Khalid Salah Al-Sheakly, F. Saheb Sharif-Askari, N. Saheb Sharif-Askari, J. E. Hundt, and R. Halwani, “The potential role of nanobodies in asthma therapy,” 2024. doi: 10.3389/fphar.2024.1510806.
21. S. Benavides, J. C. Rodriguez, and M. Maniscalco-Feichtl, “Pharmacist involvement in improving asthma outcomes in various healthcare settings: 1997 to present,” 2009. doi: 10.1345/aph.1K612.
22. A. Shrivastava et al., “Heart Failure Management in the Modern Era: A Comprehensive Review on Medical and Device-based Interventions,” Curr. Cardiol. Rev., vol. 21, no. 6, pp. 1–14, 2025, doi: 10.2174/011573403x338702250226075044.

23. K. C. Doerschug, M. W. Peterson, C. S. Dayton, and J. N. Kline, “Asthma guidelines: An assessment of physician understanding and practice,” Am. J. Respir. Crit. Care Med., 1999, doi: 10.1164/ajrccm.159.6.9809051.
24. D. Dorscheid et al., “Airway epithelial cells as drivers of severe asthma pathogenesis,” 2025. doi: 10.1016/j.mucimm.2025.03.003.
25. S. et al. Singh, K., Gupta, J. K., Chanchal, D. K., Khan, S., Varma, A., Shanno, K., Kumar, S., & Shamim, “Deciphering the Genetic Landscape: Exploring the Relationship Between HLA-DQA1, HLA-DQB1, and HLA-DRB1 Genes in Diabetes Mellitus,” Curr. Pharmacogenomics Person. Med., vol. 21, no. 3, pp. 1–11, 2024, doi: 10.2174/0118756921310081240821065036.
26. N. Habib, M. A. Pasha, and D. D. Tang, “Current Understanding of Asthma Pathogenesis and Biomarkers,” 2022. doi: 10.3390/cells11172764.
27. A. Anand et al., “Neuroprotective Efficacy and Complementary Treatment with Medicinal Herbs: A Comprehensive Review of Recent Therapeutic Approaches in Epilepsy Management,” CNS Neurol. Disord. - Drug Targets, vol. 24, no. 1, pp. 60–73, 2024, doi: 10.2174/0118715273332140240724093837.
28. X. Zhang et al., “The onset, development and pathogenesis of severe neutrophilic asthma,” 2022. doi: 10.1111/imcb.12522.
29. P. Kumar et al., “Fused Deposition Modeling 3D-Printed Scafolds for Bone Tissue.pdf,” Appl. Biochem. Biotechnol., vol. 12, no. 22, pp. 1–11, 2024, doi: 10.54085/ap.2023.12.2.19.
30. Z. Qin et al., “Immunometabolism in the pathogenesis of asthma,” 2024. doi: 10.1111/imm.13688.
31. F. Cui, R. Liu, L. Wang, J. He, and Y. Xu, “Identification and validation of differentially expressed genes in allergic asthma pathogenesis using whole-transcriptome sequencing,” Front. Med., 2025, doi: 10.3389/fmed.2025.1545095.
32. J. Sleziak, A. Gawor, M. Błażejewska, K. Antosz, and K. Gomułka, “ADAM33′s Role in Asthma Pathogenesis: An Overview,” 2024. doi: 10.3390/ijms25042318.
33. M. W. Pijnenburg, U. Frey, J. C. De Jongste, and S. Saglani, “Childhood asthma: pathogenesis and phenotypes,” Eur. Respir. J., 2022, doi: 10.1183/13993003.00731-2021.
34. R. Louis et al., “European Respiratory Society guidelines for the diagnosis of asthma in adults,” 2022. doi: 10.1183/13993003.01585-2021.
35. D. Hassoun, L. Rose, F. X. Blanc, A. Magnan, G. Loirand, and V. Sauzeau, “Bronchial smooth muscle cell in asthma: where does it fit?,” 2022. doi: 10.1136/bmjresp-2022-001351.
