Bronchial Asthma

Objectives:

- Know the basic definition.
- Learn some epidemiology.
- Know Pathophysiology and types.
- Explain methods of diagnosis.
- Discuss treatment options.
- Follow prevention measures.

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Recourses: 435 team + Davidson + Kumar + Recall question step up to medicine

- Editing file
- Feedback

Bronchial Asthma:

Asthma is a **chronic, heterogeneous inflammatory disorder** of the airway that has three characteristics:

1. **Airway hyper-responsiveness** that causes **recurrent episodes of coughing, wheezing[1], breathlessness, and chest tightness,** which are typically **worse at night and in early morning.**
2. A widespread but variable **airflow obstruction[2],** which is **reversible** either spontaneously or with treatment.
3. **Inflammation of the bronchi** where many cellular elements play a role, in particular: **T lymphocytes, mast cells, eosinophils, neutrophils, macrophages, and epithelial cells,** associated with plasma exudation, oedema, smooth muscle hypertrophy, matrix deposition, mucus plugging and epithelial damage.

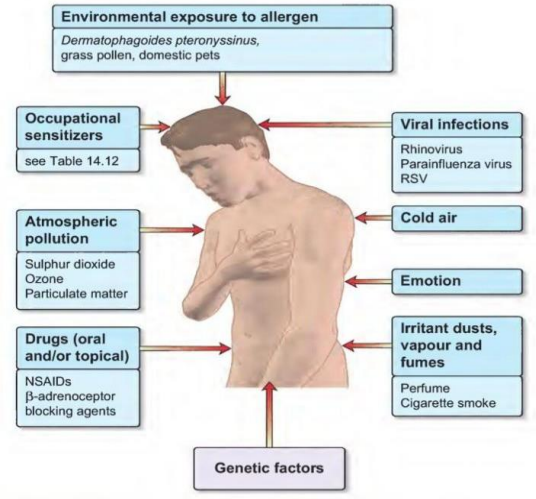
#If someone is breathless all the time it's unlikely to be Asthma and It might was just chest infection in the first place.

Epidemiology:

- Start at any age, (75% Dx age <7 "childhood"). يمكن ما يجيك المريض بهذا العمر بس لو أخذت منه هيستوري كويس حتعرف إنه كان عنده أعراض بعمر مبكر
- Remission around **puberty**.
- Prevalence is **increasing** with wide geographical variation (4-25%, more common in more developed countries) and higher prevalence in **females.** More than 95% are poorly or partially uncontrolled.
- Likely Multifactorial.
- **Environmental factors** are critically important in the development and expression of the disease.
- Asthma causes significant **morbidity and cost** to healthcare resources (10%) especially severe asthma. Those pts are seen frequently.

Etiology:

Although asthma is **multifactorial** in origin "genetic+environmental", inflammation is believed to be the cornerstone of the disease and is thought to result from **inappropriate immune responses** to a variety of antigens in genetically **susceptible individuals.**



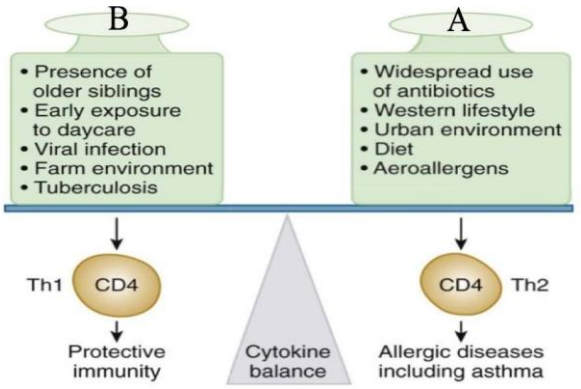
[1] Not every wheezes is asthma, e.g. In HF pulmonary edema, pt will have a lots of wheezing "cardiac asthma". Cardiac asthma is not a form of asthma. It's a type of coughing or wheezing that occurs with left heart failure.

[2] In chronic asthma, inflammation may be accompanied by irreversible airflow limitation as a result of airway wall remodeling that may involve large and small airways and mucus impaction. Kumar 7th edition p 846.

Causes:

- **Atopy and allergy**[3]
- **Genetics**
- Smoking "controversial[4]"
- Obesity "under investigation". Some patients who were obese and asthmatic got bariatric surgery and their symptoms disappeared.
- **Hygiene Hypothesis:**

If a child has a more contaminated environment he gains stronger immunity. While having a less contaminated environment (western lifestyle) may predispose to asthma. This hypothesis has role in SLE and other immune diseases.



A- in developed societies (use of antibiotics, reduced exposure to pathogens "clean environment" may predispose towards an IgE response to **allergens (Th2 pathway).**
B- growing up in "dirty environment" allow the immune system to avoid developing allergic response. "organism component > immune response toward the **protective pathway (Th1) and Treg".**
Kumar 7th edition p 847

This proposes that infections in early life are critically important in maturation and bias of the immune system against the development of allergies. It is suggested that the high prevalence of allergic disease is the penalty for the decreased exposure to infection that has resulted from improvements in sanitation and health care!! Older people that had contact with microallergant a lot, they have stronger immunity than younger people who clean their houses with anti-bacterial per day and take a shower 3 times a day.

