PAKISTAN CHEST SOCIETY

PAKISTAN
CHEST SOCIETY
STRIVING FOR PULMONARY CARE

Clinical Practice Guidelines

Asthma

PAKISTAN CHEST SOCIETY-2026

Guidelines On

Asthma

March 2026

PAKISTAN CHEST SOCIETY

PAKISTAN
CHEST SOCIETY
STRIVING FOR PULMONARY CARE

Table of Contents

Chapter 03

Chapter 04

Chapter 05

Chapter 06

Preface

Asthma remains one of the most prevalent chronic respiratory diseases in Pakistan, affecting children and adults across all socioeconomic strata. Despite the availability of effective therapies, asthma continues to contribute substantially to avoidable morbidity, emergency healthcare utilization, loss of productivity, and impaired quality of life in our population. Contributing factors include delayed diagnosis, inappropriate reliance on short-acting β_2-agonists, poor inhaler technique, limited access to specialist care, and unique environmental exposures such as biomass fuel smoke, urban air pollution, occupational hazards, and recurrent respiratory infections.

The Pakistan Chest Society (PCS) Asthma Guidelines 2026 have been developed to address these challenges by providing contextualized, evidence-based, and practical recommendations tailored to the realities of clinical practice in Pakistan. These guidelines synthesize high-quality evidence from leading international sources—including the Global Initiative for Asthma (GINA), British Thoracic Society (BTS), ERS/ATS, NICE, and peer-reviewed literature—while adapting recommendations to local healthcare infrastructure, medication availability, patient behavior, and environmental risk factors.

A major emphasis of these guidelines is the paradigm shift away from SABA-only treatment toward anti-inflammatory reliever strategies, including AIR and MART pathways, reflecting contemporary evidence on asthma safety and outcomes. Equal importance has been placed on accurate diagnosis, assessment of future risk, phenotype-driven management, patient education, inhaler technique, and structured follow-up. Special sections addressing asthma exacerbations, asthma–COPD overlap, pediatric asthma, pregnancy, occupational asthma, and other special situations further enhance the applicability of this document.

The development of these guidelines represents a collective national effort. I would like to acknowledge the dedication, intellectual rigor, and voluntary commitment of all members of the PCS Asthma Guidelines Working Group, reviewers, and contributors who invested significant time in evidence appraisal, drafting, and consensus building. Their multidisciplinary expertise has ensured that these recommendations are scientifically robust yet clinically pragmatic.

It is my sincere hope that these guidelines will serve as a trusted reference for pulmonologists, internists, family physicians, pediatricians, trainees, and allied healthcare professionals, ultimately contributing to improved asthma outcomes and reduced disease burden in Pakistan.

On behalf of the Pakistan Chest Society, I commend this document to the medical community and encourage its widespread adoption, regular updating, and continuous audit in clinical practice.

Prof. Saadia Ashraf
Chairperson
PCS Asthma Guidelines Working Group

Message by the President

Pakistan Chest Society

Asthma is a prevalent chronic respiratory disease with significant impact on quality of life when inadequately controlled. These guidelines emphasize early diagnosis, severity assessment, stepwise pharmacological management, correct inhaler technique and patient education. The Pakistan Chest Society encourages clinicians to adopt these recommendations to achieve optimal asthma control and reduce preventable morbidity.

Prof. Shereen Khan
President
Pakistan Chest Society

Message by the Chairman

Guideline Committee, Pakistan Chest Society

It gives me great pleasure to present the Guidelines for the Management of Asthma developed by the Working Group for Asthma guidelines under chairperson Prof. Sadia Ashraf. Asthma remains one of the most common chronic respiratory diseases worldwide and continues to pose a significant public health burden in Pakistan. Despite the availability of effective therapies, many patients remain underdiagnosed or inadequately treated due to limited awareness, environmental exposures, and cultural misconceptions regarding inhaler use.

In Pakistan, factors such as air pollution, tobacco smoke exposure, respiratory infections, and rapid urbanization contribute to the rising prevalence of asthma. Recognizing these challenges, these guidelines aim to provide practical, evidence-based recommendations tailored to the realities of our healthcare system. The document outlines the epidemiology, early diagnosis, severity assessment, and stepwise management of asthma, with emphasis on appropriate inhaler therapy, patient education, trigger avoidance, and regular follow-up to achieve optimal disease control and prevent exacerbations.

These guidelines also highlight the importance of addressing cultural stigma associated with asthma and improving awareness among both healthcare professionals and patients. By promoting standardized and evidence-based care, we hope to reduce the disease burden and improve the quality of life of asthma patients across Pakistan.

On behalf of the Guidelines Committee, I extend my sincere gratitude to all experts and contributors who dedicated their time and expertise to the development of these guidelines.

Prof. Muhammad Ashraf Jamal
Chairman, Guideline Committee
Pakistan Chest Society

Pakistan Chest Society

Guideline Committee

Prof. Muhammad Ashraf Jamal

Chairman, Guidelines Committee
Pakistan Chest Society

Prof. Nisar Ahmed Rao

Professor of Pulmonology
Fazaia Ruth Pfau Medical College & Hospital, Karachi

Prof. Saadia Ashraf

Head of the Pulmonology Department
Khyber Teaching Hospital, MTI, Peshawar

Brig (R) Jamal Ahmad

Head of the Pulmonology Department
Fauji Foundation Hospital Rawalpindi

Prof. Talha Mahmud

Professor & Head of Department (Pulmonology)
Shaikh Zayed Medical Complex, Lahore

Dr. Maqbool A Langove

Associate Professor, Department of Pulmonology
Fatima Jinnah General and Chest Hospital, Quetta

Dr. Kamran Khan Sumalani

Associate Professor, Department of Pulmonology
Jinnah Postgraduate Medical Center, Karachi

Asthma
Guideline Working Group

Dr. Saadia Ashraf (Chairperson)
Prof & HOD Pulmonology & Critical Care Division
Khyber Medical College/Khyber Teaching Hospital, Peshawar
Email: saadiaashraf1@gmail.com

Dr. Kamran Khan Sumalani
Associate Professor Pulmonology,
Jinnah Post graduate Medical Centre, Karachi.
Email: drkamrankn@hotmail.com

Dr. Nousheen Akhter
Assistant Professor Pulmonology,
Bahria University Health Sciences Campus Consultant
Pulmonologist, PNS Shifa Hospital Karachi.
Email: dr.noshi_87@hotmail.com

Dr. Muhammad Sajjad Sarwar
Assistant Professor Pulmonology
Bahawal Victoria Hospital/ QAMC Bahawalpur, Punjab
Email: sajjadshahzad99@gmail.com

Dr. Zia Ul Haq
Assistant Professor Pulmonology
Pak international medical college, Hayatabad, Peshawar
Email: drziaulhaqlrh@gmail.com

Dr. Fouzia Raza
Consultant Pulmonologist
Fatima Jinnah Institute of Chest Diseases, Quetta
Email: raza.fouzia@gmail.com

Dr Hafiz Usman Khalid
Senior Registrar Pulmonology
Fauji Foundation hospital, Rawalpindi
Email: usmanr1256@gmail.com

Abbreviations

ACQ: Asthma Control Questionnaire

ACT: Asthma Control Test

AIR: Anti-inflammatory reliever

BDP: Beclomethasone dipropionate

BHR: Bronchial Hyper Responsiveness

BTS: British Thoracic Society

DPI: Dry powder inhaler

EIA: Exercise-induced asthma

FeNO: Fractional concentration of exhaled nitric oxide

FEV1: Forced expiratory volume in 1 second

FVC: Forced Vital Capacity

GINA: Global Initiative for Asthma

HFA: Hydro fluoroalkane

ICS: Inhaled corticosteroid

LABA: Long-acting β-agonist

LTRA: Leukotriene receptor antagonist

MART: Maintenance-and-reliever therapy

OCS: Oral corticosteroid

PCS: Pakistan Chest Society

PBD: Post-bronchodilator

PEF: Peak expiratory flow

pMDI: Pressurized metered dose inhaler

SABA: Short-acting β_2-agonist

TSLP: Thymic Stromal Lymphopoietin

Chapter 01:

Introduction, Scope and Epidemiology of Asthma

Asthma is a heterogeneous chronic inflammatory disease of the airways, characterized by a history of respiratory symptoms such as wheeze, shortness of breath, chest tightness, and cough that vary over time and in intensity [1]. In Pakistan, asthma represents a significant public health challenge due to environmental and socio-economic factors.

Scope of the Guideline

These guidelines provide an evidence-based approach to the prevention, diagnosis, and management of asthma in Pakistan. While global standards provide the scientific foundation, this document is tailored to address resource stratification, strategies to mitigate local triggers like pollen, molds, smog, etc. and accessibility of medicines.

Target Audience

This document serves as the national standard for pulmonologists, pediatricians managing asthma, general practitioners, emergency physicians, respiratory therapists and counsellors.

Adaptation from International Guidelines

These guidelines are primarily adapted from the Global Initiative for Asthma (GINA 2025) and the British Thoracic Society (BTS). These guidelines emphasize a pragmatic approach when objective testing like spirometry and phenotyping is unavailable, ensuring patients are not denied controller therapy due to resource constraints.

Epidemiology

Asthma is a major non-communicable disease, affecting both children and adults, and is the most common chronic disease among children. Asthma affected an estimated 262 million people in 2019[2] and caused 455 000 deaths.

Pakistan is the world's fifth most populated country with 221 million inhabitants. Approximately 4.3% of these are suspected to suffer from asthma[3]. Based on 2020 WHO mortality statistics, asthma remains a significant cause of death in Pakistan, claiming 20,750 lives annually and accounting for 1.42% of the country's total mortality [3]. With an age-adjusted death rate of 16.73 per 100,000 population, Pakistan is currently ranked 30th globally in asthma-related mortality [4].

Local Barriers

A sound asthma program in Pakistan must address these four indigenous challenges:

- Inhalers are often perceived as a "last resort" or "addictive."
- Environmental factors like paper mulberry pollen, biomass exposure, smog
- High out-of-pocket costs of controller medicines
- High prevalence of certain infections like TB, leading to under or over diagnosis of asthma

References

1.Global Initiative for Asthma. Global Strategy for Asthma Management and Prevention [Internet]. 2025. Available from: https://ginasthma.org/

2.Global burden of 369 diseases and injuries in 204 countries and territories, 1990–2019: a systematic analysis for the Global Burden of Disease Study 2019. Lancet. 2020;396(10258):1204-22

3.Yusuf O. Pakistan. In: Global Asthma Network. The Global Asthma Report 2022. Auckland, New Zealand: Global Asthma Network; 2022. Available from: https://globalasthmareport.org/regions/pakistan.php

4.World Life Expectancy. Pakistan: Asthma [Internet]. 2020. Available from: https://www.worldlifeexpectancy.com/pakistan-asthma

Chapter 02:

Asthma Definition & Diagnosis

Definition

Asthma is a heterogeneous disease characterized by chronic airway inflammation. It is defined by the history of respiratory symptoms, such as wheeze, shortness of breath, chest tightness and cough, that vary over time and in intensity, together with variable expiratory airflow.

Adopted from GINA 2025

History

A pattern of respiratory symptoms that occur following exposure to triggers (e.g., allergen, exercise, viral infection) and resolve with trigger avoidance or asthma medication is typical of asthma. Some patients will report all four of the classic symptoms of asthma, while others may report only one or two. "Wheezing" does not have a standard meaning for patients and may be used by those without a medical background to describe a variety of sounds, including upper airway noises emanating from the nose or throat. Cough may be dry or productive of clear mucoid or pale-yellow sputum (made discolored by the presence of eosinophils). Asthma is a potential cause of unexplained chronic cough. Some patients describe chest tightness, a band-like constriction, or the sensation of a heavy weight on the chest. In contrast, sharp chest pain is rarely used to describe the sensation of asthma. Because the symptoms of asthma are also seen in several other respiratory diseases, it is difficult to be certain of the diagnosis of asthma based upon history alone.[1,2] However, certain features on history increase the probability of asthma:

- Symptoms vary over time and in intensity
- Symptoms often worse at night or on waking[3,4]
- Symptoms are often triggered by allergens (dust mites, molds, furry animals, cockroaches, and pollens), cold air, exercise, laughter[5]
- Symptoms worsen after end-exercise
- Symptoms often appear or worsen with viral infections

A strong family history of asthma and allergies or a personal history of atopic diseases (e.g., atopic dermatitis, seasonal or perennial allergic rhinitis and conjunctivitis) favors a diagnosis of asthma in a patient with suggestive respiratory symptoms.

Recollection of childhood symptoms of chronic cough, nocturnal cough in the absence of respiratory infections, or a childhood diagnosis of "recurrent bronchitis" or "wheezy bronchitis" favors asthma but may also be reported in someone with bronchiectasis or simply frequent childhood respiratory infections. A history of childhood asthma that abated in late childhood or early adulthood combined with “new onset” of asthmatic symptoms in adulthood favors a diagnosis of recurrent asthma.

Certain features in history decrease the probability of asthma. These include:

- Lack of improvement following anti-asthmatic medications – Patients who have tried an inhaled bronchodilator (with proper inhaler technique) and obtained no relief of their symptoms are less likely to have asthma. Similarly, lack of dramatic improvement with a course of oral glucocorticoids suggests a diagnosis other than asthma.
- Onset of symptoms after age 50 – In middle-aged and older patients, other respiratory and cardiovascular diseases with overlapping manifestations become the more likely explanation for shortness of breath, cough, and wheeze, although the new onset of asthma remains a possibility.
- Concomitant cardiovascular symptoms – Symptoms such as chest pain,

lightheadedness, syncope, or palpitations suggest an alternate cardiovascular diagnosis (e.g., pulmonary vascular disease, cardiomyopathy, early coronary artery disease, or pericardial disease).
- History of cigarette smoking for many years – In patients with more than 20 pack-years of cigarette smoking, the likely etiology of cough, wheeze, and shortness of breath shifts away from asthma toward chronic obstructive pulmonary disease, although the two diseases can coexist.

Physical Examination

High-pitched expiratory wheezing is the most common pulmonary examination finding in patients with asthma. Widespread, high-pitched, musical wheezes are a characteristic feature of asthma, although wheezes are not specific for asthma and are usually absent between asthma exacerbations. Wheezes are heard most commonly on expiration but can also occur during inspiration. Asthmatic wheezing usually involves sounds of multiple different pitches, or "polyphonic," starting and stopping at various points in the respiratory cycle and varying in tone and duration over time. It is different from the monophasic wheezing of a local bronchial narrowing (e.g., due to an aspirated foreign body or bronchogenic cancer), which has single pitch and repeatedly begins and ends at the same point in each respiratory cycle.

Importantly, the presence or absence of wheezing on physical examination is a poor predictor of the severity of airflow obstruction in asthma. Wheezing may be heard in patients with mild, moderate, or severe airway narrowing, while widespread airway narrowing may be present in individuals without wheezing. Thus, the presence of wheezing alerts one to the likely presence of airway narrowing, but not its severity.

Expiratory noises transmitted from the upper airway (e.g., larynx, pharynx) can mimic wheezing and are often described as wheezing by patients. They are often audible without the use of a stethoscope. However, these upper airway noises are typically loudest over the neck and greatly diminished over the chest in contrast to true wheezes that are typically louder over the chest. Patients may be able to identify respiratory noises as inspiratory rather than expiratory, a description generally favoring an etiology other than asthma. Clinicians can usually distinguish the low-pitched wheezes (also called "rhonchi") that clear with cough, a sign of increased airway secretions as may be seen in bronchitis or bronchiectasis, from the typical high-pitched expiratory wheezes of asthma.

Other manifestations of severe airflow obstruction (e.g., use of accessory muscles, tachypnea, tachycardia, pulsus paradoxus, prolonged expiratory phase of respiration, silent chest, tripod position).[1,4] However, these signs are insensitive manifestations of severe airflow obstruction; their absence does not exclude the possibility of a severe asthmatic attack.

Extrapulmonary manifestations of allergic diseases in the upper airways or skin also increase the likelihood of an asthma diagnosis, like:

- Pale, swollen membranes on examination of the nasal cavities with an otoscope and a cobblestone appearance to the posterior pharyngeal wall suggest associated allergic rhinitis, a common condition among patients with allergic asthma.
- Nasal polyps, which appear as glistening, gray, mucoid masses within the nasal cavities, should prompt questioning about concomitant aspirin sensitivity, anosmia, and chronic sinusitis.
- Atopic dermatitis with typical lichenified plaques in a flexural distribution, especially of the antecubital and popliteal fossae, volar aspect of the wrists, ankles, and neck, may

accompany asthma in adolescents and adults. In early childhood it is a risk factor for the later development of asthma, with as many as a third of children with atopic dermatitis progressing to asthma.

Clubbing is not a feature of asthma; its presence should direct the clinician toward alternative diagnoses such as interstitial lung disease, lung cancer, and diffuse bronchiectasis, including cystic fibrosis.

Diagnosis of Asthma

Diagnosing asthma remains a clinical challenge due to the absence of a single definitive test and the variability of symptoms. Asthma diagnosis should not rely solely on spirometry, especially in children, given its low sensitivity in detecting intermittent airway obstruction. Clinical recognition must consider several key elements:

- **Variability of Symptoms:** Symptoms (wheezing, shortness of breath, chest tightness, and cough) may be episodic and are often triggered by factors such as exercise, allergens, viral infections, or environmental exposures.
- **Reversibility of Airflow Limitation:** Objective testing (e.g., spirometry or peak expiratory flow variability) demonstrating reversible obstruction is central but not always present at initial evaluation.
- **Phenotypic Diversity:** The presence of distinct phenotypes—including allergic, non-allergic, late-onset, or asthma with persistent airflow limitation—adds further complexity to diagnosis.
- **Misdiagnosis Risks:** Over-diagnosis may occur when symptoms mimic other conditions (e.g., vocal cord dysfunction or cardiac issues), while under-diagnosis is common when variable symptoms are not fully appreciated.

Criteria for initial diagnosis of asthma requires:

1. History of typical variable respiratory symptoms (discussed above)
2. Confirmed variable expiratory airflow (Table 1)

Table 1: Parameters for assessing variable expiratory airflow and criteria to diagnose asthma.