36. M. A. Romero-Falcón et al., “Evaluation of the Diagnostic Accuracy of Non-Specific Bronchial Provocation Tests in the Diagnosis of Asthma: A Randomized Cross-Over Study,” Arch. Bronconeumol., 2023, doi: 10.1016/j.arbres.2022.10.008.
37. X. Wang, Y. Gao, H. Wang, X. Gong, and P. Bao, “Tumor markers for lipid metabolism-related genes: Based on small cell lung cancer and bronchial asthma dual analysis,” Environ. Toxicol., 2024, doi: 10.1002/tox.24152.
38. Shamim, S. Ali, T. Ali, H. Sharma, B. N. Kishor, and S. K. Jha, “Recent Advances in Monodisperse Gold Nanoparticle Delivery, Synthesis, and Emerging Applications in Cancer Therapy,” Plasmonics, no. 0123456789, 2025, doi: 10.1007/s11468-024-02732-4.
39. K. Miyake et al., “Maternal smoking status before and during pregnancy and bronchial asthma at 3 years of age: a prospective cohort study,” Sci. Rep., 2023, doi: 10.1038/s41598-023-30304-9.
40. D. Anikin, I. A. Soloveva, I. V. Demko, E. A. Sobko, N. V. Gordeeva, and A. Y. Kraposhina, “Bronchial Asthma and Obesity: Features of Systemic Inflammation Depending on the Time of Asthma Onset,” Russ. Med., 2025, doi: 10.17816/medjrf642919.
41. M. R. Khan, D. Kumar, S. Shamim, K. Sunand, S. Sharma, and G. Rawat, “Ethnopharmacological relevance of Citrus limon (L.) Burm. f. as adjuvant therapy,” Ann. Phytomedicine An Int. J., vol. 12, no. 2, pp. 169–179, 2023, doi: 10.54085/ap.2023.12.2.19.
42. B. E. Kim, J. W. Hui-Beckman, M. Z. Nevid, E. Goleva, and D. Y. M. Leung, “Air pollutants contribute to epithelial barrier dysfunction and allergic diseases,” 2024. doi: 10.1016/j.anai.2023.11.014.
43. M. Zhang, L. Zhao, P. Zeng, X. Mu, and J. Zhao, “Inflammatory and pulmonary function characteristics of bronchial asthma induced by COVID-19 infection,” Biomed. Reports, 2025, doi: 10.3892/br.2025.1979.
44. Rani Khan et al., “Formulation and Characterisation of Herbal Ethosomal Gel of Luliconazole and Clove Oil for Modified Drug Diffusion to the Skin,” World J. Adv. Res. Rev., vol. 18, no. 4, pp. 488–501, 2025, doi: 10.1016/j.jaim.2024.100947.
45. J. Wieczfińska and R. Pawliczak, “The impact of commercially available air purifiers on bronchial asthma and allergy symptoms,” Alergol. Pol. - Polish J. Allergol., 2022, doi: 10.5114/pja.2022.114396.
46. A. Salama, M. S. O. Gouida, N. N. Yassen, and A. A. Sedik, “Immunoregulatory role of hesperidin against ovalbumin (OVA)-induced bronchial asthma and depression in rats,” Naunyn. Schmiedebergs. Arch. Pharmacol., 2024, doi: 10.1007/s00210-023-02833-7.
47. T. Ali, “Chromatography and Spectroscopic Characterization of Nano-Carrier Pharmaceuticals,” Pharm. Nanotechnol., 2024, doi: 10.2174/0122117385319695240911115239.
48. B. G, “Bronchial Asthma: Etiology, Pathophysiology, Diagnosis and Management,” Austin J. Pulm. Respir. Med., 2022, doi: 10.26420/austinjpulmrespirmed.2022.1085.
49. H. S. S. & A. B. Pawan Kumar, Shamim, Mohammad Muztaba, Tarmeen Ali, Jyoti Bala,

"Fused Deposition Modeling 3D-Printed Scafolds for Bone Tissue.pdf," 2024, Springer Nature. doi: 10.1007/s10439-024-03479.