- A number of factors predispose to allergic diseases, the strongest of which is a **family history**[5] and **Contributory environmental factors**[6].

○ Asthma due to specific triggers:

◇ Occupational asthma:

- accounting for 15% of all asthma cases
- The risk of developing some forms of occupational asthma increases in smokers.
- The proportion of employees developing occupational asthma depends primarily upon the level of exposure.
- Proper enclosure of industrial processes or appropriate ventilation greatly reduces the risk.
- Atopic[7] individuals develop occupational asthma more rapidly when exposed to agents causing the development of specific IgE antibody.

Table 14.12 Occupational asthma

Cause	Source/Occupation
Low molecular weight (non-IgE related)	
Isocyanates	Polyurethane varnishes Industrial coatings Spray painting
Colophony fumes	Soldering/welders Electronics industry
Wood dust	
Drugs	
Bleaches and dyes	
Complex metal salts, e.g. nickel, platinum, chromium	
High molecular weight (IgE related)	
Allergens from animals and insects	Farmers, workers in poultry and seafood processing industry; laboratory workers
Antidotes	Nurses, health industry
Latex	Health workers
Proteolytic enzymes	Manufacture (but not use) of 'biological' washing powders
Complex salts of platinum	Metal refining
Acid anhydrides and polyamine hardening agents	Industrial coatings

- Non-atopic individuals can also develop asthma when exposed to such agents, but after a longer period of exposure.

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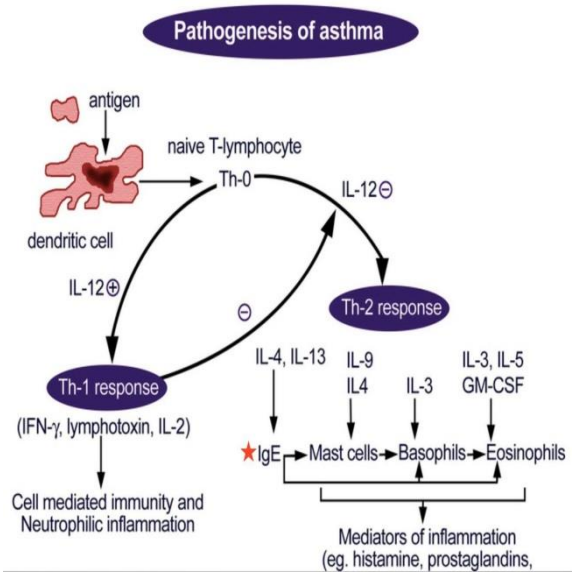
[3] Triggers include pollens, house dust, molds, cockroaches, cats, dogs, cold air, viral infections, smoking, **medications (β-blockers, aspirin and NSAIDs)**, and **exercise**.
4 Giving rise or likely to give rise to controversy or public disagreement.
[5] Genes controlling innate immune responses, cytokine production, IgE levels and the ability of the epithelial barrier to protect against environmental agents.
[6] Include bacterial and viral infection, pollutants and cigarette smoke. See davidson p89.
7 The propensity to produce IgE in response to allergens.

Pathophysiology: Pathophysiology of asthma: 7:45

The pathogenesis of asthma is complex and not fully understood. It involves a number of cells, mediators, nerves and several different mechanisms, of which exposure to allergens is among the most significant.

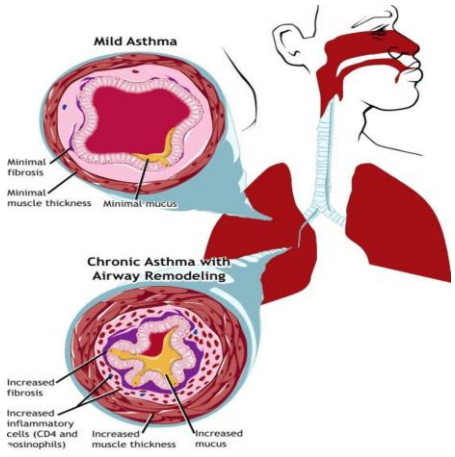
- Cells involved are **dendritic cells, CD4 Th2 lymphocytes** **"**abundant in the mucous membranes of the airways and the alveoli." the release of their cytokines plays a key part in the inflammation process.

Type of cytokine: Very important to know when it comes to treatment.	IL13, IL9, IL4, IL3	IL-3, IL-5, GM-CSF[8]
The activated cell:	**Mast cell** "Release histamine and other cytokines"	**Eosinophils** "Release basic proteins and leukotriene"
The effect of activated cell:	On the smooth muscles and mucosa of the airway, blood vessels and sensory nerves. - contribute in both immediate and late asthmatic reaction	On the bronchial wall and secretions. - Sputum eosinophilia.



The varying clinical severity and chronicity of asthma is dependent on interplay between airway inflammation and airway wall remodeling.

- **Remodeling** occurs to the epithelium, basement membrane, smooth muscles, and nerves:
  - Smooth muscles become **thickened** and, **mucosa swollen** with **increased secretion,** leading to fixed narrowing of the airway and a reduced response to bronchodilator medication.
  - The epithelium becomes damaged replaced by **fibrosis**, basement membrane thickened and the nerves cause more irritability.