Post-bronchodilator (PBD) reversibility on spirometry*	Adults: ≥12% and ≥200 mL increase in FEV1 or FVC from baseline Children: ≥12% increase in FEV1 from baseline
Post-bronchodilator reversibility on PEF*	Adults: ≥ 20% increase in PEF Children: ≥ 15% increase in PEF
Diurnal variability in PEF over 2 weeks#	Adults: >10% daily variability Children: >13% daily variability
Increase in lung function after 4 weeks of ICS-containing treatment	Adults: Same as for PBD reversibility on spirometry or PEF Children: Same as for PBD reversibility on spirometry or PEF
Positive bronchial challenge test	Adults: ≥ 20% fall from baseline in FEV1 (methacholine) ≥ 15% fall from baseline in FEV1 (mannitol, hypertonic saline, hyperventilation) > 10% and > 200ml (exercise) Children: > 12% fall in FEV1, or > 15% fall in PEF (exercise)
Excessive variation in lung function between visits	Adults: Same as for PBD reversibility on spirometry or PEF Children: Same as for PBD reversibility on spirometry or PEF

* Measure baseline readings of FEV1, FVC or PEF. Administer 200 – 400 mcg nebulized salbutamol or equivalent. Measure PBD readings after 10 – 15 minutes. A rapid acting ICS-LABA may also be used.

Daily diurnal peak flow variability = $\frac{\text{PEF max} - \text{PEF min}}{\text{Mean of PEF max and PEF min}}$

(Averaged over 2 weeks)

Note: The criteria for a "positive" response have been simplified and made more stringent. A positive bronchodilator response can be taken as an increase in FEV1 or FVC of > 10% of the predicted value.[6]
Withholding periods of bronchodilators for PBD reversibility testing are given in Table 2:

Table 2: Withholding periods of bronchodilators for PBD reversibility testing.

SABA	≥ 4 hours
Formoterol, Salmeterol	24 hours
Indacaterol, Vilanterol	36 hours
Tiotropium, Umeclidinium, Aclidinium, Glycopyrronium	36 – 48 hours

Diagnosis of Asthma in Patients Already on ICS
If the basis of diagnosis of asthma is not documented previously and patient is already on treatment, diagnosis shall be confirmed. Plan is to step down ICS by 25 – 50% or stop other maintenance medications. If risk of exacerbation, step down treatment under close observation. Choose a suitable time to step down (not pregnant, not on holidays and away, no infection, etc.). Ensure a written asthma action plan is given and patient has enough medicines to be able to resume their previous dose if asthma symptoms worsen. Repeat assessment of asthma control and lung functions after 2 – 4 weeks.

If symptoms worsen or excessive airflow limitation:
- Confirm asthma diagnosis
- Patient to return to previous lowest effective treatment

If symptoms do not worsen nor there is excessive airflow limitation:
- Stop ICS
- Repeat asthma control assessment and lung function in 2 – 3 weeks
- Follow patient for at least 1 year[7]

References

1.Global Initiative for Asthma. Global strategy for asthma management and prevention [Internet]. 2025. Available from: https://ginasthma.org/2025-gina-strategy-report/

2.Aaron SD, Vandemheen KL, FitzGerald JM, Ainslie M, Gupta S, Lemière C, et al. Reevaluation of diagnosis in adults with physician-diagnosed asthma. JAMA. 2017;317(3):269–79.

3.Fagnano M, Bayer AL, Isensee CA, Hernandez T, Halterman JS. Nocturnal asthma symptoms and poor sleep quality among urban school children with asthma. Acad Pediatr. 2011 Nov;11(6):493-9.

4.Horner CC, Dula C, Bacharier LB, Garbutt JM, Gonzalez C, Deych E, et al. Daily global stress is associated with nocturnal asthma awakenings in school-age children. J Allergy Clin Immunol. 2016;138(4):1196-8.

5.National Heart, Lung, and Blood Institute. Guidelines for the diagnosis and management of asthma (EPR-3) [Internet]. Bethesda (MD): NHLBI; 2012. Available from: https://www.nhlbi.nih.gov/guidelines/asthma/asthgdln.htm

6.Stanojevic S, Kaminsky DA, Miller MR, Thompson B, Aliverti A, Barjaktarevic I, et al. ERS/ATS technical standard on interpretive strategies for routine lung function tests. Eur Respir J. 2022 Jan 1;59(1):2101499. doi: 10.1183/13993003.01499-2021.

7.Aaron SD, Vandemheen KL, FitzGerald JM, et al. Reevaluation of diagnosis in adults with physician-diagnosed asthma. JAMA. 2017;317(3):269-279.

Chapter 03:

Differential Diagnosis of Asthma

Not all that wheezes is asthma!
Differential diagnosis of asthma vary across age groups and can be differentiated on clues from history or examination (Table 1).

Table 1: Clues to differential diagnosis of asthma

Age	Differential Diagnosis	Clue to Diagnosis Other Than Asthma
ALL AGES	Tuberculosis	Fever, weight loss, cough, hemoptysis
	Pertussis	Prolonged paroxysms of coughing, stridor
6 –11 YEARS	Chronic upper airway cough syndrome	Sneezing, itching, blocked nose, throat-clearing
	Inhaled foreign body	Sudden onset symptoms, unilateral wheeze
	Bronchiectasis	Productive cough, recurrent infections
	Primary ciliary dyskinesia	Productive cough, recurrent infections, sinusitis
	Congenital heart disease	Heart murmurs
	Bronchopulmonary dysplasia	Preterm delivery, symptoms since birth
	Cystic fibrosis	Excessive cough, mucus, gastrointestinal symptoms
12 –39 YEARS	Chronic upper airway cough syndrome	Sneezing, itching, blocked nose, throat-clearing
	Inducible laryngeal obstruction	Dyspnea, stridor
	Hyperventilation (dysfunctional breathing)	Dizziness, paresthesia, sighing
	Bronchiectasis	Productive cough, recurrent infections.
	Cystic fibrosis	Excessive cough, mucus, gastrointestinal symptoms
	Heart disease	Heart murmurs
	Alpha1-antitrypsin deficiency	Dyspnea, family history of early emphysema
	Inhaled foreign body	Sudden onset symptoms, unilateral wheeze
40 + YEARS	Inducible laryngeal obstruction	Dyspnea, stridor
	Hyperventilation (dysfunctional breathing)	Dizziness, paresthesia, sighing
	COPD	Dyspnea on exertion, cough, sputum, smoking
	Bronchiectasis	Productive cough, recurrent infections
	Heart failure	Dyspnea on exertion, orthopnea, ankle edema
	Medication related	ACE inhibitors
	Parenchymal lung disease	Dyspnea on exertion, dry cough, clubbing
	Pulmonary embolism	Sudden onset dyspnea and chest pain
	Central airway obstruction	Dyspnea, unresponsive to bronchodilators

Asthma phenotypes & endo-types

Phenotypes

Recognizable clusters of demographic features, clinical characteristics, lung function and inflammation are called asthma phenotypes.1-6 Some of the most common phenotypes are mentioned below:

1. Allergic asthma
    a. Starts in childhood
    b. Prior history of allergic diseases
    c. Family history of allergic diseases
    d. Sputum eosinophilia
    e. Responds well to ICS

2. Non-allergic asthma
    a. Sputum may be neutrophilic, eosinophilic or paucigranulocytic
    b. Lesser short-term response to ICS

3. Cough variant asthma and cough predominant asthma
    a. Only symptom is cough
    b. Airflow limitation absent except on bronchial provocation test
    c. ICS are effective

4. Adult-onset (Late-onset) asthma
    a. Usually females
    b. Non-allergic
    c. Requires higher doses of ICS, or refractory to corticosteroids

5. Asthma with persistent airflow limitation
    a. Airway wall remodeling in longstanding asthma

6. Asthma with obesity
    a. Little eosinophilic inflammation

Endo-types

An endo-type is defined as a specific biological pathway that explains the observable properties of a phenotype. There are two recognized endo-types of asthma:

1. T2 high asthma[7]
    a. Sputum eosinophilia
    b. Blood eosinophilia
    c. High FeNO
    d. High serum IgE
    e. Mostly allergic
    f. May include late-onset eosinophilic asthma
    g. Responds well to ICS and biologic agents

2. T2 low asthma (Non-type 2 asthma)[7]
    a. Lacks markers of type 2 inflammation
    b. Difficult to treat
    c. Linked to obesity and neutrophilic inflammation

References

1.Bel EH. Clinical phenotypes of asthma. Curr Opin Pulm Med. 2004;10(1):44-50.
2.Moore WC, Meyers DA, Wenzel SE, et al. Identification of asthma phenotypes using cluster analysis in the Severe Asthma Research Program. Am J Respir Crit Care Med. 2010;181(4):315-23.
3.Wenzel SE. Asthma phenotypes: the evolution from clinical to molecular approaches. Nat Med. 2012;18(5):716-25.
4.Zhou J, Yi F, Wu F, et al. Characteristics of different asthma phenotypes associated with cough: a prospective, multicenter survey in China. Respir Res. 2022;23:243.
5.Lai K, Zhan W, Wu F, et al. Clinical and inflammatory characteristics of the Chinese APAC Cough Variant Asthma Cohort. Front Med (Lausanne). 2021;8:807385.
6.Scott HA, Ng SH, McLoughlin RF, et al. Effect of obesity on airway and systemic inflammation in adults with asthma: a systematic review and meta-analysis. Thorax. 2023;78(10):957-65.
7.Fahy J. Type 2 inflammation in asthma — present in most, absent in many. Nat Rev Immunol. 2015;15(1):57-65.

Chapter 04:
Asthma Severity

Asthma severity reflects the underlying intensity of the disease that how difficult it is to treat patient's asthma.

Mild asthma

- Defined by minimal symptoms and minimal risk of exacerbations in patients who are not using inhaled therapies, who are using reliever therapy alone, or who are using low-dose inhaled glucocorticoids along with reliever therapy.[1]
- The term "mild asthma" should not be used to avoid giving the impression that mild symptoms equates with low risk.[2]

Moderate asthma

- Defined by good asthma control with medium-dose inhaled glucocorticoids or low-medium-dose inhaled glucocorticoids with additional controller therapies.

Severe asthma

- Defined by asthma requiring high-dose inhaled glucocorticoids with additional controller agents to maintain good control or uncontrolled asthma despite these therapies.[1,3,4]

By these retrospective definitions, asthma severity can only be assessed after achieving good control and stepping down therapy to find the minimum effective controller therapy (or unless asthma remains uncontrolled despite maximized therapy). Severity should only be assessed after several months of controller treatment when asthma is stable.

References

1. Global Initiative for Asthma. Global strategy for asthma management and prevention, 2025. Available from: https://ginasthma.org/2025-gina-strategy-report/
2. Mohan A, Lugogo NL, Hanania NA, Reddel HK, Akuthota P, O'Byrne PM, et al. Questions in mild asthma: an official American Thoracic Society research statement. Am J Respir Crit Care Med. 2023 Jun 1;207(11):e77-e96.
3. Chung KF, Wenzel SE, Brozek JL, Bush A, Castro M, Sterk PJ, et al. International ERS/ATS guidelines on definition, evaluation and treatment of severe asthma. Eur Respir J. 2014 Feb;43(2):343-73.
4. Holguin F, Cardet JC, Chung KF, Diver S, Ferreira DS, Fitzpatrick A, et al. Management of severe asthma: a European Respiratory Society/American Thoracic Society guideline. Eur Respir J. 2020 Jan;55(1):1900588.

Chapter 05:
Asthma Control

Assessing asthma control at every visit is essential to determine both:

- Current symptom burden
- Future risk of exacerbations and lung function decline

Current Symptom Burden

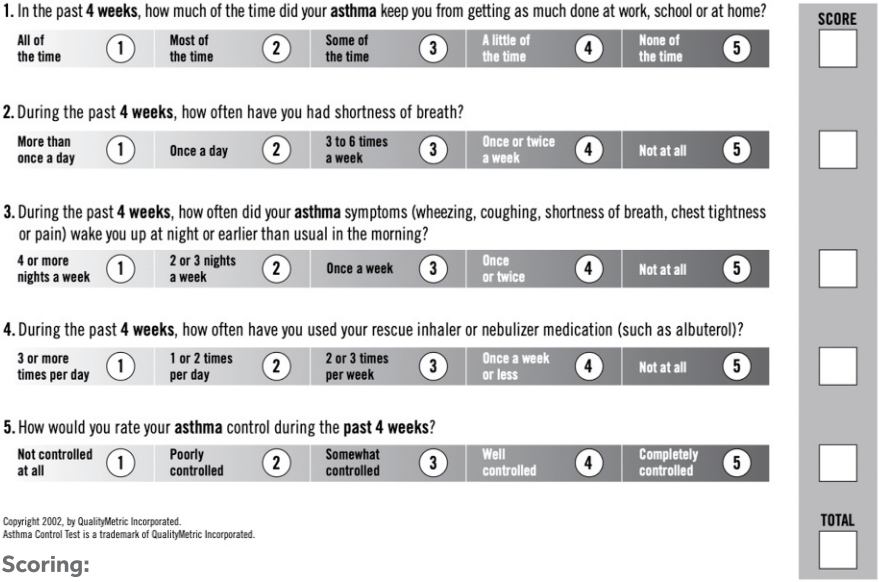
Use of both clinical judgement and validated tools is used to assess asthma control:

- Asthma Control Test (ACT)
- Asthma Control Questionnaire (ACQ)
- GINA Symptom Control Assessment (most commonly used in clinical settings)

Asthma Control Test

It has four symptom and reliever questions plus patient's self-assessed symptom control. It has not been validated for ICS-formoterol or ICS-SABA reliever. Score ranges from 5 – 25, the higher the better.

Figure 1: Asthma control test



Scoring:

20 – 25: Well controlled
16 – 19: Not well controlled
5 – 15: Very poorly controlled

Asthma control questionnaire

It asks the patient to reflect on the past 7 days regarding:

1. Awoken at night by symptoms.
2. Severity of symptoms upon waking in the morning.
3. Limitation of activities due to asthma.
4. Shortness of breath experienced.
5. Wheezing frequency.

Each question is scored on a scale of 0 (totally controlled) to 6 (severely poorly controlled). The final score is the average of all responses.
Score 0.0 – 0.75: Well-controlled asthma.
0.75 – 1.5: Control is adequate but could be improved.
> 1.5: Poorly controlled asthma. This usually triggers a change in medication or lifestyle.

GINA symptom control tool – past 4 weeks
Ask the patient the following four questions:

ASTHMA SYMPTOM CONTROL QUESTIONS (past 4 weeks)	WELL-CONTROLLED	PARTLY CONTROLLED	UNCONTROLLED
Daytime symptoms more than twice/week?	None of these	1–2 of these	3–4 of these
Any night-time waking due to asthma?			
Need for reliever more than twice/week?			
Activity limitation due to asthma?			

Action Based on Control Level

Level	Next Steps
Well-Controlled	- Maintain current therapy- Consider step-down if control stable for ≥ 3 months
Partly Controlled	- Check adherence, inhaler use, and triggers- Consider stepping up treatment
Uncontrolled	- Reassess urgently- Consider step-up in therapy- Refer if needed

Future Risk of Exacerbations and Lung Function Decline
Every patient with asthma should be assessed for future risk, in addition to symptom control. This includes the risk of:

- Severe exacerbations (Table 1)[1,2]
- Persistent airflow limitation (Table 2)[3,4]
- Adverse effects of treatment (Table 3)

This proactive approach allows for tailored intervention to prevent long-term complications.

Table 1: Risk factors for severe exacerbations

Category	Risk Factors
Medication Use	High SABA over-use = 3 x 200-dose canisters/year; = Icanister/month increases mortality risk).
ICS Issues	Inadequate ICS (not prescribed, poor adherence, or incorrect technique).
Medical Conditions	Obesity, chronic rhinosinusitis, GERD, confirmed food allergy, and pregnancy.
Exposures	Smoking, e-cigarettes, allergen exposure (if sensitized), and air pollution.
Psychosocial	Major psychological or socioeconomic problems.
Lung Function	Low FEV1 (especially <60% predicted) and high bronchodilator responsiveness.
Inflammation	Type 2 inflammatory markers: raised blood eosinophils or high FeNO.
History	Ever intubated/ICU for asthma or = 1 severe exacerbation in the last year.

Table 2: Risk factors for persistent airflow limitation.

Category	Risk Factors
Early History	Preterm birth, low birth weight, and greater infant weight gain, frequent productive cough
Medications	Lack of ICS treatment in patients with a history of severe exacerbation.
Exposures	Tobacco smoke, noxious chemicals, and occupational or domestic exposures.
Investigations	Low initial FEV1, sputum eosinophilia, or blood eosinophilia.

Table 3: Risk factors for medication side- effects

Type	Risk Factors
Systemic	Frequent OCS, long-term high-dose/potent ICS, and use of cytochrome P450 inhibitors.
Local	High-dose or potent ICS and poor inhaler technique.

References

1. Global Initiative for Asthma. Risk factors for exacerbations, persistent airflow limitation, and medication side-effects. 2023.
2. Juniper EF, Svensson K, Mork AC, Stahl E. Measurement properties and interpretation of three shortened versions of the asthma control questionnaire. Respir Med. 2005;99(5):553-8.
3. Nathan RA, Sorkness CA, Kosinski M, Schatz M, Li JT, Marcus P, et al. Development of the asthma control test: a survey for assessing asthma control. J Allergy Clin Immunol. 2004;113(1):59-65.
4. Schatz M, Kosinski M, Yarlas AS, Hanlon J, Blaiss M, Zeiger RS. The minimally important difference of the Asthma Control Test. J Allergy Clin Immunol. 2009;124(4):719-23.e1.
5. Foster JM, Aucott L, van der Werf RH, van der Molen T, Postma DS. Higher patient perceived side effects related to higher daily doses of inhaled corticosteroids in the community: a cross-sectional analysis. Respir Med. 2006;100(8):1318-1336.
6. Roland NJ, Bhalla RK, Earis J. The local side effects of inhaled corticosteroids: current understanding and review of the literature. Chest. 2004;126(1):213-219.
7. Schatz M, Kosinski M, Yarlas AS, Hanlon J, Blaiss M, Zeiger RS. The minimally important difference of the Asthma Control Test. J Allergy Clin Immunol. 2009;124(4):719-723.e1.

Chapter 06:

Management of Asthma

For the development of national guidelines, an extensive review of the existing literature (PubMed, EmBase, and Cochrane) and the major international guidelines (Global Initiative for Asthma, British Thoracic Society, NICE, SIGN and National Asthma Education and Prevention Program of the National Heart, Lung, and Blood Institute) were reviewed.[1-3] Due to scarcity of available local data, we adapt recommendations from internationally available resources.

Goals of Asthma Management

Figure 1: Goals of asthma management

Non-Pharmacological Management

Non-pharmacological strategies play an indispensable role in the holistic management of asthma, complementing pharmacological treatment to achieve better control and reduce exacerbations. These measures should be integrated alongside pharmacotherapy and tailored to each patient's context to achieve optimal control and empower self-management. Following image shows non-pharmacological interventions that can help reduce asthma symptoms and exacerbations.

