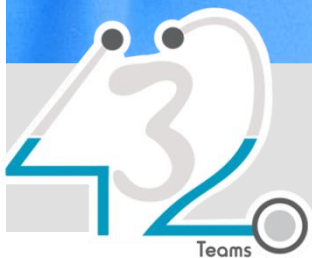



MEDICINE

432 Team

10 Bronchial Asthma



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COLOR GUIDE: • Females' Notes • Males' Notes • Important • Additional

Objectives

1. Know the basic definition
2. Learn some epidemiology
3. Explain methods of diagnosis
4. Discuss treatment options
5. Follow Prevention measures



<http://www.youtube.com/watch?v=7EDo9pUYvPE>

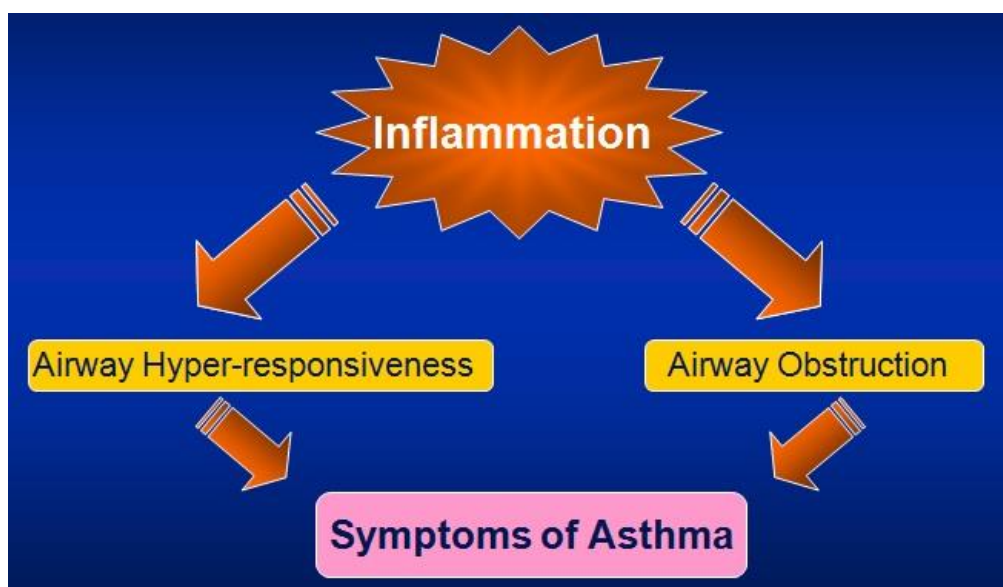
What is asthma?

- A chronic inflammatory disorder of the airway (with Infiltration of mast cells, eosinophils and lymphocytes in response to allergens).
- Airway hyper-responsiveness (twitchy airways). to a wide range of stimuli
- Recurrent episodes of wheezing, coughing and shortness of breath.
- Variable and often reversible airflow limitation (airway obstruction).