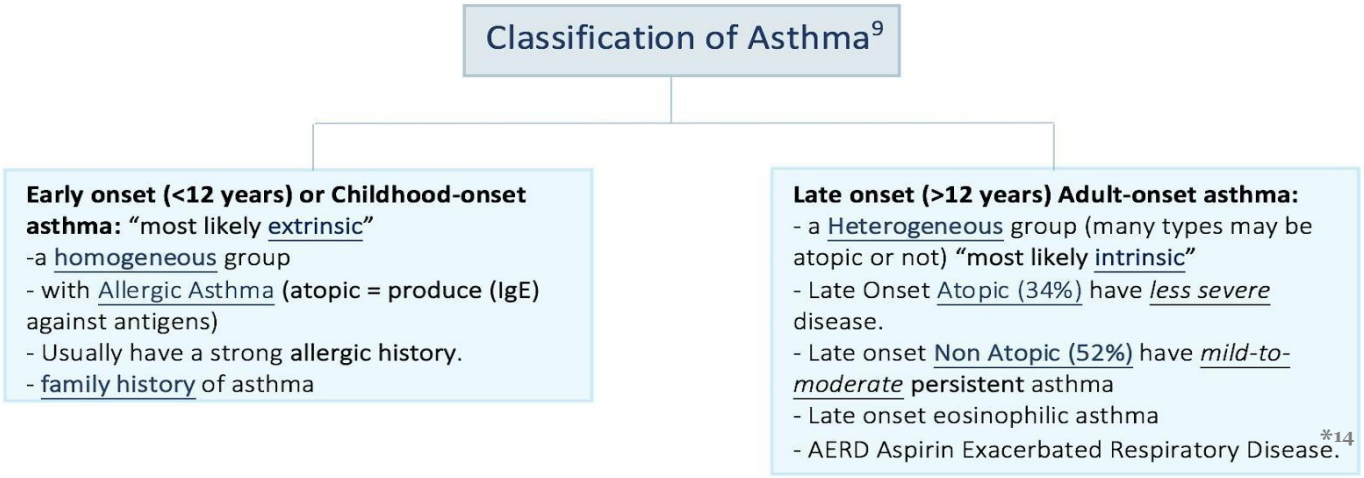
We can notice that the airway is similar to normal from the outside, but narrowed from the inside with increase in mucus secretions and inflammatory cells.

8 Granulocyte-macrophage colony-stimulating factor (GM-CSF), also known as colony-stimulating factor 2 (CSF2), is a monomeric glycoprotein secreted by macrophages, T cells, mast cells, natural killer cells, endothelial cells and fibroblasts that functions as a cytokine.

Clinical features:

- **Typical symptoms** include, recurrent[9] episodes "intermittent" of: **wheezing**, chest tightness, breathlessness and cough[10], in **diurnal pattern[11].**
    - Usually occur within 30 minutes of exposure to triggers.
    - There is a symptoms fluctuation over the course of one day, or from day to day or month to month.
    - Poorly controlled, symptoms such as cough and wheeze disturb sleep and have led to the term 'nocturnal asthma'.
        - **Classical precipitants** include exercise, particularly in cold weather[12], exposure to airborne allergens or pollutants, dust, viral upper respiratory tract infections, and medications[13].

Classification:



★ Types:

Different Pts. come to you with the same symptoms of asthma, if you in were 10 years ago you will give all of them the same treatment which is steroids. But now things have changed and we know different types of asthma "allergic, eosinophilic, neutrophilic.. etc." that will change the drug of choice depending on it.

- **Phenotype:** all the clinical characteristics that you can get from the history. Ex: is it late onset, early onset.

Is a **clinical character** like: age, gender, obesity, smoking and even the **pathophysiology**.

- **Endotype:** refer to test with multiple antigens. Things you can measure. Ex: skin test, IgE, eosinophil count, nitric oxid count which is increase in any inflammation.
    - **Precision medicine (personalized medicine).**
    - Not treating as one asthma, instead **treat according to the type** of asthma (eosinophilic, allergic, etc…), **WHY? Because of different pathophysiological mechanism.**