- Cessation of tobacco exposure, vaping
- Encourage physical activity
- Structured pulmonary rehabilitation program
- Avoid allergen exposure (indoor, outdoor, occupational)
- Avoid medicine triggers
- Encourage healthy diet
- Weight reduction
- Breathing exercises
- Avoid air pollution
- Deal stress

Figure 2: Non-pharmacological interventions

Advise vaccination against influenza, pneumococcus, pertussis, RSV, Covid.

Pharmacological management

Recommendations for pharmacologic therapy of asthma are based on a stepwise approach. This includes shared decision-making to attain and sustain control of asthma at the lowest effective therapeutic regimen. Before embarking on to the details of management, a list of nomenclature used is given below:

Reliever

- Inhaler taken as needed, for quick relief of symptoms
- Also called "rescue inhaler"
- Can be used before exercise to prevent EIA
- Example: SABA, ICS-SABA, ICS-formoterol

Controller

- Controls symptoms and future risk

Maintenance treatment

- Prescribed for everyday use, on a regularly scheduled basis
- Intended to use continuously even when no asthma symptoms
- Example: ICS, ICS-LABA, ICS-LABA-LAMA, LTRA, biologic therapy

Anti-inflammatory reliever (AIR)

- Inhaler that contains low dose ICS and rapid-acting bronchodilator
- Example: ICS-salbutamol, budesonide-formoterol, beclometasone-formoterol
- ICS-formoterol shall never be used as reliever with maintenance ICS-non formetrol LABA
- Can be used before exercise or allergen exposure to prevent bronchoconstriction

Maintenance-and-reliever therapy (MART)

- ICS-formoterol inhaler being used as maintenance as well as reliever
- SMART is synonym: Single-inhaler maintenance-and-reliever therapy
- ICS-non formoterol and ICS-SABA can not be used as MART

Table 1: Asthma management nomenclature

Treatment for patients newly diagnosed with asthma

Managing asthma is a three-phase process, as shown in image below: Adapted from Global Initiative for Asthma. Global Strategy for Asthma Management and Prevention, 2025 update. Fontana (WI): Global Initiative for Asthma; 2025 May.

Figure 3: Plan of asthma management

For patient presenting for the first time, who is not on any asthma medicines, the choice of initial drug depends on the severity of symptoms. SABA free pathways are recommended to reduce the risks associated with SABA overuse. These include AIR and MART.

First presentation with exacerbation: Treat a s exacerbation, add OCS short-course if severe. Start medium-dose MART

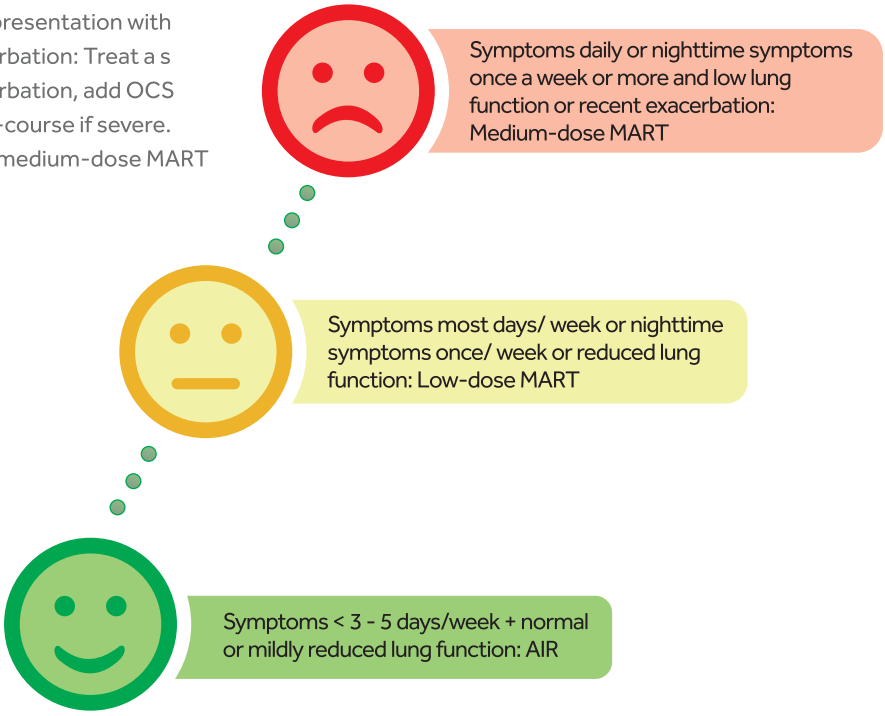


Figure 4: Initiating asthma treatment

Alternate pathways include as needed ICS+SABA (or low dose ICS + as needed SABA or ICS-SABA); low dose ICS-LABA + as needed SABA or ICS-SABA; and medium dose ICS-LABA + as needed SABA or ICS-SABA for increasing level of symptoms severity respectively.

Management And Treatment Of People With An Existing Diagnosis Of Asthma
For patients already on asthma treatment, again follow the same cycle:

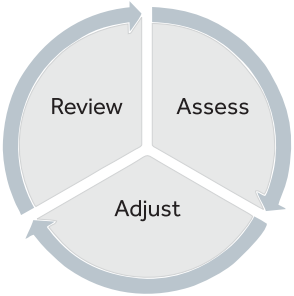


Figure 5: Asthma management plan
SABA free pathway is recommended to reduce the risks associated with SABA overuse

The MART pathway

Traditionally management of asthma is done in a step-wise approach. At each step assess symptom control and may step up if disease is uncontrolled. We recommend the pathway centered on the use of low-dose ICS-formoterol as both the reliever and, in moderate to severe cases, as the daily maintenance therapy—a strategy known as Maintenance and Reliever Therapy (MART).

MART strategy involves using a single inhaler containing a combination of inhaled corticosteroid and formoterol, a fast-acting and long-acting beta agonist for both daily maintenance and as-needed symptom relief. So, MART simplifies therapy by combining both roles (controller and reliever) into one inhaler. MART reduces hospitalizations and asthma deaths. MART reduces over-reliance on SABA, reducing exacerbations and also reduce the overall dose of steroid for majority of asthma cases. One of the benefits is that using ICS and formoterol together works synergistically.

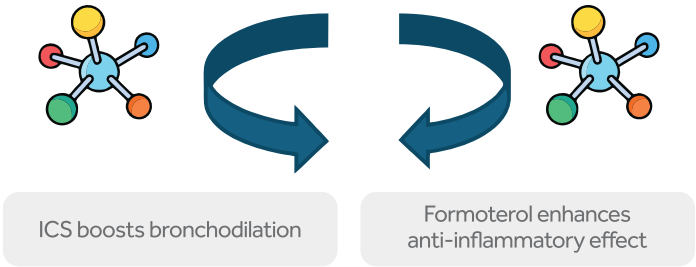


Figure 6: Role of ICS-formoterol

An added advantage of MART for the environment is that combining both drugs in a single inhaler halves the CO_2 emissions. So, it is greener.

MART is a more patient-centered approach. Using a single inhaler for both symptom relief and asthma control increases compliance and patient only needs to learn one technique.

For patients with mild intermittent or mild persistent asthma (Step 1), as-needed ICS-formoterol is advised. This as-needed use of ICS-formoterol has been shown to significantly reduce the risk of severe asthma exacerbations compared to using SABA alone.

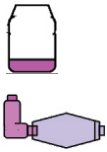
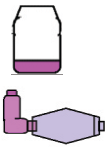
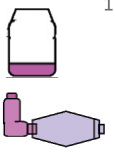
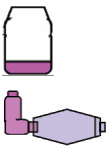
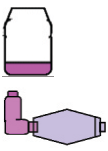
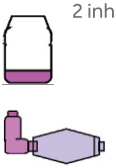
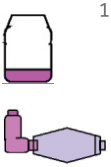
In moderate asthma (step 2), low-dose maintenance ICS-formoterol (MART) is recommended; reliever is the same inhaler on as-needed basis. Hence there is added flexibility to treat worsening symptoms using the same inhaler.

In step 3, moderate to severe asthma, MART continues, now with medium-dose maintenance ICS-formoterol. Here reliever is again the same inhaler on as-needed basis. So, a simplified one inhaler strategy is maintained.

In step 4, severe asthma, high dose maintenance ICS-formoterol may be considered. Add-on LAMA may be used. Also check phenotype. Patient shall be referred to specialist asthma care, may benefit from biologics, like anti-IgE, anti-IL5/5R, anti-IL4Rα, anti-TSLP. Reliever medicine is the same as-needed ICS-formoterol.
MART represents a major shift in asthma management, moving away from SABA, and towards a more integrated, preventative approach.

Following table represents the recommendations:

Table 2: Stepwise approach to asthma management in adults (MART pathway)

Step	Treatment			Reliever
1-2	AIR only Low dose ICS-formoterol	-----	-----	1 inhalation PRN 
3	MART Low-dose maintenance ICS-formoterol	1 inhalation BD 	-----	1 inhalation PRN 
4	MART Medium-dose maintenance ICS-formoterol	2 inhalations BD 	-----	1 inhalation PRN 
5	Add-on LAMA, Phenotype assessment, trial of high-dose maintenance ICS-formoterol, anti-IgE, anti-IL5/5R, anti-IL4Rα, anti-TSLP	2 inhalations BD 		1 inhalation PRN 

AIR: Budesonide-formoterol DPI 200/6 mcg OR Beclometasone dipropionate (BDP)-formoterol 100/6 mcg

MART: Budesonide-formoterol DPI 200/6 mcg, Beclometasone dipropionate (BDP)-formoterol 100/6 mcg

Only above 2 doses are recommended for AIR and MART. Maximum total 12 inhalations/ day are recommended for both budesonide-formoterol and BDP-formoterol, whichever is used. Maximum total includes maintenance as well as reliever doses. Use of spacer is recommended for ICS-formoterol pMDI.

Most research is done on AIR with budesonide-formoterol DPI 200/6 mcg metered dose. Budesonide formoterol 400/12 mcg and 100/6 mcg shall not be used as AIR.
Beclometasone dipropionate (BDP)-formoterol 100/6 mcg is not studied for use as AIR, but it is postulated that it may be effective as it is effective for MART in adults. Other ICS-formoterol combinations are not studied.

The traditional – alternate pathway

Alternate pathway includes SABA reliever (as-needed ICS-SABA or as-needed SABA). Before considering this regimen, check whether the patient will compliantly use controller treatment. This regimen may also be continued for patients who are already on this regimen and have controlled disease.

This pathway relies on the previously used traditional regimen: a daily controller inhaler (such as ICS or ICS combined with a long-acting beta-agonist, or LABA) used regularly, with a short-acting beta-agonist (SABA) used as-needed for symptom relief. It may be used for patients who cannot access ICS-formoterol, do not tolerate it, or are already highly compliant to daily controller use.

One of the key concerns with this pathway is that patients often rely too much on SABA relievers, leading to poor asthma control and increased risk of exacerbations and hospitalizations. Patients following this traditional management pathway shall receive proper education on asthma self-management, the risks of SABA overuse, and the importance of compliance to controller inhaler. This pathway is less preferred due to its higher risk profile and complexity compared to the simplified, more protective MART-based treatment strategies.

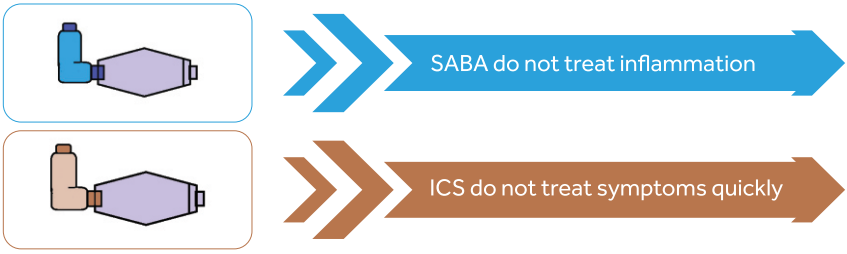


Figure 7: Role of SABA and ICS

In step 1: where there are mild, infrequent symptoms, it is recommended to use as needed low dose ICS whenever SABA is used. This ensures patients get anti-inflammatory medicine without daily treatment, helping reduce exacerbation risk.

In step 2, i.e. mild persistent asthma daily low-dose ICS is given as controller and reliever remains as needed SABA alone or as needed ICS-SABA.

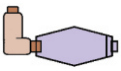
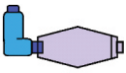
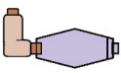
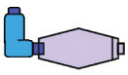
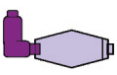
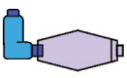
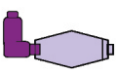
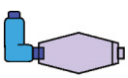
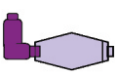
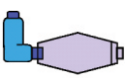
In step 3, moderate asthma where patient is more symptomatic and have nighttime awakenings, low-dose ICS-LABA maintenance is advised. The reliever remains the same as in step 2.

In step 4, moderate to severe asthma, medium dose ICS-LABA maintenance is advised. Reliever being the same as in other steps.

In step 5, severe or difficult to treat asthma, high dose ICS-LABA may be considered. Add-on LAMA may be used. Also check phenotype. Patient shall be referred to specialist asthma care, may benefit from biologics, like anti-IgE, anti-IL5/5R, anti-IL4Rα, anti-TSLP. Reliever medicine is the same as-needed SABA or as-needed SABA-ICS.

Following table represents the traditional alternate pathway for asthma management.

Table 3: Stepwise approach to asthma management in adults (Alternate pathway)

Step	Treatment			Reliever
1	Reliever only (As-needed SABA or as-needed SABA-ICS)		-----	
2	Low-dose maintenance ICS		-----	
3	Low-dose maintenance ICS-LABA		-----	
4	Medium-dose maintenance ICS-LABA		-----	
5	Add-on LAMA, Phenotype assessment, trial of high-dose maintenance ICS-LABA, anti-IgE, anti-IL5/5R, anti-IL4Rα, anti-TSLP			

Whichever pathway is used, if patient remains uncontrolled on medium-dose maintenance treatment, also consult guideline section on difficult-to-treat and severe asthma.

Commonly available inhaled corticosteroids
Adapted from Global Initiative for Asthma. Global Strategy for Asthma Management and Prevention, 2025 update. Fontana (WI): Global Initiative for Asthma; 2025 May.

Table 4: Doses of inhaled corticosteroids

Inhaled corticosteroid	Total daily ICS dose (mcg)		
	Low	Medium	High
Beclometasone dipropionate (pMDI, standard particle, HFA)	200 – 500	>500 – 1000	>1000
Beclometasone dipropionate (pMDI or DPI, extra fine particle, HFA)	100 – 200	>200 – 400	>400
Budesonide (pMDI or DPI, standard particle, HFA)	200 – 400	>400 – 800	>800
Ciclesonide (pMDI, extrafine particle, HFA)	80 – 160	>160 – 320	>320
Fluticasone furoate (DPI)	100		200
Fluticasone propionate (DPI)	100 – 250	>250 – 500	>500
Fluticasone propionate (pMDI, standard particle, HFA)	100 – 250	>250 – 500	>500
Mometasone furoate (DPI)	Depends on DPI device		
Mometasone furoate (pMDI, standard particle, HFA)	200 – 400		> 400

Above table doesn't reflect potency equivalence. For example, if patient is switched from medium dose of one ICS to medium dose of other ICS, this may represent an increase or decrease in potency.

Switching to SABA-free regimen

Identify adults and children 12+ who can be switched to SABA free treatment, particularly where asthma is not controlled. Initiate a discussion with the patient about switching their treatment regimen, particularly if they remain symptomatic. If they are not symptomatic and are happy on their current treatment pathway, it is not recommended that they are transferred to SABA free treatment. Following table gives some practical points on how to switch treatment to SABA free pathway. (Adapted from Joint BTS/NICE/SIGN Guideline Ng245).

Table 5: Medicine options for switching to SABA-free regimen

Current treatment	Switch
SABA only	Low-dose ICS/formoterol PRN (AIR)
Regular low-dose ICS + SABA PRN	Low-dose MART
Regular low-dose ICS/LABA + SABA PRN	
Regular low-dose ICS + LTRA and/or LAMA + SABA PRN	
Regular low-dose ICS/LABA + LTRA and/or LAMA + SABA PRN	
Regular moderate-dose ICS + SABA PRN	Moderate-dose MART
Regular moderate-dose ICS/LABA + SABA PRN	
Regular moderate-dose ICS + LTRA and/or LAMA + SABA PRN	
Regular moderate-dose ICS/LABA + LTRA and/or LAMA + SABA PRN	
High dose ICS containing regimen	Refer to specialist asthma care

Review Response And Adjust Treatment
Assess asthma control, treatment compliance and inhaler technique on each visit. Ideally, patient shall be reviewed 1 – 3 months after starting treatment, and every 3 – 12 months thereafter. After exacerbation, review after 1 week.

Step Up Asthma Treatment
Asthma treatment may need adjustment by the clinician or the patient from time to time. A written asthma action plan shall be available to every patient so that patient may also step up his / her treatment. In day to day adjustments, patient may adjust the number of as needed doses of ICS-formoterol from day to day according to their symptoms. Short-term step up may be needed during viral infections, seasonal allergen exposures etc. Here a short-term increase in maintenance ICS dose is done for 1 – 2 weeks. This may be initiated by the patient according to asthma action plan, or the treating clinician. Sustained step-up is to be done by the treating clinician. This is increasing to higher step in asthma treatment pathway (example switch from step 2 to step 3). Any step up is to be regarded is trial and if no response after 2 – 3 months, revert to previous step, and alternative treatment or referral are to be considered.

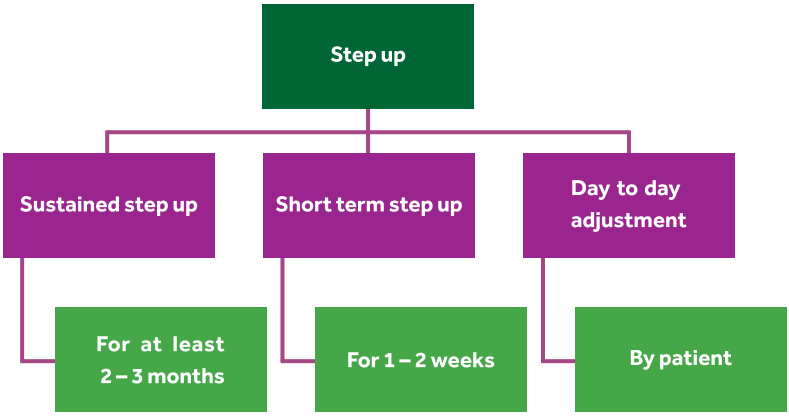


Figure 8: Step up asthma treatment

Step Down Asthma Treatment
Step down of asthma treatment can be done when a good control of the disease is achieved and lung functions have reached a plateau. Step down is to be done when disease control is maintained for 2 – 3 months. Stepping down minimize the cost of treatment and reduce risk of potential side-effects. Before stepping down, provide written asthma action plan to the patient, and educate on how and when to restart their previous treatment if symptoms worsen.