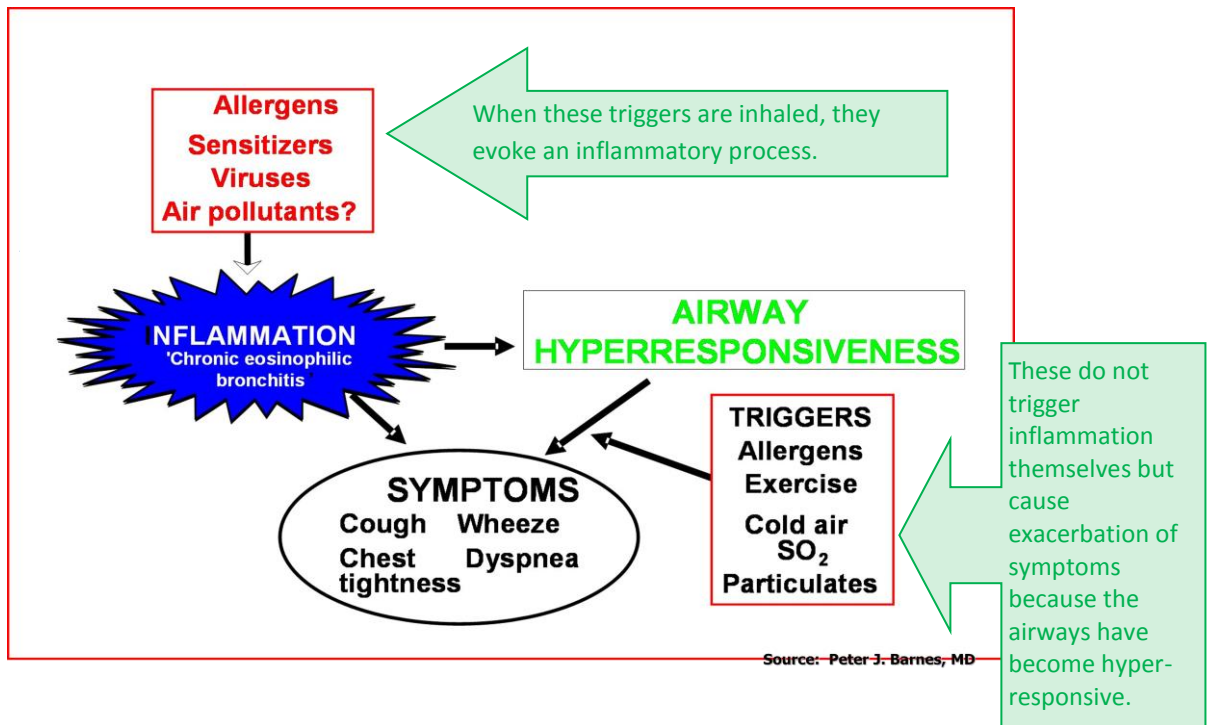
Asthma prevalence in Saudi Arabia:

- Children and Adolescents: 20 %
- Adults : 10 %

Pathology of Asthma:



- Airways become twitchy → easy to go into bronchospasm.
- Airway hyper-reactivity, the tendency for airways to narrow excessively in response to triggers that have little or no effect in normal individuals, is integral for the diagnosis of asthma. (Davidson).
- Airway obstruction by inflammatory process, edema and secretions. which is usually reversible spontaneously or with treatment



What makes an individual develop asthma and inflammation in response to such factors and another not?

Asthmatics are genetically programmed to become hyper-responsive to these triggers. Hence, asthma can be controlled but not cured.