50. A. H. Aljohani, H. Alsufiani, and G. Ahmed, "Non-invasive management of severe subcutaneous emphysema in a pediatric asthma exacerbation: a case report and review," Int. J. Emerg. Med., 2024, doi: 10.1186/s12245-024-00804-0.
51. D. Ukena, "Bronchial Asthma: Diagnosis and Long-Term Treatment in Adults: In Reply," Dtsch. Arztebl. Int., 2008, doi: 10.3238/arztebl.2008.0842b.
52. H. Shi and K. Huang, "Annual progress on bronchial asthma diagnosis and treatment in 2023," 2024. doi: 10.3760/cma.j.cn112147-20231129-00348.
53. M. P. Muñoz, V. Iglesias, M. Saavedra, G. Saavedra, K. Yohannessen, and P. Pino, "Urinary Arsenic Concentration and Its Relationship with Bronchial Asthma in Children from Arica, Chile," Toxics, 2022, doi: 10.3390/toxics10100625.
54. S. Mandilwar, S. M. Thorve, V. Gupta, and P. Prabhudesai, "Role of impulse oscillometry in diagnosis and follow-up in bronchial asthma," Lung India, 2023, doi: 10.4103/lungindia.lungindia_251_22.
55. C. G. Byers and N. Dhupa, "Feline bronchial asthma: Pathophysiology and diagnosis," 2005.
56. A. Firmansyah, S. Nurwahidah, D. Hamdani, A. Fitriani, and A. Gunawan, "The Effectiveness of Coughing Effectively for Removing Secretions In Clients of Bronchial Asthma : Case study," Heal. Care Nurs. J., 2023.
57. K. Singh et al., "Recent Advances in the Synthesis of Antioxidant Derivatives: Pharmacological Insights for Neurological Disorders," Curr. Top. Med. Chem., vol. 24, no. 22, pp. 1940–1959, 2024, doi: 10.2174/0115680266305736240725052825.
58. X. Gai, B. Allwood, and Y. Sun, "Post-tuberculosis lung disease and chronic obstructive pulmonary disease," 2023. doi: 10.1097/CM9.0000000000002771.
59. M. Wakazono et al., "Prevalence and clinical impact of asthma-COPD overlap in severe asthma," Allergol. Int., 2025, doi: 10.1016/j.alit.2024.11.003.
60. K. Singh et al., "Deciphering the Genetic Landscape: Exploring the Relationship Between HLA-DQA1, HLA-DQB1, and HLA-DRB1 Genes in Diabetes Mellitus," Curr. Pharmacogenomics Person. Med., vol. 21, pp. 1–11, 2024, doi: 10.2174/0118756921310081240821065036.
61. S. Cox and C. Cornelius, "Congestive Heart Failure," in Hospice and Palliative Care for Companion Animals: Principles and Practice, Second Edition, 2024. doi: 10.1002/9781119808817.ch13.
62. H. Sharma, A. P. Singh, D. Pathak, D. Taumar, and V. Chaudhary, "The Role of approved Kinase Inhibitors in Cancer Treatment : Medicinal Chemistry and Pharmacological Insights," Med. Chem. (Los. Angeles)., pp. 1–18, 2025, doi: 10.2174/0115734064404334251024112120.
63. S. George and S. Suresh, "Vocal cord dysfunction: Analysis of 27 cases and updated review of pathophysiology & management," 2019. doi: 10.1055/s-0038-1661358.
64. T. Leech and M. Peiris, "Mucosal neuroimmune mechanisms in gastro-oesophageal reflux disease (GORD) pathogenesis," 2024. doi: 10.1007/s00535-023-02065-9.
65. M. Lucanska, A. Hajtman, V. Calkovsky, P. Kunc, and R. Pecova, "Upper airway cough syndrome in pathogenesis of chronic cough," 2020. doi: 10.33549/physiolres.934400.
66. J. Kumar et al., "CFTR mRNA-Based Gene Therapy for Cystic Fibrosis: A Mutation-Agnostic Strategy to Restore Ion Transport Function," Curr. Gene Ther., no. January, pp. 1–18, 2025, doi: 10.2174/0115665232413980251103133741.