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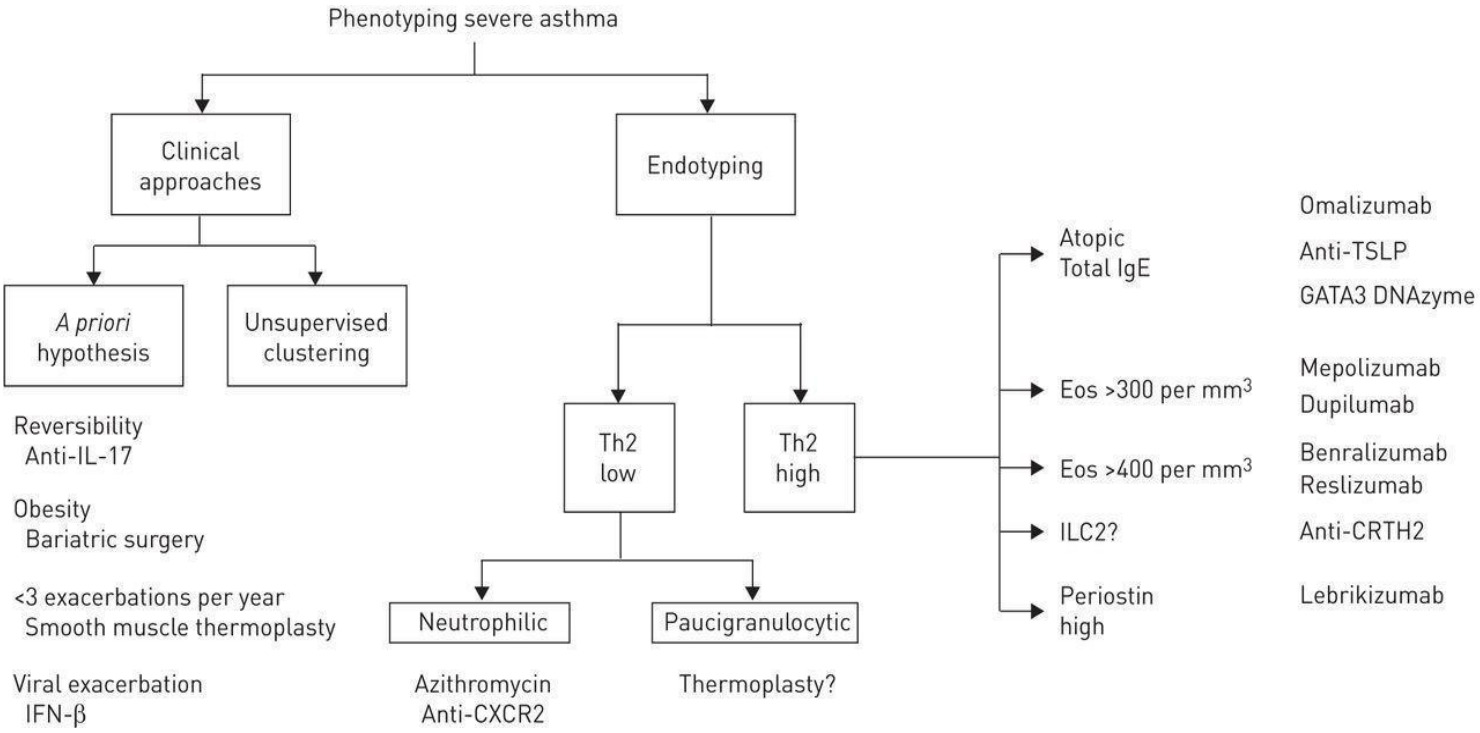
[9] Commonly mistaken for a cold or chest infection which is taking time to resolve.
[10] When Cough is the dominant symptom in some patients, and the lack of wheeze or breathlessness may lead to a delay in reaching the diagnosis of so-called 'cough-variant asthma'.
[11] Symptoms and lung function being worse at night and in the early morning.
[12] winter sports enthusiasts
[13] (β-blockers, aspirin and NSAIDs).

- **Mixed or overlapping features:**

Some with eosinophilic asthma may have allergic component as well, like aspirin sensitive asthma.



Methods of diagnosis

1- History:

- Symptoms: SOB, Wheeze, chest tightness, usually dry Cough, Sputum small mucoid.
- If symptoms are episodic, and triggers**.**

Initial structured clinical assessment of asthma
Factors to consider in an initial structured clinical assessment include: • **Episodic symptoms:** ○ More than one symptoms of asthma occur in episode, With period of no (or minimal) symptoms between the episode, e.g.: - Recurrent intermittent episode of triggered symptoms by any allergen or medication. - Acute wheezing with improvement after treatment. ○ Monitor **FEV_1 or PEF** during and after the episode, if **lower** "during" than "after" > obstructive nature. • **Wheezing:** ○ Repeated normal examination during the episode will decrease the probability of asthma. • **Evidence of diurnal variability.** • **Atopic history:** ○ **PHx** of: eczema, allergic rhinitis, or **FHx** of asthma, ○ **Record** of: raised antigen-specific IgE level, positive skin-prick test to aeroallergen or **blood eosinophilia**. Absence of symptoms, signs or clinical Hx suggest **alternative diagnosis.**

Clinical Feature that ⇑ the probability of asthma	Clinical Feature that ⇓ the probability of asthma
○ **More than one of the following symptoms:** • **wheeze, breathlessness, chest tightness and cough, particularly if:** **- symptoms worse at night and in the early morning** **- symptoms in response to exercise, allergen exposure** **- symptoms after taking aspirin or beta blockers.** ○ **History of atopic disorder,** ○ **Family history of asthma and/or atopic disorder** ○ **Widespread wheeze heard on auscultation of the chest** ○ **Otherwise unexplained low FEV, or PEF (historical or serial readings)** ○ **Otherwise unexplained peripheral blood eosinophilia.**	○ **Prominent dizziness, light-headedness peripheral tingling** ○ **Chronic productive cough in the absence of wheeze or breathless** ○ **Repeatedly normal physical examination of chest when symptomatic** ○ **Voice disturbance** ○ **Symptoms with colds only** ○ **Significant smoking history (i.e. > 20 pack- years)** ○ **Cardiac disease** **Normal PEF or spirometry when symptomatic**

◆ [14]Differential Diagnosis:

- Other Illness with **wheezing**[15] **/ SOB:**

  - **COPD (Smoker)**, - Bronchiectasis (Large amount of sputum), - Heart failure / Pulmonary Edema, - Airway obstruction (Tumors, FB), - Vocal cord dysfunction

- May **Coexist** and complicate Dx of asthma:

  - GERD, - OSA (obstructive sleep apnea), - ABPA (Allergic bronchopulmonary aspergillosis)

[14] "If I'm going to put a third year MCQs I will concentrate on HISTORY, HISTORY, HISTORY. What makes asthma more or less likely".

