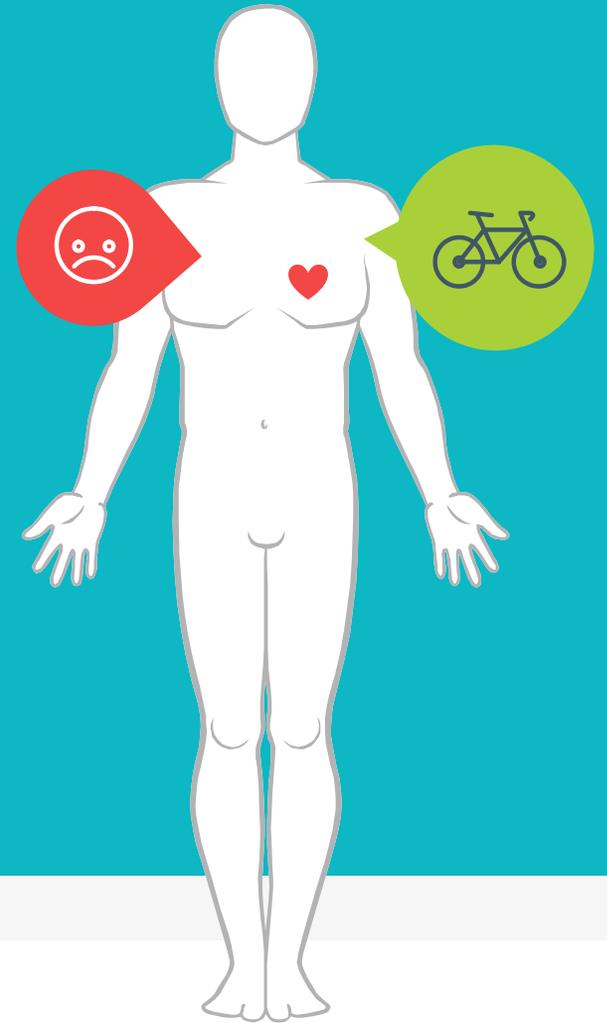
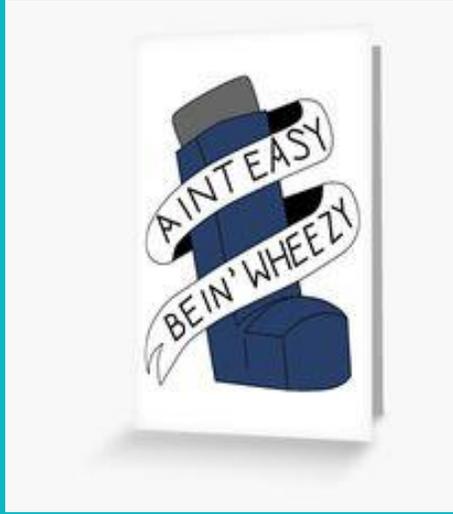