67. M. Barbosa and J. D. Chalmers, "Bronchiectasis," 2023. doi: 10.1016/j.lpm.2023.104174.
68. G. Pavliša and E. Tolić, "Pulmonary Embolism," Medicus, 2024, doi: 10.12968/bjha.2014.8.6.268.
69. A. P. Singh et al., "Revolutionizing Drug and Gene Delivery: Cutting-Edge Smart Polymers for Precision Release," Curr. Gene Ther., no. January, pp. 1–19, 2025, doi: 10.2174/0115665232390238251121074917.
70. M. A. Althobiani et al., "Interstitial lung disease: a review of classification, etiology, epidemiology, clinical diagnosis, pharmacological and non-pharmacological treatment," 2024. doi: 10.3389/fmed.2024.1296890.
71. D. E. Mîndru et al., "Foreign Body Aspiration in Children—Retrospective Study and Management Novelties," Med., 2023, doi: 10.3390/medicina59061113.
72. A. Ozkaraman, "Obstructive sleep apnea," in Medical Nursing, 2023. doi: 10.21474/ijar01/12634.
73. P. Chandel, S. Shamim, and S. Ali, "A Comprehensive Review on Nanoemulsions for Improved Bioavailability and Therapeutic Efficacy in Gastrointestinal Disorders," Drug Deliv. Lett., no. August, pp. 1–18, 2021, doi: 10.2174/0122103031410345251205075031.
74. S. F. Javaid, I. J. Hashim, M. J. Hashim, E. Stip, M. A. Samad, and A. Al Ahbabi, "Epidemiology of anxiety disorders: global burden and sociodemographic associations," Middle East Curr. Psychiatry, 2023, doi: 10.1186/s43045-023-00315-3.
75. P. Kumar et al., "Trends of Nanobiosensors in Modern Agriculture Systems," Appl. Biochem. Biotechnol., vol. 197, no. 1, pp. 667–690, 2024, doi: 10.1007/s12010-024-05039-6.
76. J. Chamorro et al., "Small cell lung cancer," Med., 2025, doi: 10.1016/j.med.2025.03.001.
77. W. Soewoto and M. Ardianti, "Tracheomalacia

following a total thyroidectomy in a patient with a large non-toxic goiter: A case report," Int. J. Surg. Case Rep., 2024, doi: 10.1016/j.ijscr.2023.109211.
78. S. A. Ali, S. Ali, S. Rastogi, B. Shivhare, and M. Muztaba, "A Comprehensive Review on Advancements in Nanocarriers-Based Peptide Delivery for Cancer Therapeutics," Micro Nanosyst., vol. 17, no. 4, pp. 283–297, 2025, doi: 10.2174/0118764029358553250325040749.
79. J. H. Hull, A. R. Jackson, C. Ranson, F. Brown, M. Wootten, and M. Loosemore, "The benefits of a systematic assessment of respiratory health in illnesssusceptible athletes," Eur. Respir. J., 2021, doi:
80. S. Chawla, R. Gupta, S. K. Jha, and K. T. Jha, "Stereoisomerism in Chemistry and Drug Development: Optical, Geometrical, and Conformational Isomers," Med. Chem. (Los. Angeles)., 2025, doi: 10.2174/0115734064366389250923044201.
81. M. Nassif et al., "Atrial septal defect in adults is associated with airway hyperresponsiveness," Congenit. Heart Dis., 2018, doi: 10.1111/chd.12665.
82. Mukesh Kumar, Shadab Ali, and Smriti Gohri, "Microsponge Drug Delivery Systems: Advancing Methotrexate Delivery for Rheumatoid Arthritis Management," Curr. Pharm. Res., vol. 1, no. 1, pp. 15–29, 2025, doi: 10.63785/cpr.2025.1.1.1529.
83. L. B. Bacharier et al., "Blood eosinophils and fractional exhaled nitric oxide are prognostic and predictive biomarkers in childhood asthma," J. Allergy Clin. Immunol., 2024, doi: 10.1016/j.jaci.2023.09.044.