[15] No wheezing either means no asthma or the pt. in his good day, it should be heard if pt. was in the episode, unless he is having a silent chest. "remember for any rule there is exceptions"

2- Examination

Upper respiratory tract	nasal secretion, Nasal dripling, mucosal swelling, **nasal polyp**[16] "common in aspirin sensitive asthma or eosinophilic"
Chest	**Wheezing*** or prolonged phase of forced exhalation, Chest hyper-expansion, accessory muscles.
Skin	atopic dermatitis, eczema.

* Wheezing is a **high-pitched** whistling[17] sounds when breathing out "expiration".

- **A lack of wheezing and a normal chest examination do not exclude asthma**.
- wheezing sometimes is not present , so we could say whenever the patient gets exertion he will develop SOB and when he relax the symptom **will not improve**. Why? Because the **inflammation is still there in the lung**. Especially in obese.
    - If symptoms at late night or early morning indicate worse condition because it is the time for circadian rhythm of airflow.
    - We forget to do UPPER RESPIRATORY TRACT EXAMINATION most of the time, but it's important!
    - If eczema or dermatitis it indicate allergic reaction in the body, and don't forget to check the lower extremities as well.

- Assume the pt. Is obese and he came to you with SOB, how to know whether it is due to obesity or asthma:
    1. Pt. will describe the symptom with association to walking, and it will stop after rest. "Asthma is an inflammation once it comes it should not go away.
    2. Listen to their chest if it is clear "no wheezing" that would probably be due to the extra weight.

  We have to be casueouse in such case18.

3- Investigations:

Asthma Dx – **variable airflow limitation:**

- Confirm presence of **airflow limitation:**
    – Document that FEV1/FVC[19] is reduced <0.75 (at least once).
- Confirm variation in lung function or **Reversibility Hyperresponsiveness:**
    – Excessive bronchodilator reversibility (FEV1 >12% and >200mL)
    – Excessive diurnal variability twice-daily PEF monitoring. When you check the graph you will notice a significant increase compared to the morning.

[16] it's noncamcerous growth within the nose
[17] صفير
[18] There is a phenotype that's called obese female phenotype, they are symptomatic and their asthma is worse than the other phenotype because obesity considered a proinflammatory condition.
[19] Forced Expiratory Volume in One Second/**Forced Vital Capacity**.

Pulmonary function test (PFT):

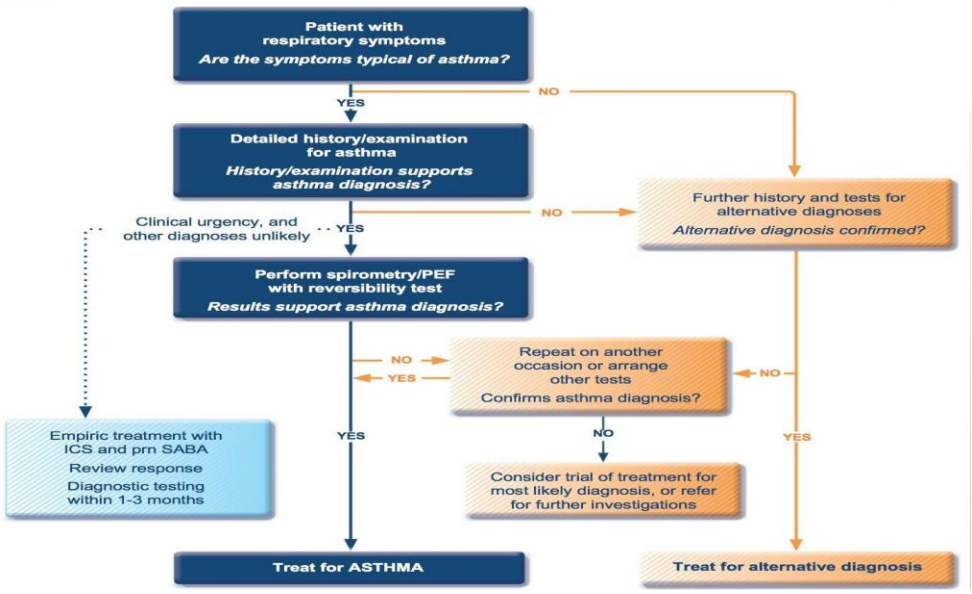
Required[20] for diagnosis, it shows: **1. Obstructive pattern 2. Decrease in expiratory flow rates, 3. Decreased FEV1 and decreased FEV1/FVC ratio (<0.70).**

1. Spirometry (routine). Every pt must do it.	- Shows decrease in the **FEV1 and FVC ratio.** - Diagnosing and assessing reversibility[21] If inhalation of a bronchodilator (β2-agonist) results in an **increase in FEV1 or FVC by at least 15% (200ml),** airflow obstruction is considered reversible.
2.Peak expiratory flow rate (PEF)[22]	- Measure of airflow obstruction by the pt, "self-monitoring ". - A **diurnal** variation in PEF of more than **20%** (the lowest values typically being recorded in the morning) is considered diagnostic[23].