Pathophysiology: Important

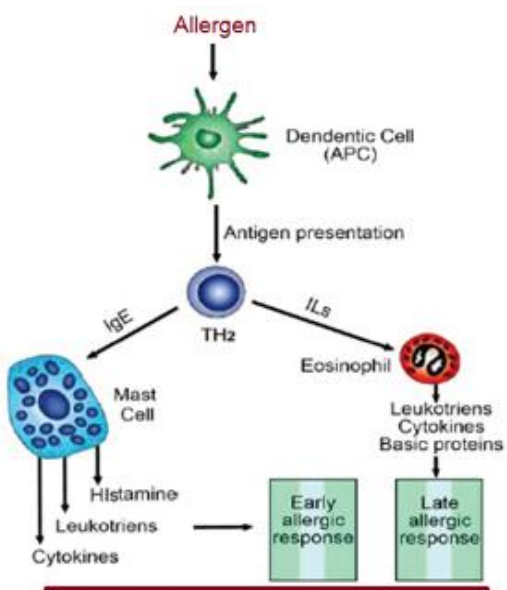
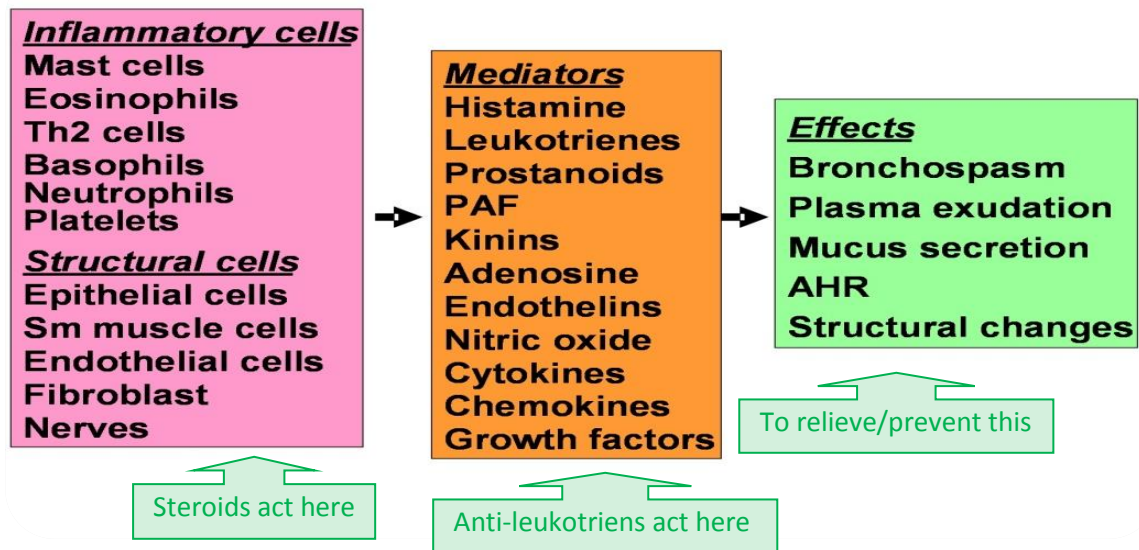


Fig. 1 : Pathophysiology of asthma
 APC = Antigen presenting cell
 ILs = Interleukins
 TH2 = T-lymphocyte Helper cell 2

1. Allergen (antigen) is engulfed by antigen presenting cells and then presented to T-lymphocytes.
2. TH2 stimulates two inflammatory pathways: Interleukins and IgE which will activate eosinophils and mast cells, respectively.
3. Mast cells degranulate and release inflammatory mediators resulting in an early allergic response (immediate).
4. Eosinophils take longer time to produce different types of mediators which induce a late allergic response (delayed). → Symptoms may appear after 6 hours from exposure.



The message from this picture is: Understanding pathophysiology is the basis for choosing the appropriate treatment.

*AHR = Airway Hyper-Responsiveness.

Allergic triggers:

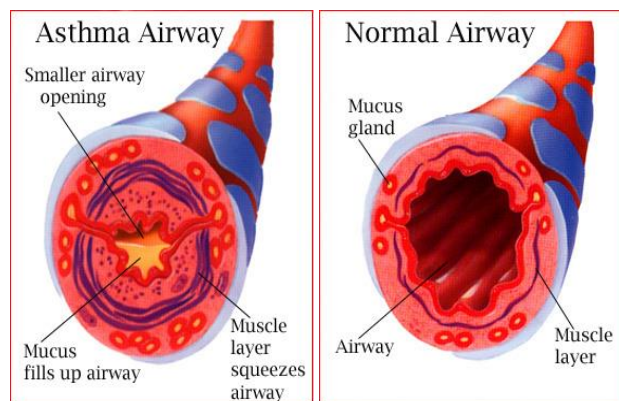
- Cat or dog dander.
- House dust mites.
- Cockroach allergen.
- Pollens.
- Fungi/molds.

Triggers of Asthma (Irritants): These are NOT allergens.

Exposure to these causes exacerbation of symptoms.