84. A. et al. Kumar, J., M., T., Musayev, "Stimuli-responsive Hydrogels for Targeted Antibiotic Delivery in Bone Tissue Engineering," AAPS PharmSciTech, vol. 26, no. 217, pp. 1–23, 2025, doi: https://doi.org/10.1208/s12249-025-03218-0.
85. S. A. Meo, M. A. Salih, J. M. Alkhalifah, A. H. Alsomali, and A. A. Almushawah, "Effect of Air Pollutants Particulate Matter PM2.5, PM10, Carbon Monoxide (CO), Nitrogen Dioxide (NO2), Sulfur Dioxide (SO2), and Ozone (O3) on Fractional Exhaled Nitric Oxide (FeNO)," Pakistan J. Med. Sci., 2024, doi: 10.12669/pjms.40.8.9630.
86. Tarmeen Ali, "Nanomedicine Approaches to Overcome Barriers in Pulmonary Drug Delivery for Respiratory Diseases," Curr. Pharm. Res., vol. 1, no. 1, pp. 30–44, 2025, doi: 10.63785/cpr.2025.1.1.3044.
87. Y. Feng, S. Zhang, Y. Shang, and N. Chen, "The Use of Exercise Challenge Testing and Fractional Exhaled Nitric Oxide in Diagnosis of Chest Tightness Variant Asthma in Children," Int. Arch. Allergy Immunol., 2022, doi: 10.1159/000522062.
88. D. Le Pham, K. M. Le, D. D. K. Truong, H. T. T. Le, and T. H. K. Trinh, "Environmental allergen reduction in asthma management: an overview," 2023. doi: 10.3389/falgy.2023.1229238.
89. Abhinay Tiwari, Anshu, Chirag Kumar, and Moh. Zaid, "Unravelling the Herbal Formulation of Floating Microspheres for Gut Microbiome Modulation: Curren1Abhinay Challenges and Future Prospects," Curr. Pharm. Res., vol. 1, no. 1, pp. 144–162, 2025, doi: 10.63785/cpr.2025.1.1.144162.
90. H. Almuhanna, M. Alenezi, M. Abualhasan, S. Alajmi, R. Alfadhli, and A. S. Karar, "AI Asthma Guard: Predictive Wearable Technology for Asthma Management in Vulnerable Populations," Appl. Syst. Innov., 2024, doi: 10.3390/asi7050078.
91. Bhanu Pratap and Pankaj Singh Jadaun, "Hydrogel Microneedles: A Breakthrough in Disease Treatment and Drug Delivery Systems," Curr. Pharm. Res., vol. 1, no. 2, pp. 60–77, 2025, doi: 10.63785/cpr.2025.1.1.6077.
92. N. BIETH, B. ROUOT, J. SCHWARTZ, and J. VELLY, "COMPARISON OF PHARMACOLOGICAL AND BINDING ASSAYS FOR TEN β-ADRENOCEPTOR BLOCKING AGENTS AND TWO β-ADRENOCEPTOR AGONISTS," Br. J. Pharmacol., 1980, doi: 10.1111/j.1476-5381.1980.tb14572.x.
93. P. Das, D. Kumar, V. Gupta, S. Gupta, R. Tanwar, and K. Behmani, "Sphingosomes: Advancements and Emerging Applications in Advanced Drug Delivery Systems," Curr. Pharm. Res., vol. 1, no. 2, pp. 199–208, 2025, [Online]. Available: https://cpr.org.in/index.php/files/article/view/134/83
94. A. Perra et al., "Potential role of two novel agonists of thyroid hormone receptor-β on liver regeneration," Cell Prolif., 2020, doi: 10.1111/cpr.12808.
95. Manisha Dev and Pallavi Chandel, "Nanostructured Lipid Carriers in Pulmonary Drug Delivery: Progress and Prospects," Curr. Pharm. Res., vol. 1, no. 1, pp. 131–143, 2025, doi: 10.63785/cpr.2025.1.1.131143.