Others[24]:

Full Blood count (FBC)	I will do it for most of pt because it helps with phenotyping the pt. (either eosinophil or neutrophils)
Arterial blood gases	- patients with an asthma attack > increased respiratory rate, so **PaCO2 decreases**[25]
Chest X-ray	**Normal in mild cases**; severe asthma reveals hyperinflation. - Only necessary in severe asthma **to exclude other conditions** (e.g., pneumonia, pneumothorax, pneumomediastinum, CHF and foreign body).
Bronchoprovocation test	- The patient is given a trigger. If a reaction occurs, he is probably asthmatic. **Useful when the main symptom is cough**.
Measurement of allergic status	- **Skin tests**: done in all asthmatic patients to identify allergen. - Total and allergen-specific **IgE**. A full blood picture may show the peripheral **blood eosinophilia.**

Diagnostic Approach:



[20] The most accurate diagnostic test.
[21] Spirometry with a bronchodilator can confirm diagnosis by proving reversible airway obstruction.
[22] **If spirometry not available.**
[23] Diurnal variability on 2 sets of PEF indicates reversibility.
[24] Exercise test: in children.
[25] Increased PaCO2 is a sign of respiratory muscle fatigue or severe airway obstruction.

- Do the spirometry test before and after bronchodilators to see if it gets better.
- If spirometry test is normal do bronchial provocation test.
- We do full lung function test because there might be different pattern of the disease.
- We do full blood count to look for anemia or eosinophilia, because anemia may cause these kind of symptoms.
- In airway hyper-responsiveness test we give the patient histamine on something to trigger allergic response, and measure by spirometry before and after and compare. Whatever you give it should be something you can control. EXERCISE can do same effect as an allergen.
- RHINITIS will explain what is happening inside the respiratory tract.
- In airway hyper- responsiveness we expose them to an allergen and if the exposure causes bronchospasm then it is +ve test.

Management

- **Education:**
  - Compliance • Inhalers techniques especially elderly • Asthma Action plans. *Specific directions for daily management and for adjusting medications in response to increasing symptoms or decreasing PEFR.*
- **Control of environmental factors:**
  - **Triggers** (Aeroallergens, Irritants),
  - **Comorbid conditions** (Obesity, GERD, Rhinitis, ABPA, VCD, stress),
  - **Medications** (Aspirin, Beta Blockers),
  - **Infections** (Vaccinations).
- **Pharmacological:**

1. Aim[26]:

The aim of asthma management is control of the disease. **Complete control is defined as:**
- **no** daytime symptoms, **no** night time awakening due to asthma, - **no** need for rescue medication, **no** asthma attacks, - **no** limitations on activity including exercise, - **normal** lung function (in practical terms EV, and/or PEF >80% predicted or best), - **minimal** side effects from medication.

2. Assessment:

- **Assessment of risk factors for poor asthma outcomes & Approach of**

Independent risk factors include:
• Ever intubated for asthma • Uncontrolled asthma symptoms • Having ≥1 exacerbation in last 12 months • Low FEV1 (measure lung function at start of treatment, at 3-6 months to assess personal best, and periodically thereafter) • Incorrect inhaler technique and/or poor adherence • Smoking • Elevated FeNO in adults with allergic asthma • Obesity, pregnancy, blood eosinophilia

*independent to the level of symptom control, GINA 2017

Approach of management:
1. Start treatment at the level most appropriate to initial severity. **2. Achieve early control.** **3. Maintain control by:** - increasing treatment as necessary - Decreasing treatment when control is good.
- Before initiating a new drug therapy practitioners should check adherence with existing therapies, check inhaler technique and eliminate trigger factors.

British guideline on the management of asthma Quick Reference Guide

[26] British guideline on the management of asthma Quick Reference Guide.

3. Treatment[27]:

Relievers:	Preventer:	Personalized Medicine:
– Short Acting Beta agonist	– Steroids[1] – Long acting Beta Agonist[2] and LAMA[2]*[27] – Leukotriene's receptors Antagonist – Theophylline	- Anti IgE or Anti IL5

1-for long-term management. 2- if asthma NOT controlled combine it with steroids

- Personalized medicine: medications prescribed specifically to each patient according to his needs, anti IgE is for allergic asthma and anti IL5 in eosinophilia.
- If the patient gets better we can reduce the medications.

Asthma is managed in a stepwise fashion of progressively adding more types of treatment if there is no response.