# Respiratory System Pathology: Obstructive Lung Disease II



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10/16/2025

# 3. Asthma



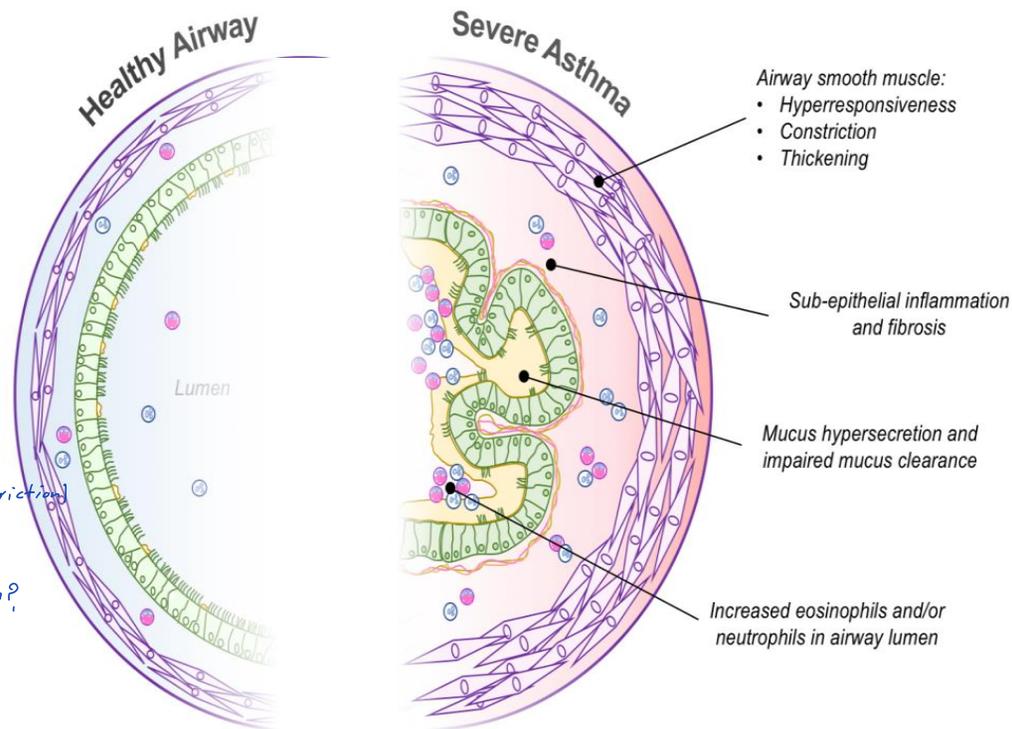
Asthma is a chronic inflammatory disorder of the airways that causes recurrent episodes of wheezing, breathlessness, chest tightness, and cough, particularly at night and/or early in the morning

فهيئة في الصدر

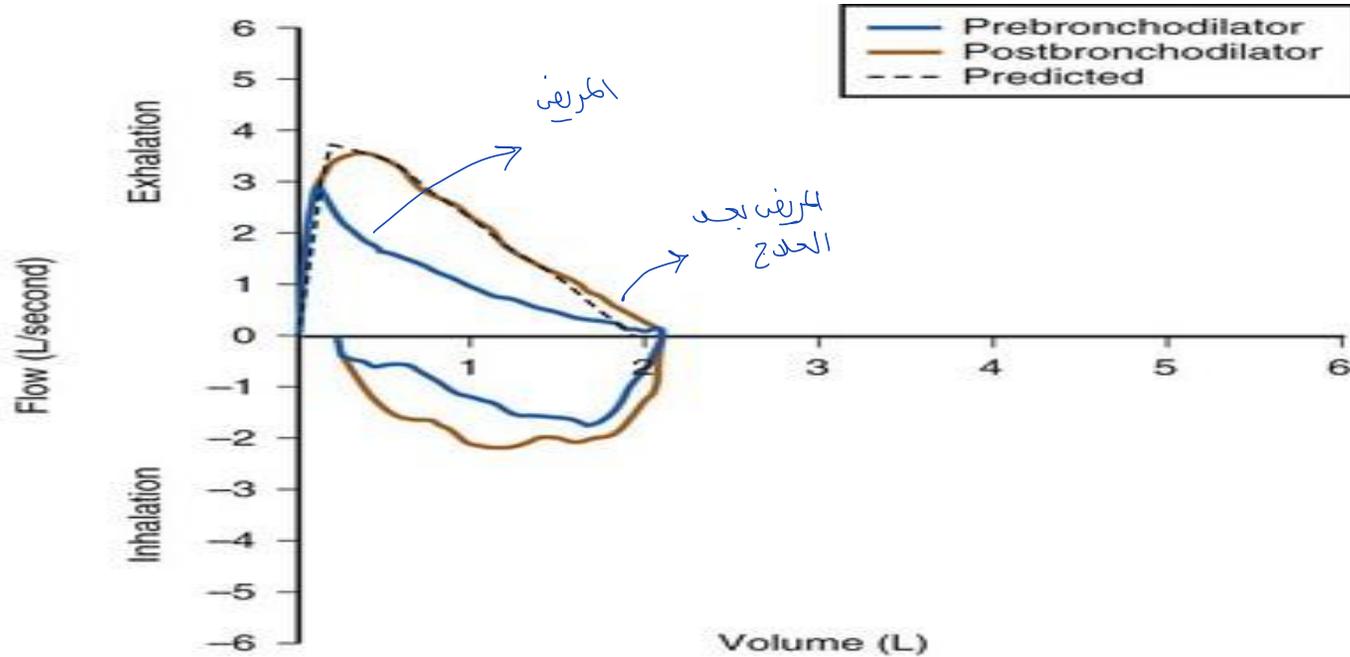
# The hallmarks of asthma

- ▶ Intermittent, reversible airway obstruction.
- ▶ Chronic bronchial inflammation with eosinophils. رسودها متقطعي asthma ال
- ▶ Bronchial (smooth muscle (constriction) cell hypertrophy & hyperreactivity.) the cause of obstruction?
- ▶ Increased mucus secretion.