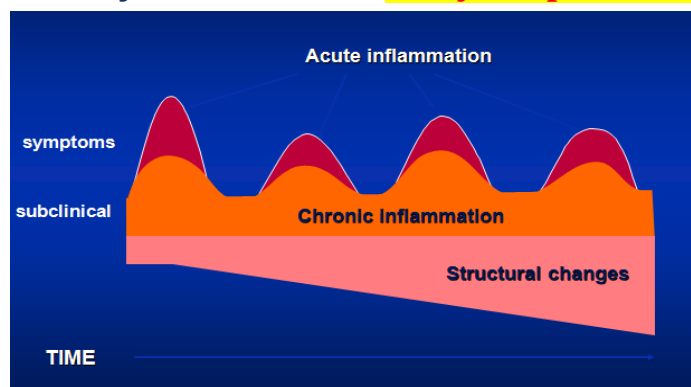
- Infections → Rhinitis, sinusitis, and viral infections.
- Chemicals.
- Diet/Medications → Aspirin sensitivity (NSAIDs), Sulfite sensitivity, Beta Blockers,
- Strong Emotions.
- Exercise.
- Cold temperature.
- Exposure to smoke.
- Dust.

When exposed to an asthma trigger:



- Bronchioles constrict to limit exposure to the trigger.
- Mucous membrane becomes irritated and swells.
- Mucous is produced to trap the irritant.
- Coughing initiated to pop open bronchioles and expel the mucous build-up.
- Air retention volume in alveolar sacs increases – can't get air out or in.
- CO₂ build-up in alveolar sacs and in system tissues which can lead to acidosis.
- The body attempts to blow off the excess CO₂ – rapid shallow breathing. (hyperventilation)
- Hungry for O₂ and trying to get rid of CO₂ at the same time.
- Fatigued muscles in this effort.
- If this continues, the person with asthma can die.

“Real Life” Variability in Asthma: **Very important**



Asthma is not a constant disease; it actually comes in recurrent episodes following exacerbations by triggers. There's an ongoing chronic inflammation in the airways of an asymptomatic asthmatic patient which worsens overtime and finally results in structural changes (if left untreated). Structural changes are irreversible and are mainly due to fibrosis leading to fixation of airways. Note that chronic inflammation will subside spontaneously in patients with mild asthma (infrequent/once per month attacks). The major issue is in patients with persistent asthma (frequent attacks) where chronic inflammation has no time to stop and disappear before another episode occurs. Bottom line: Even if a patient with persistent asthma has no symptoms, he/she must continue on anti-inflammatory medication to prevent the progression to structural changes which are very difficult to treat.

Diagnosis of Asthma:

Asthma is diagnosed **clinically most of the time** by **its typical** history and physical exam.

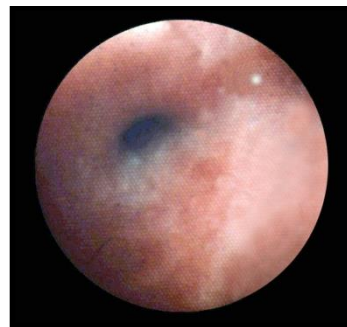
In case of doubt:

- Spirometry
- Methacholine challenge test: **It's not uncommon** for patients whose symptoms are suggestive of asthma to have normal lung function. In these circumstances the demonstration of airway hyper-responsiveness by challenge tests may be useful in confirming the diagnosis. (Davidson)

For more: <http://www.healthgrades.com/procedures/methacholine-challenge-test>



Before



10 mins after challenge test

History:

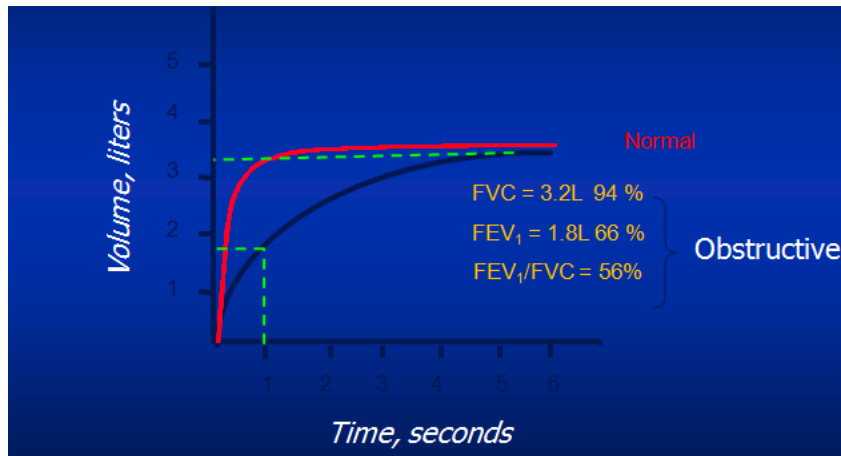
- Tightness of the chest, cough & expectoration, wheeze.
- Comes in episodes, (recurrent).
- With exposure to allergens and irritants.
- History of asthma attacks.
- Relieve using salbutamol.
- Allergy in skin, eyes, nose. **Like eczema or allergic rhinitis.**
- Family history of asthma or allergy.

Physical examination:

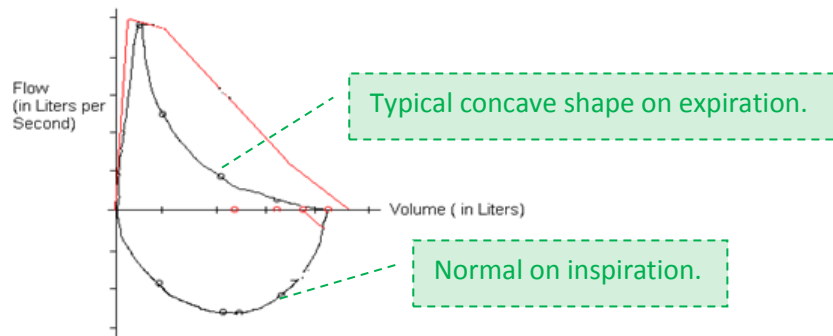
- Wheeze /Rhonchi (**no crackles**) → **Crackles arise from pathology within the alveoli.** So if you hear crackles in a suspected case of asthma, it's either **not** asthma OR asthma with another disease.
- Tachypnea.
- Signs of allergy of skin, nose and eyes.
- **Remember: Absence of symptoms at the time of examination does not exclude the diagnosis of asthma.**

Investigations:

❖ **Spirometry:**



- Volume/Time traces from forced expiration in a normal subject and a patient with obstructive disease.
- In obstructive diseases: Slow, prolonged and limited exhalation. → FEV1/FVC is reduced.



Spirometry		Pre-Drug*		
		Predicted	Actual	%Pred
FVC	(L)	4.01	2.95	74
FEV1	(L)	3.18	1.66	52
FEV.5	(L)		1.13	
FEV1/FVC	(%)	80	56	70
FEF25-75%	(L/S)	3.20	0.80	25
PEFmax	(L/S)	8.14	4.28	53
MET	(SEC)		1.85	
FIV.5	(L)		2.13	
FEF50%	(L/S)		0.95	
FIF50%	(L/S)		4.83	
FEF50/FIF50	(%)		20	

- Same data plotted as Flow/Volume loops. -*Red trace illustrates normal subject's spirometry.
- If you give the patient Salbutamol and this improves by at least 12% → confirm the diagnosis of Asthma.

❖ **Peak Expiratory Flow (PEF) meter: Measures the speed**

Allows the patient to assess the status of his or her asthma.

Measures the amount of air a person can blow out in liters per minute. You set the indicator to zero, take a deep breath, put your lips around the mouthpiece and exhale as hard and fast as you can. Then you read the number on the scale. The proper use of PEF meters can help predict asthma episodes and monitor response to therapy. The goal is to monitor the airflow consistently to recognize any changes from normal.