Patient Education
Effective asthma care is more than just prescribing medications—it requires empowering patients with the knowledge and confidence to manage their condition daily. Asthma is a chronic respiratory disease that demands a comprehensive, personalized approach to management. As healthcare providers, it is crucial to address common myths and misconceptions that can interfere with treatment, teach and reinforce correct inhaler techniques, and guide patients in developing and using a written asthma action plan. Preventive education is also key, helping patients identify and avoid triggers to reduce the

risk of exacerbations. This section provides guidance to support clinicians in delivering clear, consistent, and actionable asthma education to patients and their caregivers.

Asthma Myths

Asthma is a common yet often misunderstood condition that affects millions of people round the globe. Despite widespread awareness, numerous myths and misconceptions about asthma persist, leading to confusion, stigma, and sometimes even harmful decisions about treatment and management. This section aims to clarify the facts, dispel common myths, and provide information to help healthcare providers so that they can educate patients confidently so that patients can make informed choices and live healthier, more empowered lives.

Table 6: Common asthma myths.

MYTHS	TRUTHS
Allergies are not related to asthma.	Most people with asthma have allergies. These may trigger cough, dyspnea, wheezing.
People with asthma shall not exercise.	Exercise is good for lung health. People with asthma may exercise as long as asthma is controlled. Some people have exercise induced asthma, they may be prescribed inhalers to be used before exercise.
Asthmatics cannot participate in sports.	As long as asthma is controlled, there is no restriction to playing.
No wheeze means no asthma.	Asthma symptoms differ for everyone. As all wheeze is not asthma, likewise if there is no wheeze it doesn't mean there is no asthma.
People with asthma 'have symptoms' routinely.	Most people on optimal treatment can live a normal, healthy life.
Only poor outdoor air quality is the trigger for asthma.	Outdoor as well as indoor air quality are important factors in preventing asthma worsening.
Vaccination triggers asthma.	Vaccinations do not trigger asthma, rather they may prevent infections that are common triggers for asthma exacerbations.

People with asthma shall remove pets from home.	Although ideal is to keep away from all known allergens. However, it may not be possible for some to remove their pets. There are ways to minimize allergen exposures like creating pet free zones (keeping them out of bedrooms, etc.), vacuum and dust regularly using a vacuum cleaner with a HEPA filter, bathe and brush pets frequently (ideally someone without asthma shall do this), HEPA air purifiers can be used at home, wash hands after playing with pets, keep pets off furniture.
Asthma is curable.	Asthma is not curable, but is treatable.
People with mild asthma cannot die of it.	Any severity of asthma can have exacerbation that may be mild to severe.
People with asthma have weak lungs.	Lungs in asthma are not weak. They react too strongly to allergens.
Asthma medicines are only used when having symptoms.	Asthma medicines shall be taken even when asymptomatic, as they control the disease and prevent exacerbations.
Asthma can be managed with salbutamol inhaler alone.	Asthma is a disease of inflammation rather than bronchoconstriction alone. Salbutamol inhaler doesn't treat inflammation.
Spacers are only for use in children.	Spacers are important for all age groups, as they help medicine reach the lungs where it is needed rather than deposition in mouth and throat.
Inhaled steroids are addictive.	Inhaled steroids are safe and not habit forming.
Inhaled steroids are harmful.	Inhalers deliver a very small amount of steroid to have any harmful effect.
Nebulizers are better than inhalers.	Inhalers are easier to use and are as efficient as nebulizers are. Many controller medicines cannot be given via nebulizers.

Using inhalers stunt children growth.	Inhalers do not have any effect on growth of children.
Inhalers are the last resort in treating asthma.	Inhalers are the first resort in treating asthma.
Inhalers can be stopped after a certain time when asymptomatic.	Inhalers shall not be stopped even when asymptomatic, as they are preventive medicines against asthma exacerbations and can be taken lifelong.

Choosing Inhaler

Different types of inhalers are available on the market. Major classes of inhalers are given below:

Table 7: Types of inhalers

Pressurized metered dose inhaler (pMDI)	Slow and steady inhalation over 3-5 seconds Coordination required Manual dexterity
Dry powder inhaler (DPI)	Quick and deep inhalation over 2-3 seconds
Breath actuated pMDI	No coordination required between actuation and inhalation
Soft mist inhaler (SMI)	Slow, gentle inhalation Aerosol released over 1.5 second Requires coordination between actuation and inhalation

Choice of inhaler is based on:

1. Patient's ability to use the device, inhalation power, coordination etc.
2. Patient's preference
3. Age of patient
4. Patient's physical ability, like rheumatoid etc.
5. Dose – counter if needed
6. The cost

Best inhaler is the one the patient can use effectively and is willing to use
Spacer device shall always be used when using pMDI. They improve effectiveness of pMDI. Spacers reduce the deposition in the mouth and oropharynx during inhalation and hence reduce side effects like candidiasis, dysphonia, etc.

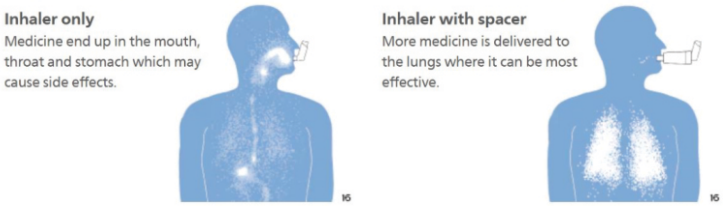


Figure 9: Benefits of using inhaler

Above picture taken from: https://www.usa.philips.com/c-e/hs/respiratory-care/what-is-asthma-spacer.html Adapted from: Hirst, PH., et al. Deposition and pharmacokinetics of an HFA formulation of Triamcinolone Acetonide delivered by pressurized metered dose inhaler. Journal of Aerosol Medicine. 2001; Volume 14 (2):155-166.

The In-Check Dial (shown in picture below) is a portable device used to measure a patient's inspiratory flow rate and assess which inhaler device will be better for patient and whether they are using their inhaler correctly. It simulates the resistance of different types of inhalers allowing healthcare providers to determine if the patient can generate the appropriate inhalation force required for their specific inhaler. This is important as each inhaler device requires a different inhalation technique and force, as already specified (DPIs need a strong, fast breath, while pMDIs require a slow, steady breath). The In-Check Dial helps tailor inhaler selection to the patient's ability. It also helps in training patient on how to use inhaler.

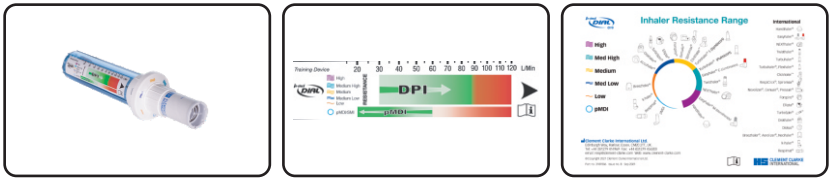


Figure 10: In-check dial

Inhaler technique
Correct inhaler technique is a keystone of effective asthma management. Despite the well-known use of inhalers, many patients use them sub-optimally. This significantly reduces the delivery of medication to the lungs and hence compromising symptom control. Educating patients on proper inhaler use—including device-specific instructions, breathing technique, and timing—is essential to ensure optimal treatment outcomes. Healthcare providers must demonstrate correct inhaler technique regularly, assess patient use at every visit, and correct errors through hands-on training and return demonstrations.
Steps of inhaler technique are explained below:
pMDI with spacer (single breath hold):

1. Hold inhaler upright and take the cap off.
2. Check there's nothing inside the mouthpiece.

3. Shake it well.
4. If there is valve on the spacer, make sure the valve is facing upwards.
5. Put inhaler into the hole at the back of the spacer.
6. If spacer has a cap, take it off.
7. Sit or stand up straight and slightly tilt chin up.
8. Breathe out gently and slowly away from the inhaler.
9. Make a tight seal around the mouthpiece of the spacer with lips.
10. Press the canister on the inhaler once and breathe in slowly and steadily until lungs feel full.
11. Take the mouthpiece of the spacer out of mouth and with lips closed, hold breath for up to ten seconds or for as long as it is comfortable.
12. Breathe out gently away from the spacer.
13. If a second puff is to be taken, with the spacer away from mouth, wait 30 seconds and then repeat the steps.
14. After using, take the inhaler out of the spacer and replace the caps on both the inhaler and the spacer.
15. If the inhaler contains steroids, rinse mouth with water and spit it out to reduce any chance of side effects.

pMDI with Spacer (tidal breathing or multiple breath technique):
All the steps are same as of using pMDI with single breath hold except step 10, 11 (see above). Here, press the canister on the inhaler once to release the medicine and breathe in and out slowly and steadily into the spacer five times. All remaining steps 12 – 15 are also the same as above.

Technique of DPI
There are different sort of dry powder inhaler devices available on the market. Make sure you are comfortable in demonstrating the steps of that particular DPI device you are prescribing. Basic inhalation technique remains the same:
Slow and steady for MDI, fast and deep for DPI

Asthma Action Plan
A written asthma action plan is an essential tool for effective self-management and improved clinical outcomes. It provides patients with clear, step-by-step instructions on how to manage their asthma daily, recognize early signs of worsening symptoms, and respond appropriately during an asthma flare-up. It increases treatment compliance, reduces hospital visits, and empowers patients to take control of their asthma with confidence. Every patient shall be provided with a written, up-to-date action plan as part of routine care of asthma.

Asthma action plans are available in different languages. Use the language that is understandable to the patient and / or the caregiver. In Pakistan, an Urdu version is available.

Figure 11: Asthma action plan (Urdu)

We recommend the preferred pathway of treatment where ICS-formoterol is the reliever. Whatever reliever is used, patient must be fully aware of it. So that he/ she may use it in case of emergency (the red part of the picture above).

Asthma preventive education

Preventive education has an important role in reducing asthma exacerbations, emergency department visits, and long-term complications. Clinicians can empower asthmatics by teaching them how to identify and avoid common triggers, use medicines compliantly, and recognize early warning signs, to take active control of their disease. Preventive education should be ongoing and tailored to each patient's lifestyle, environment, and risk factors. Below are some practical tips in avoiding common triggers:

Table 8: Asthma triggers and their avoidance.

Dust mites	Use allergen-proof mattress and pillow covers Wash bedding regularly in hot water Remove carpets and heavy curtains
Pet dander	Create pet-free zone Bathe and groom pets regularly Use HEPA air filters at home

Plant pollen	Keep windows closed during high pollen seasons Shower and change clothes after outdoor exposure
Tobacco smoke	Do not allow smoking in the home or car Avoid secondhand smoke and areas where people smoke
Air pollution	Limit outdoor activity on high-pollution days Use air purifiers indoors
Strong odors or fumes	Avoid scented candles, sprays, or cleaning products with strong fumes Ensure good ventilation when using household chemicals
Respiratory infections	Annual flu vaccine COVID vaccine Wash hands frequently Avoid contact with sick individuals
Cold, dry air	Cover mouth and nose with a cloth in cold weather Use a humidifier in dry environment
Exercise	Some individuals have exercise induced asthma They may be prescribed medicines to be used before exercise to prevent asthma

Stress	Relaxation techniques, deep breathing Adequate sleep Seek psychiatry/ psychology input if needed for anxiety or depression, etc.

Evidence used

MART has been widely studied and endorsed by major guidelines for asthma treatment. Following **table 9** mentions some high-quality studies and reviews:[4-8]

Study	Population	Intervention	Comparator	Conclusion
SYGMA 1 (2018) RCT	Mild asthma	Budesonide-formoterol PRN	Terbutaline PRN or daily budesonide + terbutaline PRN	SMART more effective than SABA, non-inferior to daily ICS
SYGMA 2 (2018) RCT	Mild asthma	Budesonide-formoterol PRN	Budesonide maintenance + SABA PRN	Effective with less ICS exposure
PRACTICAL (2019) Open-label pragmatic RCT	Mild to moderate asthma, real-world primary care setting	Budesonide-formoterol PRN	Budesonide maintenance + SABA PRN	SMART superior for exacerbation reduction
Novel START (2019) Randomized, open-label trial	Mild asthma	Budesonide-formoterol PRN	SABA PRN or maintenance ICS + SABA	Supports SMART as first-line in mild asthma
Cochrane Review (2022)	Adults with moderate–severe asthma	SMART (various ICS-formoterol combos)	Fixed-dose ICS/LABA + SABA	Strong evidence for SMART over traditional fixed ICS/LABA

Following is a grouped summary of major studies favoring MART according to severity of asthma:[4-7,9-11]

Mild asthma
Table 10

Study	Design	Key Findings
SYGMA 1 (2018)	RCT	SMART reduced exacerbations vs SABA; similar control to maintenance ICS
SYGMA 2 (2018)	RCT	SMART non-inferior to maintenance ICS with 75% less steroid exposure
NOVEL START (2019)	Pragmatic RCT	SMART more effective than SABA, similar to daily ICS
PRACTICAL (2019)	Real-world RCT	SMART superior in reducing exacerbations compared to maintenance ICS + SABA

Moderate to severe asthma
Table 11

Study	Design	Key Findings
MANDALA (2022)	RCT	Albuterol–budesonide combo reduced severe exacerbations vs albuterol alone
SMILE (2013)	Real-world study	SMART improved control and reduced exacerbations vs fixed-dose ICS/LABA + SABA
BEST (2000)	RCT	Validated flexible dosing with budesonide–formoterol (early SMART concept)

Safety Concerns Regarding LABA/ICS

Safety concerns surrounding the use of long-acting beta-agonists in the management of asthma have considerably shaped clinical treatment strategies over the past two decades. Early reports suggested increased risks of asthma-related deaths with LABA monotherapy—most notably from the 2006 SMART trial—hence a black-box warning was issued by U.S FDA against using LABA monotherapy in asthma. In response, the safety of LABA/ICS was evaluated in large trials, including AUSTRI and VESTRI.[12,13] Black-box warning was later removed for combination inhalers having LABA/ICS, and paved the way for the widespread adoption of SMART as a preferred strategy in existing asthma care.

Table 12.

Study	Population	Intervention	Comparator	Conclusion
AUSTRI Trial (2016) RCT	Adults with persistent asthma	Fluticasone–salmeterol	Fluticasone alone	LABAs are safe when used with ICS
VESTRI Trial (2016) RCT	Children (ages 4–11) with asthma	Fluticasone–salmeterol	Fluticasone alone	ICS–LABA therapy is safe in children when used in combination

Asian Studies

While MART with ICS–formoterol has gained widespread acceptance internationally for asthma management, there is a notable absence of robust clinical data from Pakistan. Most of the current evidence stems from randomized controlled trials and real-world studies conducted in affluent countries or multi-center Asian cohorts. There remains a significant lack of high-quality data from Pakistan. The management strategy recommended in current guidelines is an extrapolation of conclusions drawn from large-scale studies done in western and other Asian countries.

Table 13

Study	Setting	Design	Population	Intervention	Comparator	Key Outcomes
SMARTASIA (2013)	Multi-country Asia (China, India, Thailand, Taiwan, Indonesia)	Real-world Phase IV	Adults with partially-controlled asthma	Budesonide–formoterol 160/4.5 µg, 2 puffs BID + PRN (same inhaler)	Budesonide–formoterol 2 puffs BID + salbutamol PRN (separate reliever)	ACQ-5 and AQLQ-S improved significantly in intervention group
COSMOS Asian sub-analysis	Asia RCT subgroup (China, Korea, Taiwan, Thailand)	Open-label RCT	= 16 years, moderate asthma	Adjustable dosing: Budesonide–formoterol 160/4.5 µg 1–4 puffs/day + PRN	Fixed-dose fluticasone–salmeterol 250/50µg BID + salbutamol PRN	38% reduced exacerbations in intervention group
Pediatric China (2020–21)	Real-world cohort and model	Retrospective + modeling	Children under 18	Budesonide–formoterol SMART (low-dose maintenance + PRN)	Traditional regimen: ICS maintenance + salbutamol PRN	MART has lower exacerbations and is cost-effective

Austri: Adult Utilization Safety and Tolerability of Respiratory Inhalers

Cosmos: Comparing Options Single Maintenance And reliever therapy versus Other Strategies

Novel START: Novel Symbicort Turbuhaler Asthma Reliever Therapy

Practical: Personalized Asthma Combination Therapy: Implementing Effective and Low-risk Treatments

Smartasia: Symbicort Maintenance and Reliever Therapy in Asia

SYGMA: Symbicort Given as Needed in Mild Asthma

VESTRI: Vilanterol Evaluation of Safety in Treating Respiratory Illness in Children

References

1.Global Initiative for Asthma. Global Strategy for Asthma Management and Prevention, 2025 update. Fontana (WI): Global Initiative for Asthma; 2025 May. Available from: www.ginasthma.org

2.British Thoracic Society; National Institute for Health and Care Excellence; Scottish Intercollegiate Guidelines Network. Asthma: diagnosis, monitoring and chronic asthma management (Joint BTS/NICE/SIGN Guideline NG245). London: NICE/BTS/SIGN; 2024 Nov 27. Available from: https://www.nice.org.uk/guidance/ng245

3.National Asthma Education and Prevention Program Coordinating Committee Expert Panel Working Group. 2020 Focused Updates to the Asthma Management Guidelines: A Report from the National Asthma Education and Prevention Program Coordinating Committee Expert Panel Working Group. J Allergy Clin Immunol. 2020;146(6):1217-1270.