← يوم آه يومك  
← الفحيح تمام، المساء تبعب



# Asthma - PFT



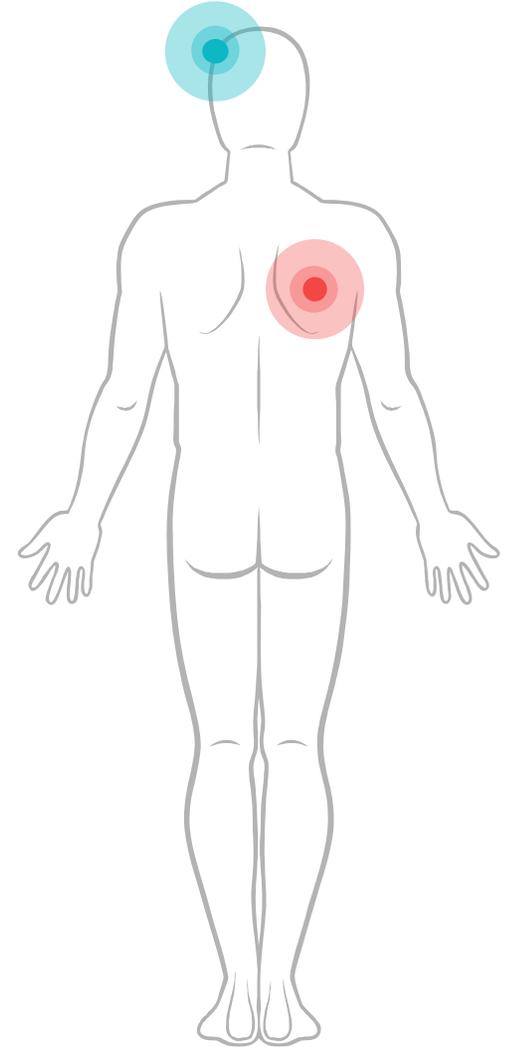
Spirometry Parameter	Units	Predicted Value	Pre	Observed Percent Pred	Post	Observed Percent Pred	Percent Change
FVC	Liter	1.94	2.15	111	2.14	110	0
FEV <sub>1</sub>	Liter	1.71	1.30	76	1.82	106	40
FEV <sub>1</sub> /FVC	Percent	90	60	67	85	94	42

post-treatment

# Asthma main categories:

## Atopic

- ▶ Most common
- ▶ Evidence of allergen sensitization.
- ▶ Usually begins in childhood.
- ▶ +ve family history.
- ▶ IgE-mediated (type I) hypersensitivity reaction.
- ▶ triggered by environmental allergens, eg. dusts, pollens.
- ▶ Ass/w allergic → URT  
rhinitis, urticaria, or eczema.  
↳ skin