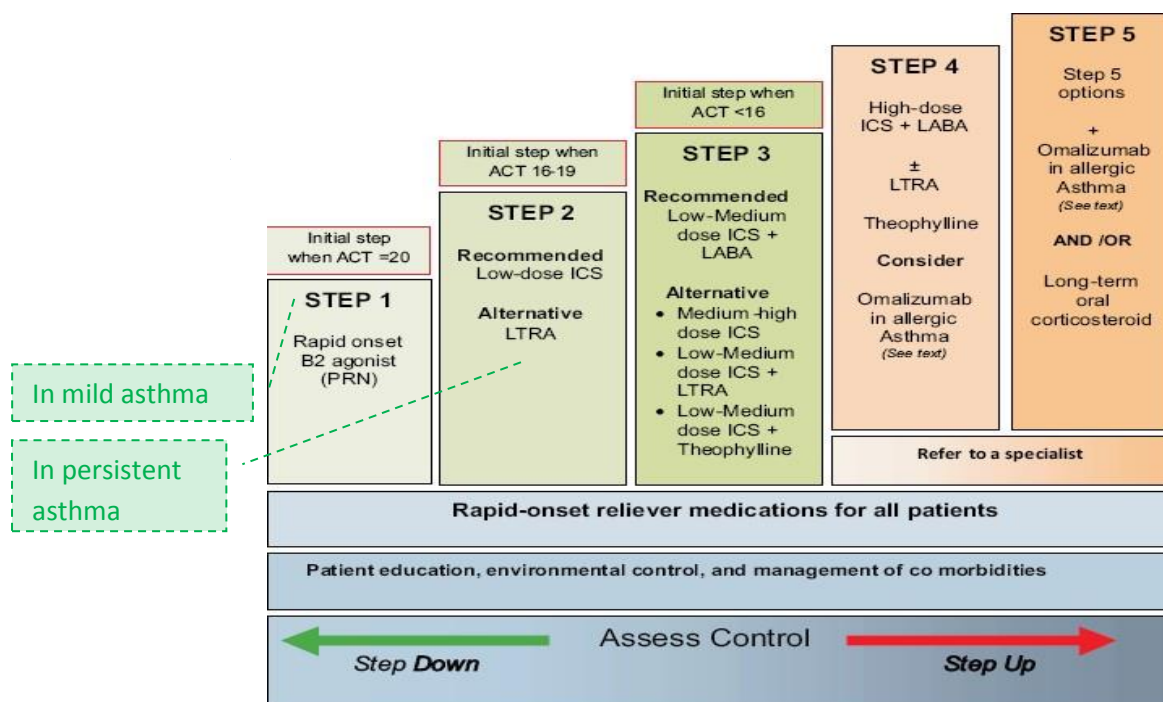
Treatment: Important

- **Inhaled corticosteroids (ICS):** Controller/preventer → Delay the progression of chronic inflammation.
Budesonide, fluticasone, beclomethasone, ciclesonide, mometasone.
-Anti-inflammatory
-Take time to act (1-3 hours).
-Long-term effect (12-24 hours).
-Only for regular use (whether well or not well).
- **Other types of controller drugs:**
-Leukotriene modifiers (montelukast).
-Anti-IgE (omalizumab =Xolair).
-Systemic steroids.
- **Beta2 Agonists (stimulants):** Relieve symptoms but have no effect on inflammatory process.
 1. Short acting (SABA) → salbutamol.
-Bronchodilator.
-Quickly relieves symptoms (within 2-3 minutes)
-Not for regular use. (PRN = used when needed)
-Duration of action = 4-6 hrs
 2. Long Acting (LABA): Duration of action = 12-24 hrs
-Rapid acting → formoterol. Effect is evident in a few minutes.
-Non- Rapid acting → salmeterol. Effect will be evident in about half an hour.
- **Anti-cholinergic drugs:** Anti-muscarinic action on bronchi (bronchodilation) but with no effect on inflammation.
-Ipratropium (Atrovent) → available as inhaler or solution for nebulizer.
-Tiotropium (Spiriva) inhaler.
- **Combinations: ICS + B₂ Agonist**
-Symbicort: budesonide + formoterol.
-Seretide: fluticasone + salmeterol.
-Foster: beclomethasone + formoterol.