96. S. Bien, D. Bartusik-Aebisher, and D. Aebisher, "Corticosteroids," in The Biochemical Guide to Hormones, 2023. doi: 10.5040/9798400694868.ch-010.
97. J. Mahmoud, M. A. Bovy, N. Heming, and D. Annane, "Corticosteroids in sepsis," 2025. doi: 10.1016/j.jointm.2025.08.006.
98. D. Kumar, Rohan Kumar, Harsh Raj Singh, Rajni Tanwar, and Vrinda Gupta, "Duvyzat (Givinostat) in Duchenne Muscular Dystrophy: Mechanisms, Clinical Impact, and Future Directions," Curr. Pharm. Res., vol.
99. D. Chaudhuri et al., "2024 Focused Update: Guidelines on Use of Corticosteroids in Sepsis, Acute Respiratory Distress Syndrome, and Community-Acquired Pneumonia," Crit. Care Med., 2024, doi: 10.1097/CCM.0000000000006172.
100. P. Devillier, N. Baccard, and C. Advenier, "Leukotrienes, leukotriene receptor

antagonists and leukotriene synthesis inhibitors in asthma: An update. Part II: Clinical studies with leukotriene receptor antagonists and leukotriene synthesis inhibitors in asthma," Pharmacol. Res., 1999, doi: 10.1006/phrs.1998.0461.

101. P. M. O'Byrne, G. M. Gauvreau, and D. M. Murphy, "Efficacy of leukotriene receptor antagonists and synthesis inhibitors in asthma," 2009. doi: 10.1016/j.jaci.2009.05.029.
102. C. Sanjinez, E. Botteri, N. C. Støer, and L. Lukas Löfling, "Antimuscarinics and lung cancer survival: A Norwegian population-based cohort study," Lung Cancer, 2023, doi: 10.1016/j.lungcan.2023.107187.
103. P. Chandel, T. Singh, A. Sharma, F. Sahib, and M. Gobindgarh, "Emerging Biomarkers for Early Detection of Alzheimer ' s Disease : Progress and Challenges School of Pharmacy , Deshbhagat University , off to NH-44 , Amloh Road , Mandi Gobindgarh District," Curr. Pharm. Des., vol. 1, no. 3, pp. 364–372, 2025.
104. S. Yamada, M. Mochizuki, K. Maruyama-Fumoto, S. Kagota, and K. Shinozuka, "Additive effects of mirabegron on muscarinic receptor binding and on relaxation of cholinergic detrusor muscle contraction by antimuscarinics," J. Pharmacol. Sci., 2025, doi: 10.1016/j.jphs.2025.05.018.
105. N. C. Thomson, "Recent developments in bronchial thermoplasty for severe asthma," 2019. doi: 10.2147/JAA.S200912.
106. S. K. Kushwaha, P. Chandel, T. Singh, M. Thakur, and U. Pradesh, "Formulation and Evaluation of a Multicomponent Herbal Shampoo for Enhanced Hair and Scalp Health Department of Pharmacy , IIMT College of Medical Sciences , IIMT University , Ganga Nagar , Meerut- School of Pharmacy , Deshbhagat University Amloh Road , Ma," Curr. Pharm. Res., vol. 1, no. 3, pp. 373–383, 2025.
107. Y. Kai, K. Suzuki, R. Kataoka, I. Sato, S. Tamaki, and S. Muro, "Efficacy of tezepelumab against uncontrolled severe non-type 2 asthma refractory to bronchial thermoplasty, benralizumab, dupilumab and mepolizumab," Respirol. Case Reports, 2024, doi: 10.1002/rcr2.1311.
108. S. Rana, B. Pratap, P. S. Jadaun, and U. Pradesh, "Development and Evaluation of Liquorice Root Extract-Based Cream for the Management of Hyperpigmentation" Curr. Pharm. Res., vol. 1, no. 3, pp. 384–392, 2025.
109. S. Vañó-Galván et al., "Characteristics and Management of Patients with Alopecia Areata and Selected Comorbid Conditions: Results from a Survey in Five European Countries," Dermatol. Ther. (Heidelb)., 2024, doi: 10.1007/s13555-024-01133-z.