# Asthma main categories:

## Non-Atopic

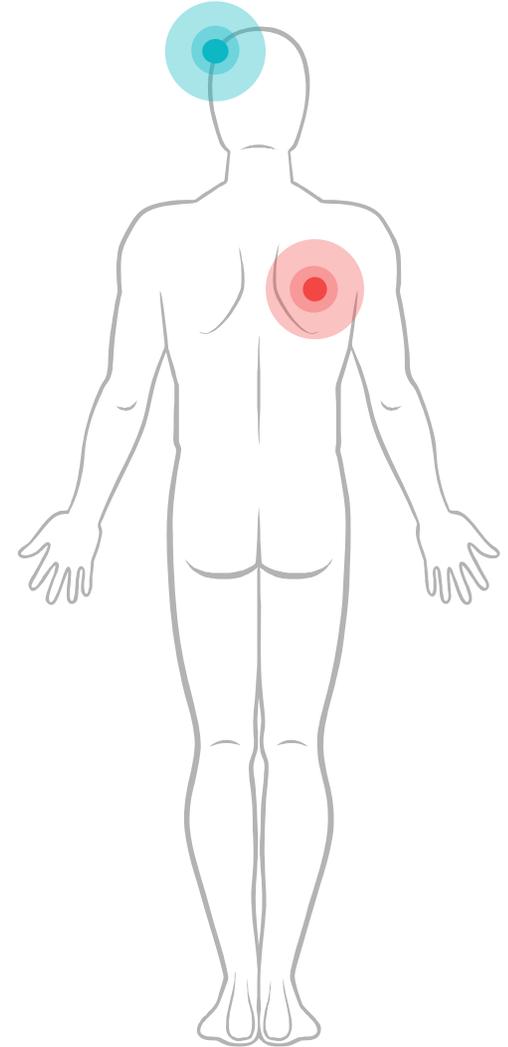
- ▶ No evidence of allergen sensitization.
- ▶ Triggers: Viral Respiratory infections & inhaled air pollutants
- ▶ Less common family Hx.

## Drug-Induced

Several pharmacologic agents provoke asthma. Aspirin is the most striking e.g.

## Occupational

Triggered by fumes, organic & chemical dusts (wood, cotton, platinum), gases, & other chemicals



# Asthma – Pathogenesis

## *T<sub>H</sub>2 Responses, IgE & Inflammation:*

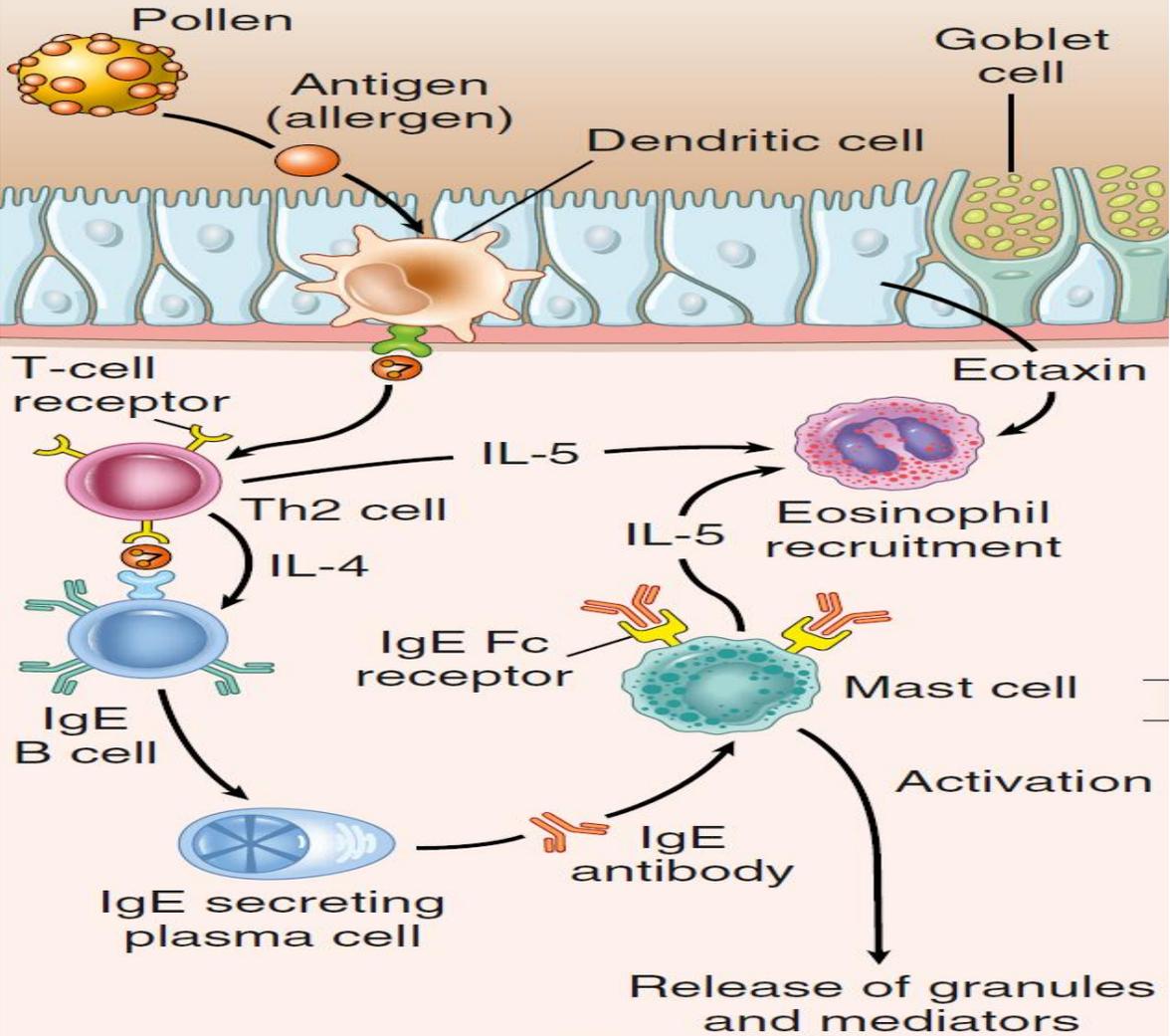
- ▶ A fundamental abnormality in asthma is an exaggerated *T<sub>H</sub>2* response to normally harmless environmental antigens (in genetically predisposed individuals.).
- ▶ *T<sub>H</sub>2* cells secrete **cytokines** → promote inflammation & stimulate B cells to produce IgE & other antibodies:
  - IL-4: stimulates IgE production
  - IL-13: stimulates mucus secretion & promotes IgE production by B cells.
  - IL-5: activates eosinophils.