4.O'Byrne PM, FitzGerald JM, Bateman ED, Barnes PJ, Zheng J, Gustafson P, et al. Inhaled combined budesonide–formoterol as needed in mild asthma. N Engl J Med. 2018 May 17;378(20):1865–76. Available from: https://doi.org/10.1056/NEJMoa1715274

5.Bateman ED, Reddel HK, O'Byrne PM, Barnes PJ, Zhong N, Bain PI, et al. As-needed budesonide–formoterol versus maintenance budesonide in mild asthma. N Engl J Med. 2018 May 17;378(20):1877–87. Available from: https://doi.org/10.1056/NEJMoa1800353

6.Hardy J, Baggott C, Fingleton J, Reddel HK, Harwood M, Shirtcliffe P, et al. Budesonide–formoterol reliever therapy versus maintenance budesonide plus terbutaline reliever therapy in adults with mild to moderate asthma (PRACTICAL): a 52-week, open-label, multicentre, superiority, randomised controlled trial. Lancet. 2019 May 18;393(10180):1754–64. Available from: https://doi.org/10.1016/S0140-6736(19)30772-X

7.Beasley R, Holliday M, Reddel HK, Braithwaite I, Ebmeier S, Hancox RJ, et al. Controlled trial of budesonide–formoterol as needed for mild asthma. N Engl J Med. 2019 May 23;380(21):2020–30. Available from: https://doi.org/10.1056/NEJMoa1901963

8.Kew KM, Dahri K, Stovold E. Single maintenance and reliever therapy versus fixed maintenance dosing plus reliever in asthma. Cochrane Database Syst Rev. 2022;(12):CD009019. Available from: https://doi.org/10.1002/14651858.CD009019.pub2

9.Papi A, Panettieri RA Jr, Bel EH, Beasley R, Chaudhuri R, Kannan H, et al. As-needed albuterol–budesonide versus albuterol in mild asthma (MANDALA). N Engl J Med. 2022 un 2;386(22):2051–61. Available from: https://doi.org/10.1056/NEJMoa2203163

10.Rabe KF, Pizzichini E, Ställberg B, Szefler SJ, Lalloo UG, Svensson K, et al. Effect of budesonide/formoterol maintenance and reliever therapy: The SMILE study. Respir Med. 2006 Apr;100(4):546–56. Available from: https://doi.org/10.1016/j.rmed.2005.05.004

11.Bateman ED, Boushey HA, Bousquet J, Chanez P, Enright PL, Gibson P, et al. Budesonide/formoterol in a single inhaler improves asthma control: The BEST study. Eur Respir J. 2000 Nov;15(5):904–14. Available from: https://doi.org/10.1183/09031936.00.15590400

12.Stempel DA, Bousquet J, Walsh JK, Walker M, Weinstein SF, Vestergaard P, et al. Serious asthma events with combination fluticasone–salmeterol therapy: AUSTRI trial in adolescents and adults. N Engl J Med. 2016 May 12;374(19):1887–96. Available from: https://doi.org/10.1056/NEJMoa1511049

13.Stempel DA, Szefler SJ, Pedersen S, Zeiger RS, Yeakey AM, Lee LA, et al. Safety of adding salmeterol to fluticasone propionate in children with asthma (VESTRI). N Engl J Med. 2016 Sep 1;375(9):840–9. Available from: https://doi.org/10.1056/NEJMoa1606356

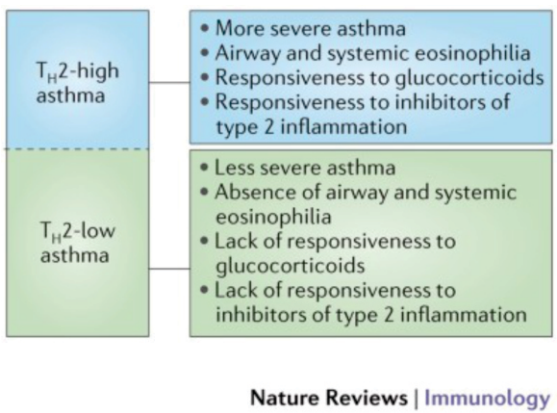
14.Zhong N, Lin J, Mehta P, Ngamjanyaporn P, Wu TC, Yunus F, et al. Real-life effectiveness of budesonide/formoterol maintenance and reliever therapy in asthma patients across Asia: SMARTASIA study. BMC Pulm Med. 2013 Apr 4;13:22. Available from: https://doi.org/10.1186/1471-2466-13-22
15.Vogelmeier C, Naya I, Ekelund J. Budesonide/formoterol maintenance and reliever therapy in Asian patients (aged ≥16 years) with asthma: a sub-analysis of the COSMOS study. Clin Drug Investig. 2012 Jul;32(7):427-35. Available from: https://doi.org/10.2165/11633230-000000000-00000
16.Yu Y, Cao W, Xiao Y, Li A, Huang H, Liu K, Hu L, Hou X, Xiang L, Wang X. Budesonide/formoterol maintenance and reliever therapy in childhood asthma: Real-world effectiveness and economic assessment. Pediatr Pulmonol. 2023 Dec;58(12):3406-3415. doi: 10.1002/ppul.26647.

Chapter 07:
Role of Biologics

Aadapted from GINA 2025.1 Mostly the biologics are not available in Pakistan.
Biologics are the cornerstone of severe asthma management. Uncontrolled asthma is an umbrella term and encompass one or both of the following:
1.Poor asthma control
2.Frequent exacerbations (≥2/year requiring OCS), or serious exacerbation (≥1/year requiring hospitalization)

Difficult to treat asthma is asthma that is uncontrolled despite on medium or high dose ICS with a second controller, or with maintenance dose OCS. Poor control is may be due to modifiable factors like incorrect inhaler technique, non-compliance, smoking or comorbidities, or incorrect diagnosis.
Severe asthma is a subset of difficult to treat asthma where asthma remains uncontrolled despite addressing modifiable factors.
This is discussed elsewhere in the document.
Before a biologic is initiated make an assessment of modifiers (confirm diagnosis, inhaler technique, compliance, manage comorbidities, optimize treatment).

Phenotyping
When diagnosis of severe asthma is made, the next step is to assess the type of inflammation. Disease pathology is caused by differing mechanisms in the different inflammatory phenotypes. Targeted therapy with monoclonal antibodies (mAbs) works by specifically blocking these disease pathways. Recognition and assessment of individual phenotypes is necessary to support a targeted therapy approach. Detailed assessment is required to inform the selection of targeted therapies which are likely to benefit individual patients.



Adopted from: Fahy, J. Type 2 inflammation in asthma — present in most, absent in many. Nat Rev Immunol 15, 57–65 (2015)

Figure 1: Characteristics of T2 high and T2 low asthma

Biomarkers
Selection of biologics depend on type of inflammation which is assessed by biomarkers (Table 1). Type 2 (T2) High inflammation is the target for most current biologics.

- Blood eosinophils
- FeNO
- Sputum eosinophils
- Positive skin prick or specific IgE for perennial allergens

Blood eosinophil count
Typical Criteria for "High" (Adults/Adolescents):
Diagnosis: BEC ≥ upper limit of normal for the population.
Severe Asthma (on high-dose ICS): BEC ≥ 150/µL suggests the presence of Type 2 inflammation. BEC ≥ 300$/µL is a common threshold for eligibility for Type 2-targeted biologics.

Table 1: Confounding factors for measurement of blood eosinophil counts

BEC is Higher in:	BEC is Lower in:
Males (compared to females)	Some specific asthma phenotypes
Early morning (compared to afternoon)	Patients taking corticosteroids (oral, inhaled, or intranasal)
Current smokers	
Parasitic infections (e.g., helminths)	
Allergic diseases (atopicdermatitis, allergic rhinitis, drug hypersensitivity)	
Allergen exposure in sensitized individuals	
Non-asthma conditions (e.g., eosinophilic bronchitis, EGPA)	

Fractional Exhaled Nitric Oxide (FeNO)
Typical Criteria for "High" (Adults/Adolescents):
ICS-naïve: > 50 ppb.
Medium-dose ICS: ≥ 25 ppb.
High-dose ICS: ≥ 20 ppb.

Table 2: Confounding factors for measurement of FeNO

FeNO is Higher in:	FeNO is Lower in:
Males (compared to females)	Currentsmokers
Afternoon (compared to early morning)	During bronchoconstriction and with lower lung function
Allergic diseases (atopic dermatitis, allergic rhinitis)	During theearly allergic response
Approx. 24 hours after allergen exposure in sensitized individuals	Patients taking corticosteroids (dose-dependent for ICS; also, oral/nasal)

Management of Severe Asthma
Following figure depicts the management plan for T2 high and T2 low asthma:

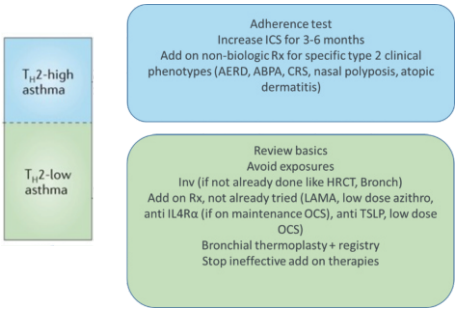


Figure 2: Management plan of severe asthma

If patient needs biologics assess availability and affordability. If targeted therapy is not an option then:

- Add on LAMA/ LTRA/ Low dose azithro if not already trialed
- Consider high dose ICS
- Low dose OCS
- Stop ineffective Rx

To start biologics sound knowledge of the immunopathogenesis of type 2 asthma and specific targets of biologics is critical. Following is the pictorial representation:

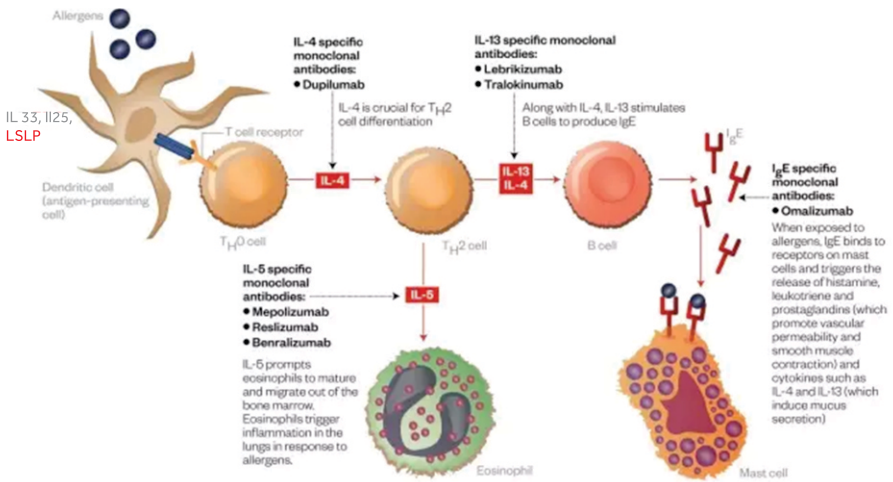


Figure 3: Severe asthma immunopathogenesis in type 2 (T2)-high asthma and the specific targets of biologic therapies. In the T2-high pathway, on exposure to allergens, pollutants or microbes, the airway epithelium releases alarmins such as interleukin (IL)-33, IL-25 and thymic stromal lymphopoietin (TSLP). The dendritic cells (DCs) present these aeroallergens to naïve CD4+ T-cells (Th0), which promotes their differentiation into Th2 cells. IL-4 plays a key role in this differentiation. The Th2 cells, along with type 2 innate lymphoid cells (ILC2), produce high levels of type 2 cytokines such as IL-4, IL5 and IL-13. Besides promoting the differentiation of Th0 to Th2, IL-4, along with IL-13, plays a major role in driving IgE isotype switching in B-lymphocytes. IgE then binds to the high-affinity receptors (FcεRI) on the surface of mast cells and basophils. On re-exposure to the same allergens, these interact with the IgE and induces the mast cells/basophils to release histamines, leukotrienes and prostaglandins resulting in bronchoconstriction. Omalizumab

inhibits the binding of IgE to the high-affinity receptors on mast cells/basophils. IL-5 stimulates proliferation, differentiation and activation of eosinophils. Activated eosinophils release leukotrienes and toxic granules, which leads to airway inflammation, tissue damage and acute asthma flare. Three biologics, mepolizumab, reslizumab and benralizumab, target the IL-5 pathway. Besides its important role in recruiting eosinophils along with IL-4, IL-13 induces nitric oxide synthase, elicits mucus hypersecretion and stimulates airway smooth muscle contraction. Dupilumab inhibits the IL-4 and IL-13 signaling pathways. Inflammation in T2-low asthma is neutrophilic or absent (pauci-granulocytic). The alarmins TSLP and IL-33 may contribute to airway hyperresponsiveness in T2-low asthma. Tezepelumab inhibits the TSLP and the downstream inflammatory cascade.

Image from Google: https://pharmaceutical-journal.com/article/feature/asthma-therapies-get-personal

Once the endotype is confirmed in severe asthma, check the eligibility of various biologics:

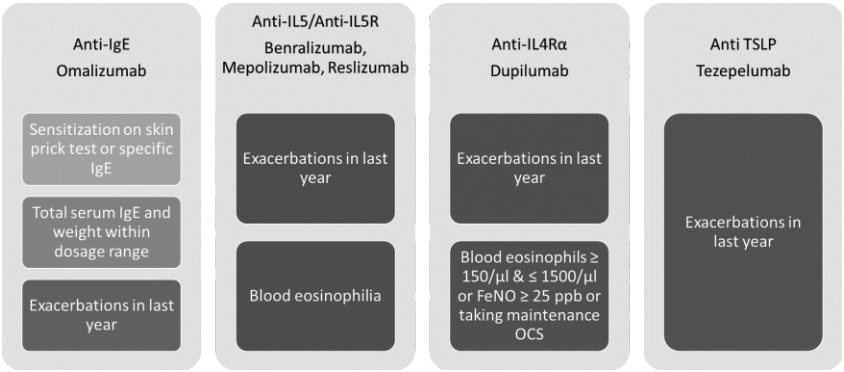


Figure 4: Eligibility of various biologics

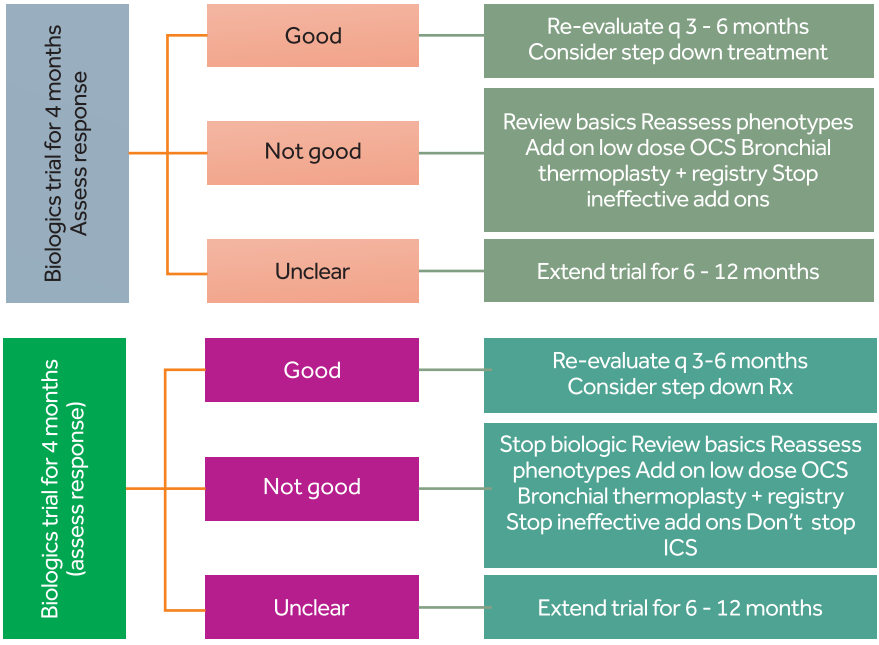
Following table 3 shows the target of different biologics and the common comorbidities that are also addressed:

Table 3: Targets of different biologics

Biologic	Target	Common Comorbidities Addressed
Omalizumab	IgE(Allergy)	Chronic Spontaneous Urticaria (Hives)
Mepolizumab	IL-5(Eosinophils)	Nasal Polyps, EGPA
Benralizumab	IL-5Receptor	Eosinophilic Asthma
Dupilumab	IL-4/IL-13	Atopic Dermatitis, Nasal Polyps
Tezepelumab	TSLP(Alarmin)	Chronic Sinusitis

Assessing Response of Biologics

Assess response to biologics after trial of 4 months.



References:

Global Initiative for Asthma. Global Strategy for Asthma Management and Prevention, 2025. Updated November 2025. Available from: https://www.ginasthma.org.

Chapter 08:

Management of Asthma in 6 – 11 years Age Group

Principles of asthma management in children 6 – 11 years of age are adopted from GINA guidelines, 2025.[1]

Stepwise approach to management of asthma in 6 – 11 years age group
Table 1 gives the stepwise management.

Table 1: Stepwise approach to treatment of asthma in 6 – 11 years age group.

Step	Preferred Controller	Other Controller Options	Reliever
1	Low-dose ICS taken whenever SABA is taken.	N/A.	As-needed SABA.
2	Daily low-dose ICS.	Daily LTRA, or low-dose ICS taken when ever SABA istaken.	As-needed SABA.
3	Low-dose ICS-LABA, OR medium-dose ICS, OR very low-dose ICS-formoterol MART.	Low-dose ICS + LTRA.	As-needed SABA (or ICS - formoterol in MART).
4	Medium-dose ICS-LABA, OR low-dose ICS-formoterol MART, OR refer for expert advice.	Add tiotropium or add LTRA.	As-needed SABA (or ICS - formoterol in MART).
5	Refer for phenotypic assessment ± higher dose ICS-LABA or add-on therapy (e.g., LAMA, anti-IgE, anti-IL4Rα, anti-IL5).	Only as a last resort, OCS (consider side-effects).	As-needed SABA.

Initial treatment selection
The starting point for therapy is determined by the frequency and severity of the child's symptoms.

Table 2 highlights where to start treatment.

Table 2: Starting point for treatment in 6 – 11 years age.

If the patient has...	Start with...	Step Level
Symptoms less than two days a week	Take low-dose ICS whenever SABA is taken.	1
Symptoms 2–5 days a week	Daily low-dose ICS plus as-needed SABA.	2

Symptoms most days, or waking at night once or more a week	Low-dose ICS-LABA, medium-dose ICS, or very-low-dose MART.	3
Symptoms most days, night waking once or more a week, and low lung function	Medium-dose ICS-LABA, or low-dose MART; consider specialist referral.	4

References

1.Global Initiative for Asthma. Global Strategy for Asthma Management and Prevention, 2025. Updated November 2025. Available from: https://www.ginasthma.org.

Chapter 09:

Asthma Exacerbations

Episodes of asthma characterized by a progressive increase in symptoms of shortness of breath, cough, wheezing or chest tightness and progressive decline in lung function, i.e., they represent a change from the patient's usual status that is sufficient to require a change in treatment.[1]
Exacerbation may occur as the first presentation of asthma. Various terms like exacerbation, episode, attack, flare-up are used; for clinical settings term flare-up is recommended as it is easily understood by patients and caregivers too.[2]

Some factors are associated with an increased risk of asthma-related death (Table 1). The presence of any of these must be documented in clinical notes and patients encouraged to seeking urgent medical help in case of exacerbation.