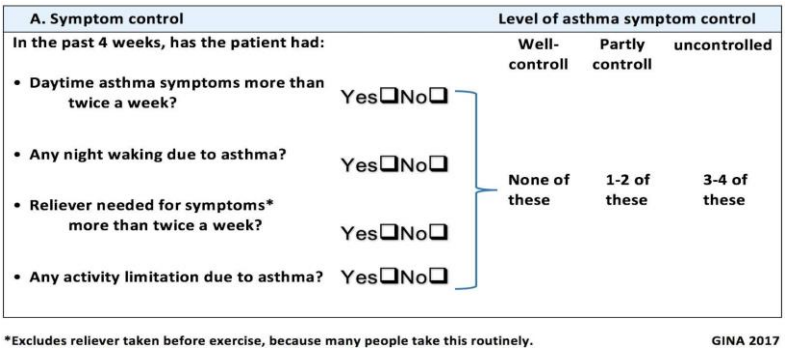
Step 1	**Always start the treatment of asthma with an inhaled short-acting beta agonist (SABA) as needed.** **Examples of SABA are:** · Albuterol. · Pirbuterol. · Levalbuterol.
Step 2	**Add a long-term control agent to a SABA. Low-dose inhaled corticosteroids (ICS) are the best initial long-term control agent.** ● **Example of ICS are:** Beclomethasone, budesonide, flunisolide, fluticasone, mometasone, triamcinolone. ● **Alternate long-term control agents include:** · Cromolyn and nedocromil to inhibit mast cell mediator release and eosinophil recruitment. · Theophylline. · Leukotriene modifiers: montelukast, zafirlukast, or zileuton (best with atopic patients).
Step 3	**Add a long-acting beta agonist (LABA) to a SABA and ICS, or increase the dose of the ICS. LABA medications are:** salmeterol vs formoterol.
Step 4	**Increase the dose of the ICS to maximum in addition to the LABA and SABA.**
Step 5	**Omalizumab may be added to the SABA, LABA, and ICS in those who have an increased IgE level.**
Step 6	**Oral corticosteroids such as *prednisone* are added when all the other therapies are not sufficient to control symptoms.**
Notes	- Adverse effects of inhaled steroids are dysphonia and oral candidiasis. - High-dose inhaled steroids rarely lead to the adverse effects associated with prednisone. - Never use LABA first or alone! - Anticholinergics role of ipratropium and tiotropium in asthma management is not clear. Anticholinergic agents will dilate bronchi and decrease secretions. They are very effective in COPD. - SEVER TREATMENT IV magnesium causes muscle relaxant.

[27] Long-acting muscarinic antagonists.

4. **Monitoring:**

• Symptoms, • Peak Flow (Home), • **Spirometry (Clinic),** • Novel FENO 'The more nitric oxide the more inflammation they have' and Sputum eosinophils, • Assess Severity and Control of asthma.

- GINA assessment of symptoms control:



A. Symptom control		Level of asthma symptom control		
In the past 4 weeks, has the patient had:		Well-controll	Partly controll	uncontrolled
• Daytime asthma symptoms more than twice a week?	Yes❑No❑	None of these	1-2 of these	3-4 of these
• Any night waking due to asthma?	Yes❑No❑			
• Reliever needed for symptoms* more than twice a week?	Yes❑No❑			
• Any activity limitation due to asthma?	Yes❑No❑			

*Excludes reliever taken before exercise, because many people take this routinely. GINA 2017

Prevention measures

Asthma Self-Management:

- Communicate and educate the patient with **a written asthma action plan** includes all the information you need to look after your asthma well, so you will fewer symptoms and significantly cut your risk of asthma attacks.
- Asthma is diagnosed on history (recurrent episodes of coughing, wheezing, breathlessness, and chest tightness often associated with allergic triggers), examination (wheeze) and lung function.

The most important measure to prevent the attack is to avoid triggering factors.

Acute severe asthma (Kumar & master the boards)

This is severe progressive asthmatic symptoms over a number of hours or days. It is a medical emergency that must be recognized and treated immediately at home with subsequent transfer to hospital. In the UK, 1400 patients die from asthma each year and 90% of these deaths are preventable by correct management.

Clinical features

Patients with acute severe asthma typically have:

- Inability to complete a sentence in one breath.
- Respiratory rate ≥ 25 breaths/min.

- Heart rate ≥ 110 beats/min.
- PEFR 33–50% of predicted normal or patient's best.

Life-threatening features are any one of the following in a patient with acute severe asthma:

- Silent chest, cyanosis or feeble respiratory effort.
- Exhaustion, altered conscious level.
- Bradycardia or hypotension.
- PEFR < 33% of predicted or best.
- PaO2 < 8 kPa.

Treatment Oxygen | Albuterol | Steroids.

The best initial therapy is **oxygen combined with inhaled short-acting beta agonists such as albuterol and a bolus of steroids**. Corticosteroids need 4 to 6 hours to begin to work, so give them right away.

- Epinephrine injections are no more effective than albuterol and have more adverse systemic effects.
- Ipratropium should be used, but does not work as rapidly as albuterol.
- Magnesium has some modest effect in bronchodilation. Magnesium is not as effective as albuterol, ipratropium, or steroids, but it does help.
- If the patient does not respond to oxygen, albuterol, and steroids or develops respiratory acidosis (increased pCO2), the patient may have to undergo endotracheal intubation for mechanical ventilation. These patients should be placed in the intensive care unit.