❖ Why inhalation therapy?

Oral	Inhalation
Slow onset of action	Rapid onset of action
Large dosage used	Less amount of drug used
Greater side effects	Better tolerated
Not useful in acute symptoms	Very effective

Stepwise approach to the management of asthma:



- Treatment of asthma is flexible. You can step down if the patient improves, or step up if they worsen.
- We should evaluate disease status to choose appropriate treatment. This can be done by either:
 1. Asking the patient directly about severity (subjective) then giving treatment accordingly.
 2. Or we can request the patient to fill Asthma Control Test, a self-administered questionnaire, for standard assessment. After the patient has completed ACT, a score will be calculated to determine suitable treatment.
- **Before increasing medications, check:**
 - Inhaler technique. → Ask to patient to use it in front of you to make sure they're doing it correctly.
 - Adherence to prescribed regimen. → Patient compliance.
 - Environmental changes. → Avoidance of aggravating factors and irritants.
 - Also consider alternative diagnoses. → The patient could have a whole other condition and has been misdiagnosed.

Rules of Two:

The patient must be given controller/preventer medication in following conditions:

- Use of a quick-relief inhaler more than: 2 times per week. → two attacks per week
- Awaken at night due to asthma symptoms more than: 2 times per month
- Consumes a quick-relief inhaler more than: 2 times per year. → Meaning if they finish two bottles of inhalers in a one year.

SUMMARY

1. Asthma can be controlled but not cured.
2. It can present at any age.
3. It produces recurrent attacks of symptoms of SOB, cough with or without wheeze.
4. Between attacks patients with asthma lead normal lives.
5. In most cases there is some history of allergy in the family.
6. Understanding the disease, learning the technique and compliance with medications is the key for good control of asthma.
7. Inflammation provoked by allergens results in airway hyper-responsiveness & obstruction.
8. Activation of eosinophils produces a late allergic response while activation of mast cells produces an immediate one.
9. Exposure to triggers such as Infections, chemicals, some medications, strong emotions, exercise, cold temperature, smoke cause an acute exacerbation of symptoms.
10. Acute inflammation in asthma is associated with bronchoconstriction, plasma exudation/edema, vasodilatation, and mucus hypersecretion.
11. Chronic inflammation in asthma is associated with subepithelial fibrosis, smooth-muscle hyperplasia/hypertrophy, mucus gland hyperplasia, and new-vessel formation.
12. If asthma remains uncontrolled or poorly controlled, the underlying chronic inflammation may lead to structural changes (remodelling) that reduce the extent of airway response to therapy.
13. Diagnosis of asthma is mostly clinical but can be supported by lung function test.
14. Treatment of asthma includes anti-inflammatory medications such as corticosteroids & bronchodilators such as β_2 agonists.

IMPORTANT NOTES FROM EXTERNAL RESOURCES

Notes

Step-up to medicine

Asthma is classified into extrinsic and intrinsic asthma but there is considerable overlap:-

(a) Extrinsic Asthma (most common):

- Patients are atopic, i.e., produce immunoglobulin E (IgE) to environmental antigens. May be associated with eczema and hay fever.

- Patients become asthmatic at a young age.

(b) Intrinsic asthma—not related to atopy or environmental triggers

Questions

1- In Asmathatic Patient FEV1 is:

- A. Increased.
- B. Normal.
- C. Reduced.

2-What is the characteristic feature of chronic untreated asthma?

- A- Airway remodeling
- B- Mucus hypersecretion
- C- Smooth muscle hyperplasia.

432 Medicine Team Leaders

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Answers:

1: C

2: A