Table 1: Factors associated with increased risk of asthma-related death

1. History of near fatal asthma
2. Emergency visit or hospitalization for asthma in past year
3. Recent or current use of OCS
4. Not currently using ICS
5. Overuse of SABA*
6. Non-compliance to ICS containing medications
7. Poor adherence to or lack written asthma action plan
8. Psychosocial problems
9. Food allergy
10. Comorbidities like pneumonia, arrhythmia, diabetes

*≥ 3 x 200-dose canister/year increase risk of exacerbation; ≥ 1 canister/month increases mortality

Classification
Keeping it simple we may broadly classify asthma exacerbations into following categories (Table 2):

Table 2: Classification of asthma exacerbation

Mild or Moderate	Severe	Life -threatening
Talks in phrases	Talks in words	Drowsy, confused, quiet chest
Prefers sitting to lying	Sits hunched forward	
Not agitated	Agitated	
RR increased	RR > 30/min	
Not using accessory muscles	Uses accessory muscles	
Pulse 100– 120/min	Pulse > 120/min	
SpO2 90– 95%	SpO2 < 90%	
PEF > 50% predicted or best	PEF = 50% predicted or best	

Self-management of exacerbation with written asthma action plan
All patients, caregivers of asthma patients must be given a written asthma action plan, and education on how to use it. It helps recognize and respond optimally to worsening asthma. Use an action plan that is appropriate for patient's reliever: ICS-formoterol, ICS-SABA, or SABA. Following table 3 gives options of medications for written asthma action plan:

Table 3: Options of medications for written asthma action plan

Pathway And Step	Usual asthma treatment	Short-term action plan change for worsening asthma
MART pathway with ICS-formoterol reliever		
Steps 1	AIR only	1 inhalation of ICS-formoterol whenever needed.*
Step 2 – 4	MART	1 inhalation of ICS-formoterol whenever needed.# Continue MART
Alternate pathway with ICS-SABA reliever		
Steps 1	ICS-SABA as-needed	2 inhalations of ICS-SABA whenever needed.$
Step 2	Maintenance ICS	2 inhalations of ICS-SABA whenever needed.$ Continue maintenance ICS
Step 3 – 4	Maintenance ICS-LABA	2 inhalations of ICS-SABA whenever needed.$ Continue maintenance IC-S LABA

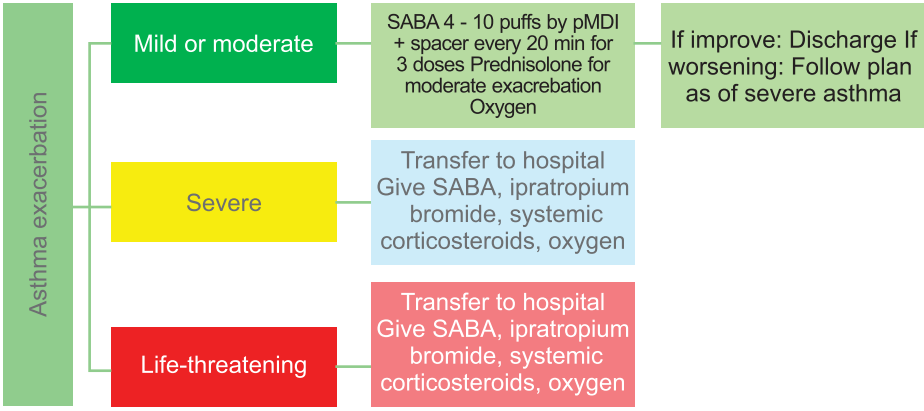
* Maximum 12 inhalations/24 hours

Maximum 12 inhalations/24 hours (as-needed + maintenance doses)

$ Maximum 6 doses (12 inhalations/24 hour)

For patient's on SABA reliever (not recommended), for symptom relief during exacerbation: 2 inhalations of SABA every 4 – 6 hourly and take ICS whenever SABA is taken. For those on maintenance ICS, ICS-formoterol or ICS-LABA: Increase dose of maintenance treatment and as reliever use SABA as already mentioned (2 inhalations 4 – 6 hourly)
For severe exacerbations, not responding to above management over 2 – 3 days, add short course OCS (40 – 50 mg/day for 5 – 7 days). For children dose of OCS is 0.5 mg/kg/day, maximum 40 mg/day for 3 – 5 days. Tapering is not needed if OCS used for less than 2 weeks.
For self-managed exacerbation follow up is needed at 1 – 2 weeks (ideally before stopping OCS)

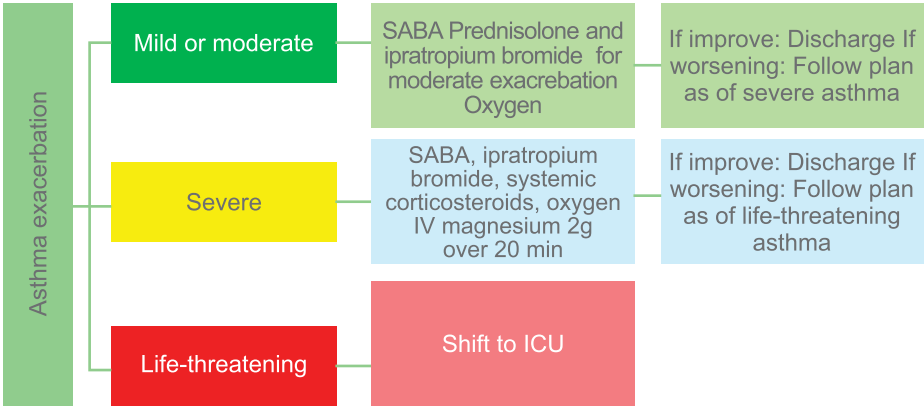
Management of Asthma Exacerbation in Primary Care
Figure 1 shows management of asthma in primary care.[3-17]



Target oxygen saturation for adults 93–95%: ≥ 94% for children

On discharge: Provide reliever as-needed; start or step-up ICS therapy as controller (MART pathway); check inhaler technique, compliance; prednisolone for 5–7 days; follow-up within 2–7 days (1–3 days for children)

Management of asthma exacerbation in emergency department



References

1.Reddel HK, Taylor DR, Bateman ED, Boulet LP, Boushey HA, Busse WW, et al. An official American Thoracic Society/European Respiratory Society statement: asthma control and exacerbations: standardizing endpoints for clinical asthma trials and clinical practice. Am J Respir Crit Care Med. 2009;180(1):59-99.

2.Blaiss MS, Nathan RA, Stoloff SW, et al. Patient and physician asthma deterioration terminology: results from the 2009 Asthma Insight and Management survey. Allergy Asthma Proc. 2012;33(1):47-53.

3.Vincent SD, Toelle BG, Aroni RA, et al. "Exasperations" of asthma. A qualitative study of patient language for worsening asthma. Med J Aust. 2006;184(9):451-454.

4.Cates CJ, Welsh EJ, Rowe BH. Holding chambers (spacers) versus nebulisers for beta-agonist treatment of acute asthma. Cochrane Database Syst Rev. 2013;9:CD000052.

5.Chien JW, Ciufo R, Novak R, et al. Uncontrolled oxygen administration and respiratory failure in acute asthma. Chest. 2000;117(3):728-733.

6.Rodrigo GJ, Rodriquez Verde M, Peregalli V, et al. Effects of short-term 28% and 100% oxygen on $PaCO_2$ and peak expiratory flow rate in acute asthma: a randomized trial. Chest. 2003;124(4):1312-1317.

7.Perrin K, Wijesinghe M, Healy B, et al. Randomised controlled trial of high concentration versus titrated oxygen therapy in severe exacerbations of asthma. Thorax. 2011;66(11):937-941.

8.Patel B, Khine H, Shah A, et al. Randomized clinical trial of high concentration versus titrated oxygen use in pediatric asthma. Pediatr Pulmonol. 2019;54(7):970-976.

9.Hasegawa T, Ishihara K, Takakura S, et al. Duration of systemic corticosteroids in the treatment of asthma exacerbation; a randomized study. Intern Med. 2000;39(10):794-797.

10.Jones AM, Munavvar M, Vail A, et al. Prospective, placebo-controlled trial of 5 vs 10 days of oral prednisolone in acute adult asthma. Respir Med. 2002;96(12):950-954.

11.Newman KB, Milne S, Hamilton C, et al. A comparison of albuterol administered by metered-dose-inhaler and spacer with albuterol by nebulizer in adults presenting to an urban emergeny department with acute asthma. Ch 121: 1036–1041.

12.Kayani S, Shannon DC. Adverse behavioral effects of treatment for acute exacerbation of asthma in children: a comparison of two doses of oral steroids. Chest. 2002;122(2):624-628.

13.Kirkland SW, Vandenberghe C, Voaklander B, et al. Combined inhaled beta-agonist and anticholinergic agents for emergency management in adults with asthma. Cochrane Database Syst Rev. 2017;1:CD001284.

14.Craig SS, Dalziel SR, Powell CV, et al. Interventions for escalation of therapy for acute exacerbations of asthma in children: an overview of Cochrane Reviews. Cochrane Database Syst Rev. 2020;8:CD0012977.

15.Rodrigo GJ, Castro-Rodriguez JA. Anticholinergics in the treatment of children and adults with acute asthma: a systematic review with meta-analysis. Thorax. 2005;60(9):740-746.

16.Nair P, Milan SJ, Rowe BH. Addition of intravenous aminophylline to inhaled beta(2)-agonists in adults with acute asthma. Cochrane Database Syst Rev. 2012;12:CD002742.

17.Ambrożej D, Adamiec A, Forno E, et al. Intravenous magnesium sulfate for asthma exacerbations in children: Systematic review with meta-analysis. Paediatr Respir Rev. 2024;52:23-30.

Chapter 10:

Immunotherapy in Asthma

Asthma affects over 300 million individuals globally and presents a significant burden on healthcare systems. In atopic asthma, which is often driven by sensitization to environmental allergens, standard therapy includes inhaled corticosteroids (ICS) and bronchodilators. However, these treatments control symptoms without modifying the underlying immunopathology. Allergen immunotherapy represents a unique intervention that can alter the natural history of allergic diseases, offering sustained clinical benefits even after discontinuation.

Mechanism of Action

Immunotherapy exerts its effects by inducing immunological tolerance through repeated administration of specific allergens. The immunological mechanisms include a shift from Th2-type to Th1-type responses, increased production of allergen-specific IgG4 "blocking antibodies", and the induction of regulatory T (Treg) and B (Breg) cells, which secrete IL-10 and TGF-β to dampen inflammatory pathways[1,2]. These changes reduce eosinophilic inflammation, mast cell activation, and cytokine release.

Clinical Efficacy

Both subcutaneous immunotherapy (SCIT) and sublingual immunotherapy (SLIT) have demonstrated efficacy in improving asthma symptoms and reducing medication use, particularly in patients with mild to moderate allergic asthma[3]. A meta-analysis by Dhami et al. found significant reductions in symptom scores and inhaled corticosteroid use with asthma immunotherapy (AIT), along with improved quality of life[4]. However, SCIT carries a risk of systemic reactions, including anaphylaxis, necessitating its administration in controlled settings. SLIT offers a safer profile and can be self-administered, though it may be less potent in some patients[5].

Patient Selection and Guidelines

The Global Initiative for Asthma (GINA) recommends considering AIT in patients with allergic asthma who have demonstrable allergen sensitization and suboptimal control despite standard therapy[6]. Patients with severe asthma, uncontrolled symptoms, or a history of anaphylaxis are typically excluded due to safety concerns. Early initiation in childhood may reduce the risk of progression to more severe disease or the development of new sensitizations[7].

Future Directions

Ongoing research aims to enhance AIT's safety and efficacy through novel adjuvants, peptide-based vaccines, and recombinant allergens. Biomarkers such as allergen-specific IgE/IgG4 ratios and basophil activation tests are under investigation to predict responsiveness[8]. Additionally, combining AIT with biologics (e.g., anti-IgE or anti-IL-5 therapies) may synergistically improve outcomes in selected patients.

Immunotherapy represents a paradigm shift in the management of allergic asthma by targeting the underlying immune dysregulation. While not suitable for all patients, it provides durable benefits for appropriately selected individuals. Future advancements may broaden its applicability and improve patient outcomes through personalized approaches.

References

1.Akdis CA, Akdis M. Mechanisms of allergen-specific immunotherapy and immune tolerance to allergens. World Allergy Organization Journal. 2015 Dec;8(1):1-2.

2.Calderon MA, Demoly P, Gerth van Wijk R, Bousquet J, Sheikh A, Frew A, Scadding G, Bachert C, Malling HJ, Valenta R, Bilo B. EAACI: A European Declaration on Immunotherapy. Designing the future of allergen specific immunotherapy. Clinical and translational allergy. 2012 Oct 30;2(1):20.

3.Abramson MJ, Puy RM, Weiner JM. Injection allergen immunotherapy for asthma. Cochrane database of systematic reviews. 2010(8).

4.Dhami S, Kakourou A, Asamoah F, Agache I, Lau S, Jutel M, Muraro A, Roberts G, Akdis CA, Bonini M, Cavkaytar O. Allergen immunotherapy for allergic asthma: a systematic review and meta-analysis. Allergy. 2017 Dec;72(12):1825-48.

5.Canonica GW, Cox L, Pawankar R, et al. Sublingual immunotherapy: World Allergy Organization position paper 2013 update. World Allergy Organ J. 2014;7(1):6.

6.Global Initiative for Asthma. Global Strategy for Asthma Management and Prevention, 2024. [Internet]. Available from: https://ginasthma.org

7.Tosca MA, Licari A, Olcese R, Castagnoli R, Marseglia A, Marseglia GL, Del Giudice MM, Martelli A, Calvani M, Caffarelli C, Duse M. Allergen immunotherapy in children and adolescents with respiratory diseases. Acta Bio Medica: Atenei Parmensis. 2020 Sep 15;91(Suppl 11):e2020006.

8.López JF, Imam MB, Satitsuksanoa P, Lems S, Yang M, Hwang YK, Losol P, Choi JP, Kim SH, Chang YS, Akdis M. Mechanisms and biomarkers of successful allergen-specific immunotherapy. Asia Pacific Allergy. 2022 Oct 1;12(4):e45.

Chapter 11:
Asthma + COPD

Asthma and chronic obstructive pulmonary disease (COPD) are two of the most commonly encountered chronic lung diseases. However, differentiating between the two may be difficult at times.

Asthma is characterized clinically by cough, shortness of breath, wheeze, and tightness of chest, and spirometrically with reversible airway obstruction, usually associated with atopy. In contrast COPD is progressively worsening shortness of breath associated with minimally reversible airway obstruction usually after exposure to smoke. However, physicians identified patients who had features of both and these patients were assigned to an entity of overlap of both asthma and COPD.

Epidemiology
In Pakistan, the prevalence of Asthma + COPD is poorly documented due to overlapping symptoms and diagnostic challenges. A hospital-based study in Sindh estimated that 9.3% of patients presenting with chronic respiratory symptoms met the criteria for Asthma + COPD.[1]

Depending on the definition, diagnosis criteria and population analyzed the prevalence of Asthma + COPD varies widely in different studies such as it is 0.9-11.1% in general population but it is also found in the range of 1.6- 4.5%, in COPD patients between 12.1% and 55.2% . In a meta-analysis review that included 19 studies, the prevalence of Asthma + COPD was 27% with a COPD diagnosis. In different studies, prevalence varies from 11- 25% due to variation in definition.[2]

Clinical Features and Risk Factors
Factors that tend to increase probability of Asthma + COPD in asthmatic patients include old age (as elderly asthmatic patients usually show more features of fixed obstruction and more severe symptoms and being a current smoker (as smoking leads to a decline in FEV1 of 66 ml/year in men and 52 ml/year in women and leads to an increased risk of airway remodeling).

Factors that tend to increase probability of Asthma + COPD in COPD patients include history of atopy (that is associated with elevated total IgE), increased sputum and/or blood eosinophilia , and a slightly increased nitric oxide level.[3]

Asthma and COPD are punctuated by exacerbations, but overlap syndrome may be associated with three times the frequency and severity of exacerbations.[4]

Asthma + COPD have more frequent exacerbations, more wheezing and dyspnoea, but similar cough and sputum production compared with COPD. In 1961, a "Dutch hypothesis," presented by Orie and colleagues5 acknowledged the frequent problems in differentiating between asthma and COPD, especially in older adults who currently smoke or have a significant history of cigarette smoking. "Dutch hypothesis" tried to answer the question, hypothesizing that asthma and Bronchial Hyper Responsiveness (BHR) predispose to COPD later in life and that asthma, COPD, chronic bronchitis, and emphysema are different expressions of a single airway disease. Several terms have been identified within the literature that described the overlap phenotype of asthma and COPD: 'Asthmatic bronchitis', 'COPD with a prominent asthmatic component', 'asthma that complicates

COPD' and 'mixed COPD-asthma'[2].[6] The consensus document selected this differential entity as "mixed COPD-asthma phenotype".[7] However the term Asthma-COPD Overlap Syndrome (ACOS) has been more frequently used lately. The prevalence of Asthma + COPD varies considerably because it has been diagnosed using different criteria, which depend on the study design and the population. Within the published literature, Asthma + COPD prevalence varied between asthmatic patient populations and COPD patient populations. This may be attributable to differences in age or smoking status, which are both risk factors for COPD and Asthma + COPD.[8] In patients with a pre-existing diagnosis of asthma, the prevalence of Asthma + COPD was 29% when they had chronic bronchitis and/or impairment in the diffusing capacity of the lung for carbon monoxide (DLCO). In patients with a pre-existing diagnosis of COPD, the prevalence of Asthma + COPD was 13% when the patients had self-reported, physician-diagnosed asthma before the age of 40 years and increased up to 55% when the patients met any criteria for asthma.[6]

Definition

ASTHMA+COPD has not yet been defined definitively. Just as asthma and COPD are heterogenous disorders, each having its own broad range of underlying mechanisms and pathophysiology. ASTHMA+COPD does not appear to be a single disease and represents a spectrum of disease process ranging from asthma at one end and COPD at the other. Asthma + COPD is still poorly characterized, both in terms of general risk factors and pathophysiology, and in terms of clinical symptoms, treatment response and prognosis.

A joint document issued by Global Initiative against Asthma (GINA) and Global Initiative for Chronic Obstructive Lung Disease (GOLD) defines that Asthma + COPD is "characterized by chronic airflow limitations with several features associated with asthma and several features associated with COPD" but this definition is vague[9] Many attempts have been made to define this collection of symptoms[10] A round table discussion tried to reach a consensus on defining Asthma + COPD.7 Three key features were identified to be included in the operational definition:

- Persistent airflow limitation on spirometry despite adequate administration of a short-acting bronchodilator in subjects 40 years of age or older;
- A "significant" history of cigarette smoking or an equivalent lifetime exposure to biomass; and
- A physician diagnosis of asthma before 40 years of age. Another consensus document[11] agreed on the same components or traits of Asthma + COPD.