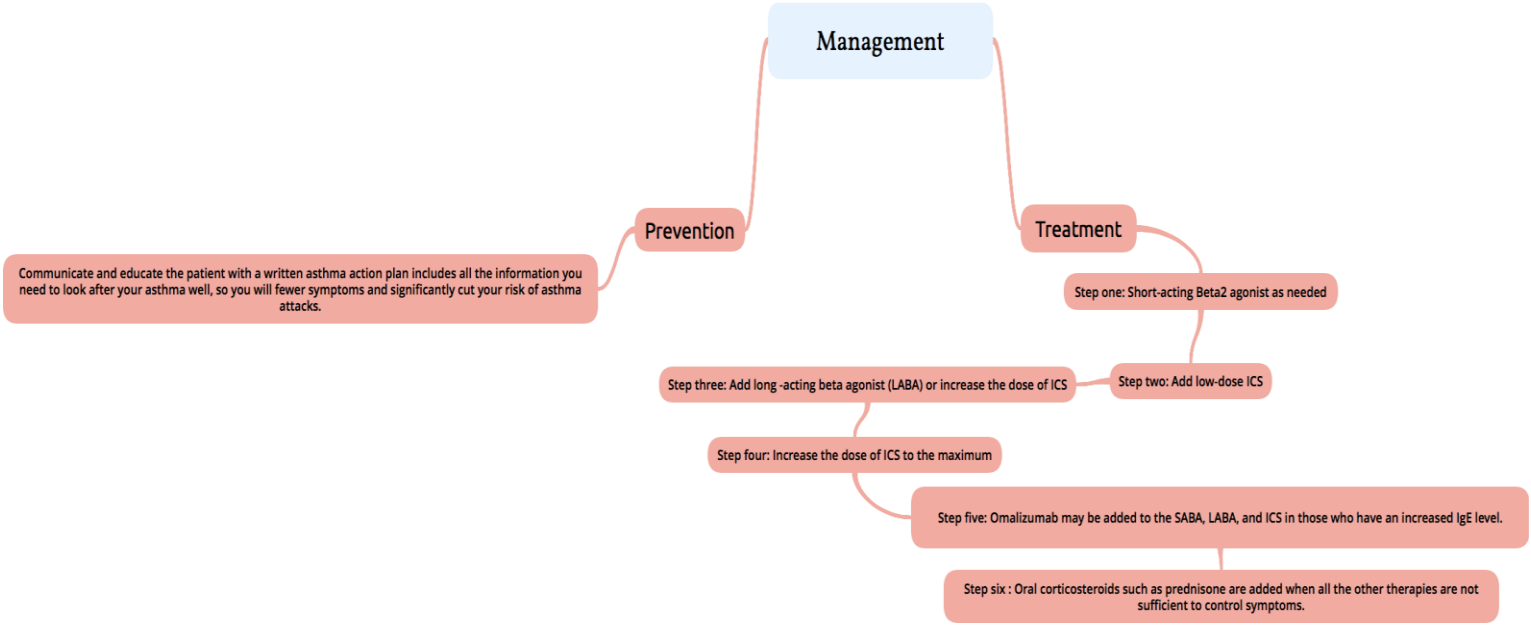
Summary

- **Diagnosis:**
  - Episodic Sx
  - Triggered
  - Wheeze
  - Atopic history

Clinical features that increase asthma probability	Clinical features that decrease asthma probability
Sx worsen at night and in early morning	Prominent dizziness, light-headedness, peripheral tingling
Sx in response to exercise, allergen exposure and cold air	Chronic productive cough, cough in the absence of wheeze or breathlessness
Sx after taking aspirin or beta blockers	Voice disturbance
Family Hx of asthma/atopic disorder	Sx with colds only
Otherwise unexplained peripheral blood eosinophilia	Cardiac disease
Otherwise unexplained low FEV1 or PEF	Significant Hx of smoking > 20 pack-years

- **Management**
  - Monitoring
  - Education
  - Control of environmental factors

- Pharmacologic
- Prevention



Questions

1- In Asthmatic Patient FEV1 is:
A. Increased. B. Normal. C. Reduced.

2- A patient with known asthma undergoing therapy with inhaled corticosteroid and intermittent (short-acting) β2-agonist presents with complaints of nocturnal awakenings secondary to cough and occasional wheezing. This episode occurs three to four times per week. Pulmonary function tests in the past have shown mild obstructive lung disease. Which of the following is the best next step?
A. Oral steroids B. Leukotriene inhibitors C. Long-acting β2-agonists
D. Theophylline E. Antireflux therapy

3- A 30-year-old athlete with asthma is also a cigarette smoker. Which of the following is characteristic of asthma but not other obstructive lung disease?
A. Hyperinflation is present on chest x-ray B. Airway obstruction is reversible
C. Hypoxia occurs as a consequence of ventilation-perfusion mismatch
D. The FEV1/FVC ratio is reduced

4- Which of the following doesn't indicate a poor prognostic finding in asthma?
A. Silent chest B. Hypercapnia C. Thoracoabdominal paradox (paradoxical respiration)
D. Pulsus paradoxus of 5 mm Hg E. Altered mental status

5- Symptoms of asthma are:
A. productive cough B. chest stabing C. breatlessness D. All above

6- 35 years old patient on SABA but symptoms not improving, what would you prescribe more?
A.ICS B.LABA C.LAMA D. Increase dose of SABA

7- Is voice disturbance a feature of Asthma?
A. Correct B. Wrong

8- In GINA assessment we ask about:
A. Night waking symptoms B. Activity limitation
C. Daytime symptoms more than twice a week D. all above

9- In management we start with:
A. medication B. Control environment C. Monitoring
D. Education