Differentiating Asthma, COPD, and Asthma + COPD

The Clinicians should assemble the features for asthma and for COPD that best describe the patient and compare the number of features in favor of each diagnosis. In practice, if three or more features of either asthma or COPD are present, that diagnosis is suggested; if there are similar numbers of features of asthma and COPD, the diagnosis of Asthma + COPD should be considered. The relevant variables are age at onset, pattern and time course of symptoms, personal history or family history, variable or persistent airflow limitation, lung function between symptoms, and severe hyperinflation.

Potential Biomarkers

Two biomarkers may be of utility in identifying Asthma + COPD. Serum periostin, a matricellular protein highly expressed in allergic diseases, and YKL-40, a glycoprotein that is secreted by inflammatory and airway epithelial cells, and is upregulated in COPD[5].

Diagnosis

Although there is no agreed, established and validated definition for Asthma + COPD, this entity is widely recognized in clinical practice as an individualized phenotype demarcated from the spectrum of COPD, or a subset of COPD. Thus, it was imperative that consensus

be reached to standardize the diagnosis. Certain diagnostic criteria have been proposed and agreed upon by national guidelines[7, 11] and studies.[12] Table 1 gives the most friendly diagnostic basis for asthma+COPD.

Table 1: Clinical phenotype of patients with chronic respiratory symptoms

Highly Likely to be Asthma	Features of Both Asthma + COPD	Likely to be COPD
Treat as Asthma	Treat as Asthma	Treat as COPD
HISTORY	HISTORY	HISTORY
Symptoms vary over time and in intensity Triggers may include laughter, exercise, allergens, seasonal Onset before age 40 years Symptoms improve spontaneously or with bronchodilators (minutes) or ICS (days to weeks) Current asthma diagnosis, or asthma diagnosis in childhood	Symptoms intermittent or episodic May have started before or after age 40 May have a history of smoking and/or other toxic exposures, or history of low birth weight or respiratory illness such as tuberculosis Any of asthma features at left (e.g. common triggers; symptoms improve spontaneously or with bronchodilators or ICS; current asthma diagnosis or asthma diagnosis in childhood)	Dyspnea persistent (most days) Onset after age 40 years Limitation of physical activity May have been preceded by cough/sputum Bronchodilator provides only limited relief History of smoking and/or other toxic exposure, or history of low birth weight or respiratory illness such as tuberculosis No past or current diagnosis of asthma
LUNG FUNCTION	LUNG FUNCTION	LUNG FUNCTION
Variable expiratory airflow limitation	Persistent expiratory airflow limitation	Persistent expiratory airflow limitation
Persistent airflow limitation may be present	With or without bronchodilator reversibility	With or without bronchodilator reversibility

Management

In principle, Asthma + COPD has similar goals of treatment as asthma and COPD:

a) control and relief of symptoms,
b) reduction in the frequency of exacerbations,
c) a reduction in the rate of decline in lung function and
d) limiting adverse effects from therapeutic treatments.[6]

The clinical evidence of pharmacological therapy is lacking as very few studies have been conducted on defined patients of Asthma + COPD and such patients with overlapping symptoms have been excluded from trials.

However, the following suggestions have been strongly recommended by consensus[6,13] Majority of patients have been reported to have benefited from a combination of inhaled corticosteroids and long acting bronchodilators (LABA). A valid concern has been raised about use of LABA alone in patients with most features of asthma. However, ICS/LABA combination has been favored by most consensus guidelines.[6,13]

Patients responding poorly to ICS/LABA or with severe disease should be offered triple therapy with ICS/LABA and LAMA (long acting anti muscarinic agents). This practice appears to benefit COPD patients with concomitant asthma (Table 2).[14]

Table 2: Management of Asthma + COPD

Highly Likely to be Asthma	Features of Both Asthma + COPD	Likely to be COPD
Treat as Asthma	Treat as Asthma + COPD	Treat as COPD
ICS-containing treatment is essential to reduce risk of severe exacerbations and death.	ICS-containing treatment is essential to reduce risk of severe exacerbations and death.	Treat as COPD
		Initially maintenance LABA-LAMA
MART pathway with ICS-formoterol as reliever is the preferred regimen.	Add-on LABA and/or LAMA usually also needed	Add ICS as per for patients with hospitalizations, ≥2 exacerbations/year requiring OCS, or blood eosinophils ≥300/µl
Do not give LABA and/or LAMA without ICS	Additional COPD treatments	Avoid high dose ICS, avoid maintenance OCS
Maintenance OCS only as last resort	Do not give LABA and/or LAMA without ICS	Reliever containing ICS is not recommended
	Maintenance OCS only as last resort	

Other components of treatment may comprise the following: a) patient education, b) smoking cessation, c) allergen avoidance, d) flu vaccination, e) pulmonary rehabilitation and f) management of any comorbidity.

Prognosis

Patients with Asthma + COPD experience more rapid decline in lung function, frequent exacerbations,[15] have poorer health-related quality-of-life (HRQoL) outcomes, and require a large amount of medical resources compared to patients with asthma or COPD alone.[14-17]

Conclusion

Asthma + COPD can be termed as interim term as many lacunae exist on definitive definition, pathophysiology, molecular basis of disease, clinical features, diagnostic biomarkers, and management principles. More studies need to be conducted to enlighten this entity.

References

1.Sabar MF, Khan AA. An Overview of Asthma COPD Overlapping Syndrome (ACOS). Journal Name. Year;Volume(Issue):Pages.

2.Shabaan AY, Daabis RG, Abdelhady AM, Ibrahim DH. Prevalence of asthma-chronic obstructive pulmonary disease overlap in patients with airflow limitation. Egypt J Bronchol. 2021;15(1):44.

3.Papaivannou A, Zarogoulidis P, Porpodis K, Spyratos D, Kioumis I, Pitsiou G, et al. Asthma-chronic obstructive pulmonary disease overlap syndrome (ACOS): current literature review. J Thorac Dis. 2014 Mar;6 Suppl 1:S146-51.

4.Barrecheguren M, Esquinas C, Miravitlles M. The asthma-chronic obstructive pulmonary disease overlap syndrome (ACOS): opportunities and challenges. Curr Opin Pulm Med. 2015 Jan;21(1):74-9.

5.Orie NGM, Sluiter HJ, editors. Bronchitis. Assen, the Netherlands: Royal Van Gorcum; 1962.

6.2. Nguyen Van T, Park HY, Nakano Y. Asthma-COPD overlap syndrome (ACOS): A diagnostic challenge. Respirology. 2016 Apr;21(3):410-8.

7.Soler-Cataluña JJ, Cosío B, Izquierdo JL, López-Campos JL, Marín JM, Agüero R, et al. Consensus document on the overlap phenotype COPD-asthma in COPD. Arch Bronconeumol. 2012 Aug;48(9):331-7.

8.Marsh S, Travers J, Weatherall M, Williams M, Aldington S, Shirtcliffe P, et al. Proportional classifications of COPD phenotypes. Thorax. 2008 Sep;63(9):761-7.

9.Global Initiative for Chronic Obstructive Lung Disease (GOLD). Diagnosis of Diseases of Chronic Airflow Limitation: Asthma, COPD and Asthma-COPD Overlap Syndrome (ACOS) [Internet]. 2015 [cited 2015 Jan]. Available from: https://goldcopd.org/wp-content/uploads/2016/04/GOLD_ACOS_2015.pdf

10.Sin DD, Miravitlles M, Mannino DM, Soriano JB, Price D, Celli BR, et al. What is asthma-COPD overlap syndrome? Towards a consensus definition from a round table discussion. Eur Respir J. 2016 Sep;48(3):664-73.

11.Koblizek V, Chlumsky J, Zindr V, Neumannova K, Zatroukal J, Zak J, et al. Chronic Obstructive Pulmonary Disease: official diagnosis and treatment guidelines of the Czech Pneumological and Phthisiological Society; a novel phenotypic approach to COPD with patient-oriented care. Biomed Pap Med Fac Univ Palacky Olomouc Czech Repub. 2013 Sep;157(2):189-201.

12.Araújo D, Padrão E, Morais-Almeida M, Cardoso J, Silva J, Sobradeiro A, et al. Asthma-chronic obstructive pulmonary disease overlap syndrome - Literature review and contributions towards a Portuguese consensus. Rev Port Pneumol (2006). 2017 Mar-

Apr;23(2):90-9.
13.Magnussen H, Bugnas B, van Noord J, Schmidt P, Gerken F, Kesten S. Improvements with tiotropium in COPD patients with concomitant asthma. Respir Med. 2008 Jan;102(1):50-6.
14.Mannino DM, Gagnon RC, Petty TL, Lydick E. Obstructive lung disease and low lung function in adults in the United States: data from the National Health and Nutrition Examination Survey, 1988-1994. Arch Intern Med. 2000 Jun 12;160(11):1683-9.
15.Kauppi P, Kupiainen H, Lindqvist A, Tammilehto L, Kilpeläinen M, Kinnula VL, et al. Overlap syndrome of asthma and COPD predicts low quality of life. J Asthma. 2011 Apr;48(3):279-85.
16.Miravitlles M, Soriano JB, Ancochea J, Muñoz L, Duran-Tauleria E, Sánchez G, et al. Characterisation of the overlap COPD-asthma phenotype. Focus on physical activity and health status. Respir Med. 2013 Jul;107(7):1053-60.
17.Habib FZ, Mahmood SU, Muhammad A, Shah U, Muzaffar S, Naqvi SS. Prevalence of undiagnosed chronic obstructive pulmonary disease (COPD) and asthma in underserved rural Sindh, Pakistan. Chest. 2023 Oct;164(4):A5039.

Chapter 12:
Asthma in Special Situations

Asthma in Pregnancy
Pregnancy induces hormonal and mechanical changes that can alter asthma control: some women worsen, others improve, and many remain stable. The primary goal is maintaining control with minimal inflammation and minimizing exacerbations. Inhaled corticosteroids (particularly budesonide), short-acting β_2-agonists (SABA), and long-acting β_2-agonists (LABA) are considered safe during pregnancy. Systemic corticosteroids are reserved for severe exacerbations. Regional guidelines in South-Asian countries align with GINA recommendations, emphasizing the importance of pre-pregnancy planning, patient education, and multidisciplinary care.[1,2]

- The "Rule of One-Thirds": During pregnancy, asthma control typically worsens in about 1/3 of women, improves in 1/3, and remains stable in 1/3.
- Inhaled Corticosteroids (ICS): These are the cornerstone of treatment. There is no evidence of increased fetal abnormalities with ICS (e.g., budesonide, fluticasone).
- Frequent Review: Asthma should be monitored every 4–6 weeks during pregnancy.
- Step-Down Therapy: While "stepping up" treatment for poor control is encouraged, "stepping down" is often deferred until after delivery to avoid a flare-up during gestation.
- Acute Exacerbations: These must be treated aggressively. Oxygen saturation should be maintained at ≥ 95% to ensure adequate fetal oxygenation.

Occupational Asthma and Work-related Exposures
Occupational asthma—linked to exposure to dusts, fumes, chemicals, and biological allergens—is a significant concern in industrial and agricultural settings. Diagnosis requires objective testing (spirometry, peak flows, specific inhalation challenge) and identification of causative agents. Management includes avoidance or reduction of exposure, pharmacotherapy, and, if needed, workplace accommodation. National surveillance data are limited in South Asia, but occupational health authorities have issued guidance aligned with international standards.[3,4]

Exercise-Induced Bronchoconstriction (EIB)
EIB is common among children, athletes, and those with poorly controlled asthma. Preventive strategies include a warm-up period, ICS/formoterol before exercise and regular controller inhaler therapy if symptoms persist. Non-pharmacological strategies such as breathing techniques and mask use in cold air are widely applicable.[5]

Asthma in Obesity and Metabolic Syndrome
Obesity is associated with more severe asthma symptoms, reduced response to corticosteroids, and increased exacerbations. Weight reduction through diet and lifestyle modifications improves control and lung function. The rising prevalence of obesity and metabolic syndrome makes this increasingly relevant. Different health programs are beginning to integrate asthma and non-communicable disease management strategies.[6]

Allergen-Induced Asthma and Atopic Dermatitis Overlap
Atopic asthma often overlaps with other allergic diseases such as atopic dermatitis and allergic rhinitis. This “atopic march” is influenced by indoor allergens (dust mites, cockroaches), air pollution, and early-life exposures. Management requires coordinated care addressing comorbidities, environmental controls, and tailored immunotherapy where available. Region-specific allergen avoidance guidance is included in some asthma and allergy association recommendations.[7]

Catamenial Asthma

Catamenial asthma is a distinct, often severe, asthma phenotype characterized by a pre-menstrual and/or menstrual worsening of asthma symptoms and a reduction in lung function, most notably peak expiratory flow (PEF)[8] This condition is believed to affect approximately 30% of premenopausal women with asthma, and its pathophysiology is strongly linked to the cyclical fluctuations in female sex hormones, particularly the sharp pre-menstrual decline in progesterone and estradiol.[9] The proposed mechanisms include a direct effect of these hormones on airway smooth muscle tone, changes in inflammatory mediator release, and altered systemic immune responses.[10] Catamenial asthma is frequently refractory to standard asthma controller therapy, often requiring specialized management that may include high-dose inhaled or oral corticosteroids, or the use of gonadotropin-releasing hormone GnRH agonists or progesterone supplements to stabilize hormone levels.[8]

Aspirin-Exacerbated Respiratory Disease

Aspirin-Exacerbated Respiratory Disease (AERD), presenting as Samter's Triad, is a chronic condition defined by the coexistence of asthma, recurrent nasal polyps, and hypersensitivity to aspirin or other NSAIDs. The prevalence of AERD in general public is 7% and in severe asthma it is 15%.[11,12] Reactions to aspirin include nasal congestion, wheezing, and chest tightness, driven by abnormal inflammatory pathways involving leukotrienes and eosinophils. Management focuses on NSAID avoidance, corticosteroids, sinus surgery, and aspirin desensitization protocols.

- AERD is driven by an imbalance in the arachidonic acid metabolism, leading to an overproduction of pro-inflammatory cysteinyl leukotrienes and a reduction in anti-inflammatory prostaglandins (PGE2).
- The primary strategy is the strict avoidance of aspirin and all COX-1 inhibiting NSAIDs.
- Most patients can safely take Acetaminophen (Paracetamol) at low doses (≤500mg) or selective COX-2 inhibitors.[13]
- Leukotriene receptor antagonists (LTRAs) or synthesis inhibitors (e.g., Montelukast) may also be used.[14]
- ICS is mainstay of treatment.
- For patients who require aspirin for cardiovascular disease or arthritis, or those with recalcitrant nasal polyps, aspirin desensitization can be performed by a specialist. This involves giving increasing doses of aspirin daily to induce tolerance, which can significantly improve both upper and lower respiratory symptoms.[15]

Allergic Bronchopulmonary aspergillosis

Allergic bronchopulmonary aspergillosis (ABPA) is a hypersensitivity reaction to Aspergillus fumigatus that occurs predominantly in patients with asthma or cystic fibrosis. It is characterized by airway inflammation, mucus plugging, and central bronchiectasis, which can lead to progressive lung damage if untreated. Clinical manifestations include wheezing, productive cough with brownish mucus plugs, and recurrent pulmonary infiltrates.[16] Diagnosis is based on a criteria which includes hypersensitivity reaction to A.fumigatus, total serum IgE, specific IgG to A.fumigatus, radiological features and blood eosinophils. The mainstay of treatment is systemic corticosteroids, followed by antifungal agents such as itraconazole or voriconazole used to reduce fungal burden and steroid dependence (in those with exacerbations or requiring long term OCS).[17] Biologic agents like omalizumab may be used in refractory cases.

Asthma and Surgery

Asthma poses significant challenges during surgery, as patients are at increased risk of perioperative bronchospasm, hypoxemia, and complications related to airway hyperreactivity. Careful preoperative assessment and optimization with bronchodilators and corticosteroids are essential to reduce risk.[18] Intraoperatively, the choice of anesthetic agents and ventilation strategies must minimize airway irritation, while vigilance for bronchospasm is critical. Postoperative care should emphasize monitoring for respiratory compromise and continuation of asthma therapy.[19]

Patients must not stop their inhaled corticosteroids or combination inhalers. They should take their usual doses on the day of surgery.

For patients with suboptimal control or those undergoing major surgery, a short course of oral corticosteroids (OCS) (e.g., prednisolone 30–40 mg/day for 5–7 days) may be prescribed leading up to the procedure to "quiet" airway inflammation.

If a patient has been on long-term high-dose ICS or frequent OCS (for more than 2 weeks during the past 6 months), they may need "stress-dose" hydrocortisone during the perioperative period to prevent an adrenal crisis.

Asthma and Air Travel

We adopt recommendations of the British Thoracic Society for air travel with asthma.[20] The recommendations are optimization of treatment before air travel and carrying all treatment and action plan during air travel.

- The patient's condition should be optimized before travel, with attention paid to inhaler technique and smoking cessation referral as required.
- All medications and spacer devices should be carried in hand luggage.
- Emergency medications, including salbutamol inhalers and spacers, must be immediately accessible.
- Individuals prescribed epinephrine auto-injectors should have them readily available.
- For acute exacerbations on board, the passenger's own bronchodilator inhaler should be given, with a spacer if needed.
- The passenger should alert the cabin crew if symptoms do not respond rapidly to use of the inhaler, or if they recur after a short interval.
- Those with severe asthma take an emergency supply of OCS in hand luggage.
- Carry copy of asthma action plan.
- Food allergy affects up to 8.5% of children and adults with asthma. Appropriate precautions for those affected include wiping tray tables and hands, informing the airline beforehand and the cabin crew of allergies, and not eating during flights or bringing known 'safe' foods from home.

Future Directions

More epidemiological and interventional research on asthma in special populations-particularly pregnant women, workers exposed to occupational hazards, and children with EIB are required specially in low- and middle-income countries. Building regional registries, integrating asthma care within maternal/child health programs, and developing culturally tailored educational tools are priority areas.

Asthma in special situations demands individualized management-balancing safety, efficacy, and context. While most evidence derives from global studies, regional guidelines in South Asia adapt these principles thoughtfully. Addressing research gaps and integrating asthma care within broader public health frameworks will enhance outcomes in these challenging clinical scenarios.

References

1.Global Initiative for Asthma. GINA strategy 2025: asthma management in pregnancy. 2025 update.

2.Fu QL, Du Y, Xu G, Zhang H, Cheng L, Wang YJ, Zhu DD, Lv W, Liu SX, Li PZ, Shi JB. Prevalence and occupational and environmental risk factors of self-reported asthma: evidence from a cross-sectional survey in seven Chinese cities. International Journal of Environmental Research and Public Health. 2016 Nov;13(11):1084.

3.Tarlo SM, Lemiere C. Occupational asthma. N Engl J Med. 2014;370(7):640-9.

4.Mishra, J., Acharya, S., Taksande, A.B., Prasad, R., Munjewar, P.K., Wanjari, M.B., Munjewar, P. and Wanjari, M., 2023. Occupational risks and chronic obstructive pulmonary disease in the Indian subcontinent: a critical review. Cureus, 15(6).

5.Parsons JP, Mastronarde JG. Exercise-induced bronchoconstriction in athletes. Chest. 2005;128(6):3966-74.

6.Forno E, Celedón JC. The effect of obesity, weight gain, and weight loss on asthma inception and control. Curr Opin Allergy Clin Immunol. 2017;17(2):123-30.

7.Brożek JL, Bousquet J, Agache I, Agarwal A, Bachert C, Bosnic-Anticevich S, Brignardello-Petersen R, Canonica GW, Casale T, Chavannes NH, De Sousa JC. Allergic Rhinitis and its Impact on Asthma (ARIA) guidelines—2016 revision. Journal of Allergy and Clinical Immunology. 2017 Oct 1;140(4):950-8.

8.Sánchez-Ramos, J.L., Pereira-Vega, A.R., Alvarado-Gómez, F., Maldonado-Pérez, J.A., Svanes, C. and Gómez-Real, F., 2017. Risk factors for premenstrual asthma: a systematic review and meta-analysis. Expert Review of Respiratory Medicine, 11(1), pp.57-72.

9.Singh G, Trehan S, Goswami K, Singh A, Puri P. Cyclical exacerbation of asthma symptoms in relation to the menstrual cycle: a case report on catamenial asthma. Cureus. 2024 Jul 14;16(7).

10.Vrieze A, Postma DS, Kerstjens HA. Perimenstrual asthma: a syndrome without known cause or cure. Journal of allergy and clinical immunology. 2003 Aug 1;112(2):271-82.

11.Morales DR, Guthrie B, Lipworth BJ, et al. NSAID-exacerbated respiratory disease: a meta-analysis evaluating prevalence, mean provocative dose of aspirin and increased asthma morbidity. Allergy. 2015;70(7):828-835.

12.Rajan JP, Wineinger NE, Stevenson DD, et al. Prevalence of aspirin-exacerbated respiratory disease among asthmatic patients: A meta-analysis of the literature. J Allergy Clin Immunol. 2015;135(3):676-681.e671.

13.El Miedany Y, Youssef S, Ahmed I, et al. Safety of etoricoxib, a specific cyclooxygenase-2 inhibitor, in asthmatic patients with aspirin-exacerbated respiratory disease. Ann Allergy Asthma Immunol. 2006;97:105-109.

14.Morales DR, Lipworth BJ, Guthrie B, et al. Safety risks for patients with aspirin-exacerbated respiratory disease after acute exposure to selective nonsteroidal anti-inflammatory drugs and COX-2 inhibitors: Meta-analysis of controlled clinical trials. J Allergy Clin Immunol. 2014;134:40-45.

15.Ref 634: Dahlen SE, Malmstrom K, Nizankowska E, et al. Improvement of aspirin-intolerant asthma by montelukast, a leukotriene antagonist: a randomized, double-blind, placebo-controlled trial. Am J Respir Crit Care Med. 2002;165:9-14.

16.Saxena P. Allergic Bronchopulmonary Aspergillosis: What's New. Curr Pulmonol Rep. 2025;14(10):1-9. doi:10.1007/s13665-025-00375-2

17.He Q, Li M, Cao J, Zhang M, Feng C. Clinical characteristics and risk factors analysis of allergic bronchopulmonary aspergillosis combined with bronchiectasis. Frontiers in Medicine. 2025 May 23;12:1557241.

18.Thejane F. Perioperative management of asthmatic patients.

19.Jordan RR. Preoperative Optimization of the Asthmatic Patient. Anesthesia eJournal. 2022 Nov 29;10(4):1-2.

20.Coker RK, Armstrong A, Church AC, Holmes S, Naylor J, Pike K, et al. BTS Clinical Statement on air travel for passengers with respiratory disease. Thorax. 2022;77(4):329-350. doi:10.1136/thoraxjnl-2021-218110

Chapter 13:

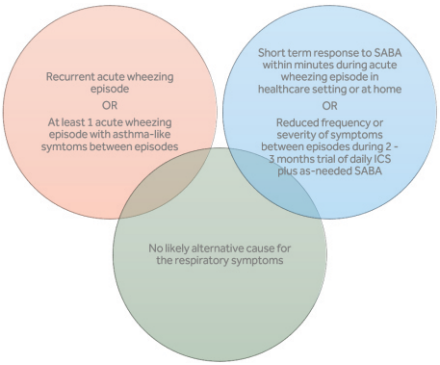
Paediatric Asthma (≤ 5 years)

This section is adopted from the GINA guideline 2025. [1]

Diagnosis

Diagnosis of asthma in children is challenging. A structured criteria-based approach to diagnosis is recommended (Figure 1).

Figure 1: Diagnostic criteria of asthma (≤ 5 years)



* All three criteria must meet to diagnose asthma.

There are some red flags, when asthma diagnosis is down the list and patient needs urgent referral to hospital.

Failure to thrive
Neonatal or very early onset of symptoms (especially if associated with failure to thrive)
Vomiting associated with respiratory symptoms
Continuous wheezing, recurrent stridor, or seal-like barking cough (suggestive of airway malacia)
Failure to respond to asthma medications (inhaled ICS, oral steroids, or SABA)
No association of symptoms with typical triggers, such as viral URTI
Focal lung or cardiovascular signs, or finger clubbing
Hypoxemia (<95%)

Management

Table 1: Management of asthma in children (≤ 5 years)

Step	Preferred Controller Choice	Other Options / Considerations	When to Consider This Step
Step 1	None (insufficient evidence)	Intermittent short-course ICS at onset of viral illness	Infrequent acute wheezing with minimal interval symptoms
Step 2	Daily low dose ICS	Daily LTRA or intermittent short-course ICS at onset of illness	Symptoms not well-controlled or ≥ 1 severe exacerbation in a year
Step 3	Double 'low dose' ICS	Consider specialist referral	Asthma not well-controlled on low dose ICS
Step 4	Continue controller and refer for specialist assessment		Asthma not well-controlled on double ICS

Inhaler device selection

Choosing the right device is critical for effective delivery in young children.

- Age 0–3 Years: Preferred device is a pMDI plus a dedicated spacer with a face mask.
- Age 4–5 Years: Preferred device is a pMDI plus a dedicated spacer with a mouthpiece.

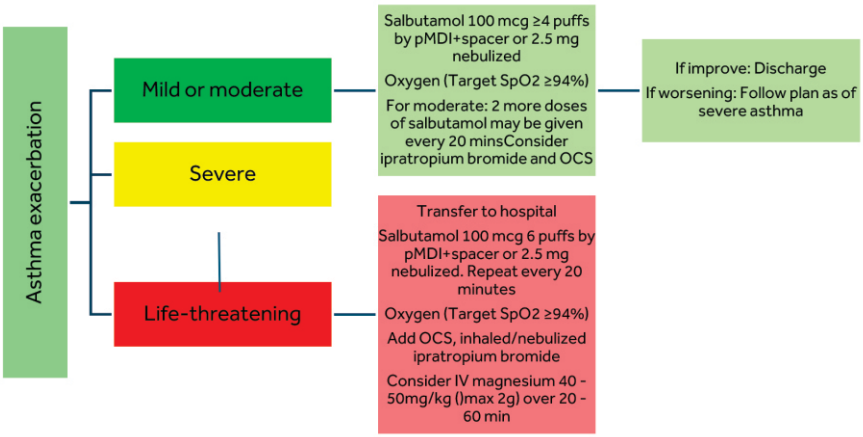
Exacerbation of asthma in children (≤ 5 years)

Table 2: Classification of asthma exacerbation in children (≤ 5 years)

Symptoms	Mild	Severe
Altered consciousness	No	Agitated, confused or drowsy
Oximetry (SaO2)	>94%	<92%
Speech	Sentences	Words
Pulse rate	<100 beats/minute	>180 (0–3 yrs) or >150 (4–5 yrs)
Respiratory rate	40/minute	>40/minute
Accessory muscle use	Absent	Present (retractions/nasal flaring)
Central cyanosis	Absent	Likely to be present
Wheeze intensity	Variable	Chest may be quiet

Management of acute asthma in primary care

Figure 2: Management of asthma exacerbation in primary care (children ≤ 5 years)



Indications of immediate transfer to hospital

Immediate transfer to hospital is indicated if a child ≤5 years with asthma has ANY of the following:

1. Child is unable to speak or drink
2. Cyanosis
3. Respiratory rate >40 per minute

4. Oxygen saturation <92% when breathing room air
5. Quiet chest on auscultation
6. Lack of response to initial bronchodilator treatment:
7. Lack of response to 12 puffs of inhaled salbutamol, administered as 4 separate puffs every 20 minutes for 3 times, over 1 hour
8. Persisting tachypnea despite three administrations of inhaled salbutamol, even if the child shows other clinical signs of improvement
9. That limits delivery of acute treatment, or parent/caregiver unable to manage asthma at home.

Management of acute asthma in emergency department

Table 3: Management of asthma exacerbation in emergency department (children ≤ 5 years)

Therapy	Dose and administration
Supplemental oxygen	Delivered by nasal prongs or mask (target oxygen saturation at ≥94%
SABA	4 or more puffs of salbutamol by spacer, or 2.5 mg by nebulizer. For moderate or severe exacerbation, consider giving SABA every 20 minutes for 3 doses, then reassess severity. If symptoms persist or deteriorate, give an additional 4 puffs or more per hour.
Systemic corticosteroids	For moderate or severe exacerbation, give initial dose of oral prednisolone (1–2 mg/kg up to a maximum 20 mg for children <2 years old; 30 mg for children 2–5 years) OR oral dexamethasone 0.3–0.6 mg/kg (max 12 mg) OR intravenous methylprednisolone 1 mg/kg 6-hourly on day 1
Ipratropium bromide	For moderately severe or severe exacerbation, give 4 puffs of 20 mcg ipratropium bromide by pMDI and spacer or 250 mcg by nebulization every 20 minutes with SABA for 3 doses. For mild exacerbation, if poor response to SABA in the initial hour, consider adding ipratropium as described above (if not already given).
Magnesium sulfate	Consider intravenous isotonic magnesium sulfate (40–50 mg/kg, maximum 2 g over 10–20 minutes) for children aged ≥2 years with severe exacerbation

Reference

1.Global Initiative for Asthma. Global Strategy for Asthma Management and Prevention, 2025. Updated November 2025. Available from: https://www.ginasthma.org

Appendix

A. Asthma action plan

For: _____ Doctor: _____ Date: _____

Doctor's Phone Number: _____ Hospital/Emergency Department Phone Number: _____

GREEN ZONE

DOING WELL

- No cough, wheeze, chest tightness, or shortness of breath during the day or night
- Can do usual activities

And, if a peak flow meter is used,

Peak flow: more than _____
(80 percent or more of my best peak flow)

My best peak flow is: _____

Daily Medications

Medicine	How much to take	When to take it
_____	_____	_____
_____	_____	_____
_____	_____	_____
_____	_____	_____
_____	_____	_____
Before exercise ☐ _____	☐ 2 or ☐ 4 puffs	5 minutes before exercise

YELLOW ZONE

ASTHMA IS GETTING WORSE

- Cough, wheeze, chest tightness, or shortness of breath, or
- Waking at night due to asthma, or
- Can do some, but not all, usual activities

–Or–

Peak flow: _____to _____
(50 to 79 percent of my best peak flow)

1st **Add: quick-relief medicine—and keep taking your GREEN ZONE medicine.**

_____ (quick-relief medicine) _____ Number of puffs **or** ☐ Nebulizer, once — Can repeat every _____ minutes up to maximum of _____ doses

2nd **If your symptoms (and peak flow, if used) return to GREEN ZONE after 1 hour of above treatment:**

☐ Continue monitoring to be sure you stay in the green zone.

–Or–

If your symptoms (and peak flow, if used) do not return to GREEN ZONE after 1 hour of above treatment:

☐ Take: _____ (quick-relief medicine) _____ Number of puffs **or** ☐ Nebulizer

☐ Add: _____ (oral steroid) mg per day For _____ (3–10) days

☐ Call the doctor ☐ before/ ☐ within_____ hours after taking the oral steroid.

RED ZONE

MEDICAL ALERT!

- Very short of breath, or
- Quick-relief medicines have not helped,
- Cannot do usual activities, or
- Symptoms are same or get worse after 24 hours in Yellow Zone

–Or–

Peak flow: less than _____
(50 percent of my best peak flow)

Take this medicine:

☐ _____ (quick-relief medicine) _____ Number of puffs **or** ☐ Nebulizer

☐ _____ (oral steroid) mg

Then call your doctor NOW. Go to the hospital or call an ambulance if:

- You are still in the red zone after 15 minutes AND
- You have not reached your doctor.

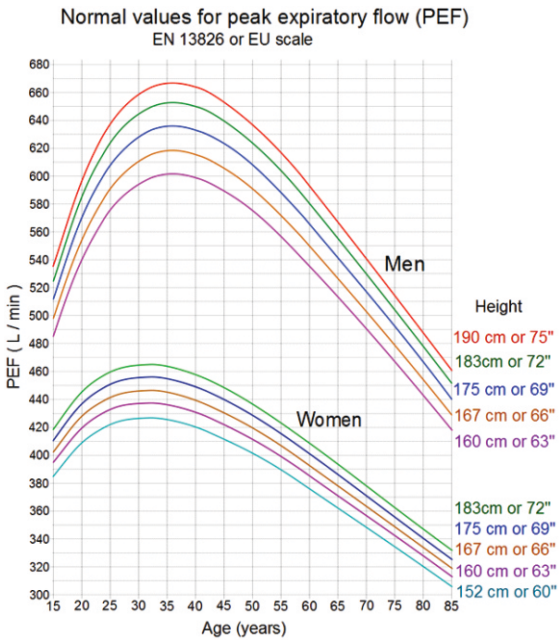
DANGER SIGNS

- **Trouble walking and talking due to shortness of breath**
- **Lips or fingernails are blue**

- **Take _____ puffs of _____ (quick relief medicine) AND**
- **Go to the hospital or call for an ambulance _____ (phone) NOW!**

See the reverse side for things you can do to avoid your asthma triggers.

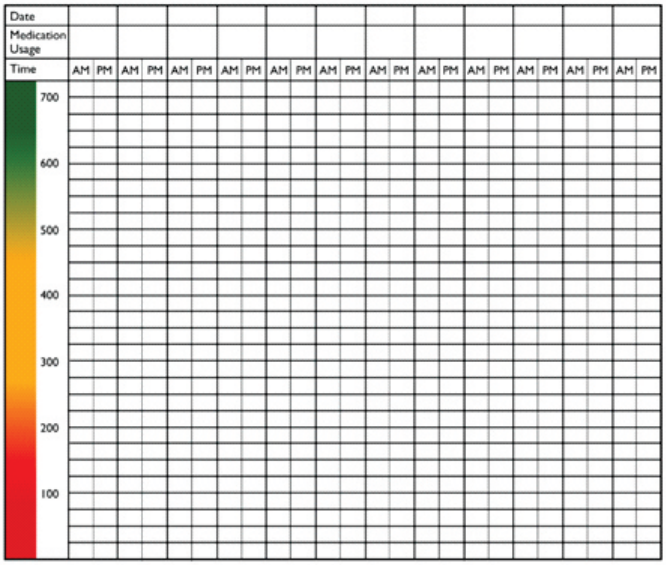
B. Peak expiratory flow rate normal values



C. Peak expiratory flow rate diary

Peak Flow Tracking Chart

Peak flow monitoring is part of the ongoing process of managing your asthma. Tracking your peak flow helps you become aware of the changes in symptoms, triggers, and even in the way your body responds to medication. Keeping these records can help you work with your doctor to keep your asthma action plan working effectively.



Name _____

Ideal peak flow number:

Know the early warning signs of a sudden asthma attack:

- Shortness of breath
- Tightness in your chest
- Difficulty breathing
- Wheezing
- Coughing
- Drop in peak flow number

Special Instructions

Use this space to write down any special information or instructions you receive from your doctor.

_____
_____
_____
_____
_____
_____
_____
_____
_____

D. Asthma medicine side effects

Drug Class	Examples	Common Side Effects	Potential Serious/Long-term Effects	Clinical Monitoring/Tips
ICS	Budesonide Fluticasone Beclometasone Ciclesonide Mometasone Trimacenilone	Oral thrush (candidiasis), hoarseness of voice (dysphonia), sore throat.	Skin bruising, increased risk of pneumonia (rare at low doses), slight growth velocity reduction in children (Step 3+).	Advise patient to rinse mouth/gargle after every use. Use a spacer with pMDIs.
SABA	Salbutamol Albuterol Levalbuterol Terbutaline	Tremors (shaky hands), palpitations, tachycardia (fast heart rate), headache.	Hypokalemia (low potassium) with high/frequent doses, cardiac arrhythmias.	Avoid overuse (= 3 canisters/year indicates poor control).

SAMA	Ipratropium bromide	Dry mouth, bitter taste, throat irritation, cough, headache, dizziness.	Acute angle-closure glaucoma (if mist enters eyes), urinary retention, blurred vision, paradoxical bronchospasm.	Use a mouthpiece instead of a face mask during nebulization to avoid eye exposure. Monitor patients with known prostatic hypertrophy.
LABA	Formoterol Salmeterol Vilanterol Indacaterol	Similar to SABA: Mild tremors, headache, palpitations.	Risk of severe exacerbation if used without ICS.	Never prescribe LABA as monotherapy; must always be combined with ICS.
LTRA	Montelukast Zafirlukast	Headache, abdominal pain, flu-like symptoms.	Neuropsychiatric effects: Sleep disturbances, agitation, depression, or suicidal ideation.	Box Warning: Screen for mood changes, especially in children and adolescents.
LAMA	Tiotropium Umeclidinium Glycopyrronium Aclidinium	Dry mouth, blurred vision (if mist enters eyes), urinary retention (rare).	Narrow-angle glaucoma exacerbation.	Primarily used as "add-on" therapy in Step 4 or 5.
OCS	Prednisolone Dexamethasone	Increased appetite, weight gain, mood swings, insomnia, dyspepsia.	Adrenal suppression, osteoporosis, cataracts, glaucoma, diabetes, hypertension.	Limit use to short "bursts" (3–5 days) for exacerbations to minimize risks.
Biologics	Omalizumab Mepolizumab Benralizumab Dupilumab Tezepelumab	Injection site reactions (pain/redness), headache, fatigue.	Anaphylaxis (rare), hypersensitivity reactions.	Must be administered in a clinical setting with post-injection monitoring.