# Asthma – Pathogenesis

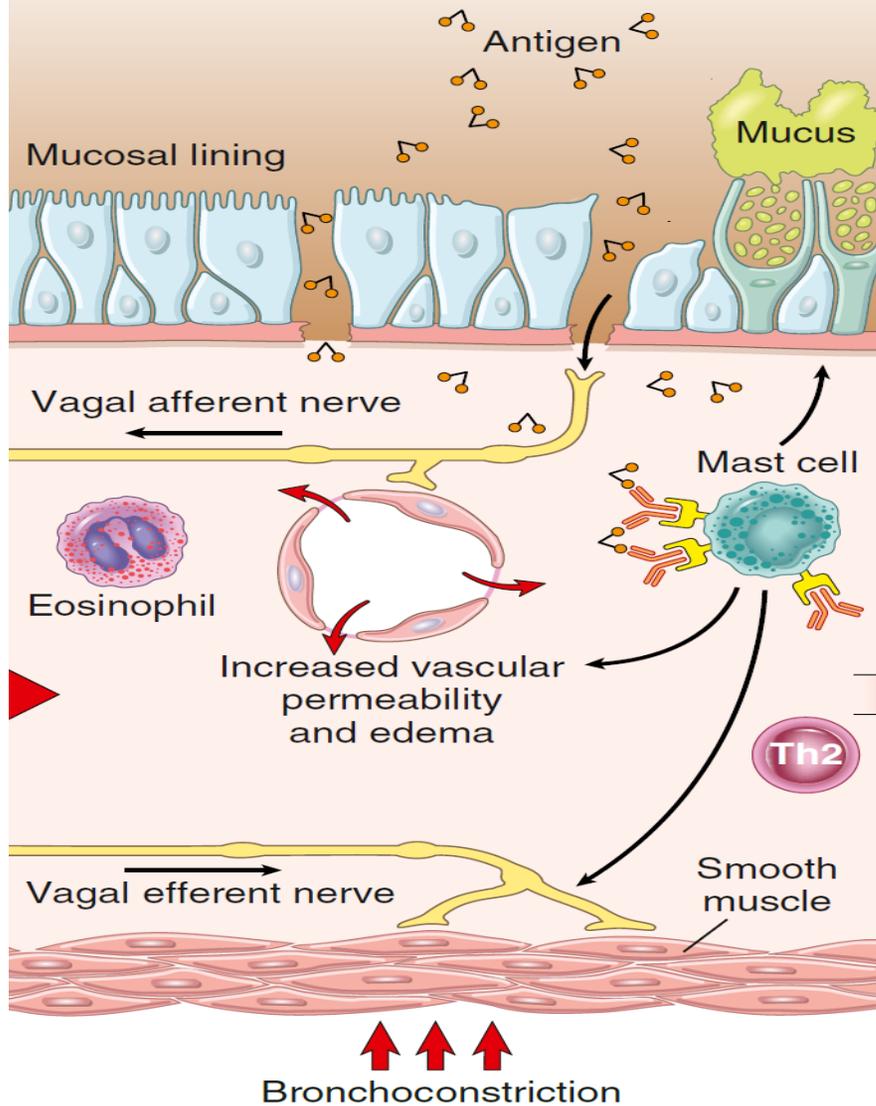
IgE coats submucosal mast cells → on exposure to allergen release their granule contents (cytokines & mediators) → two reactions:

- ▶ **Early (immediate) phase reaction:** dominated by (bronchoconstriction, ↑mucus production, & vasodilation) Bronchoconstriction is triggered by mediators (histamine, prostaglandin D<sub>2</sub>, & leukotrienes C<sub>4</sub>, D<sub>4</sub>, & E<sub>4</sub>) & also by reflex neural pathways.
- ▶ **Late-phase reaction:** Inflammatory in nature; mediators stimulate epithelial to produce chemokines → recruitment of TH<sub>2</sub> cells, eosinophils & other leukocytes → amplifying the inflammatory reaction.

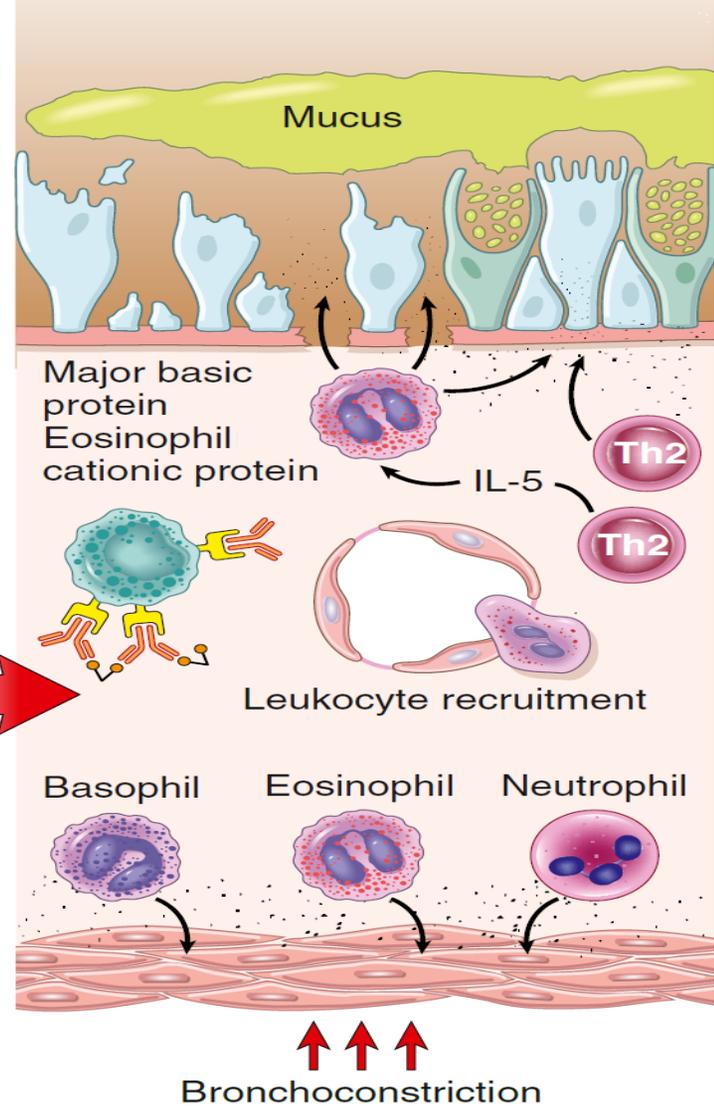
# C TRIGGERING OF ASTHMA



### D IMMEDIATE PHASE (MINUTES)



### E LATE PHASE (HOURS)



# Asthma – Pathogenesis

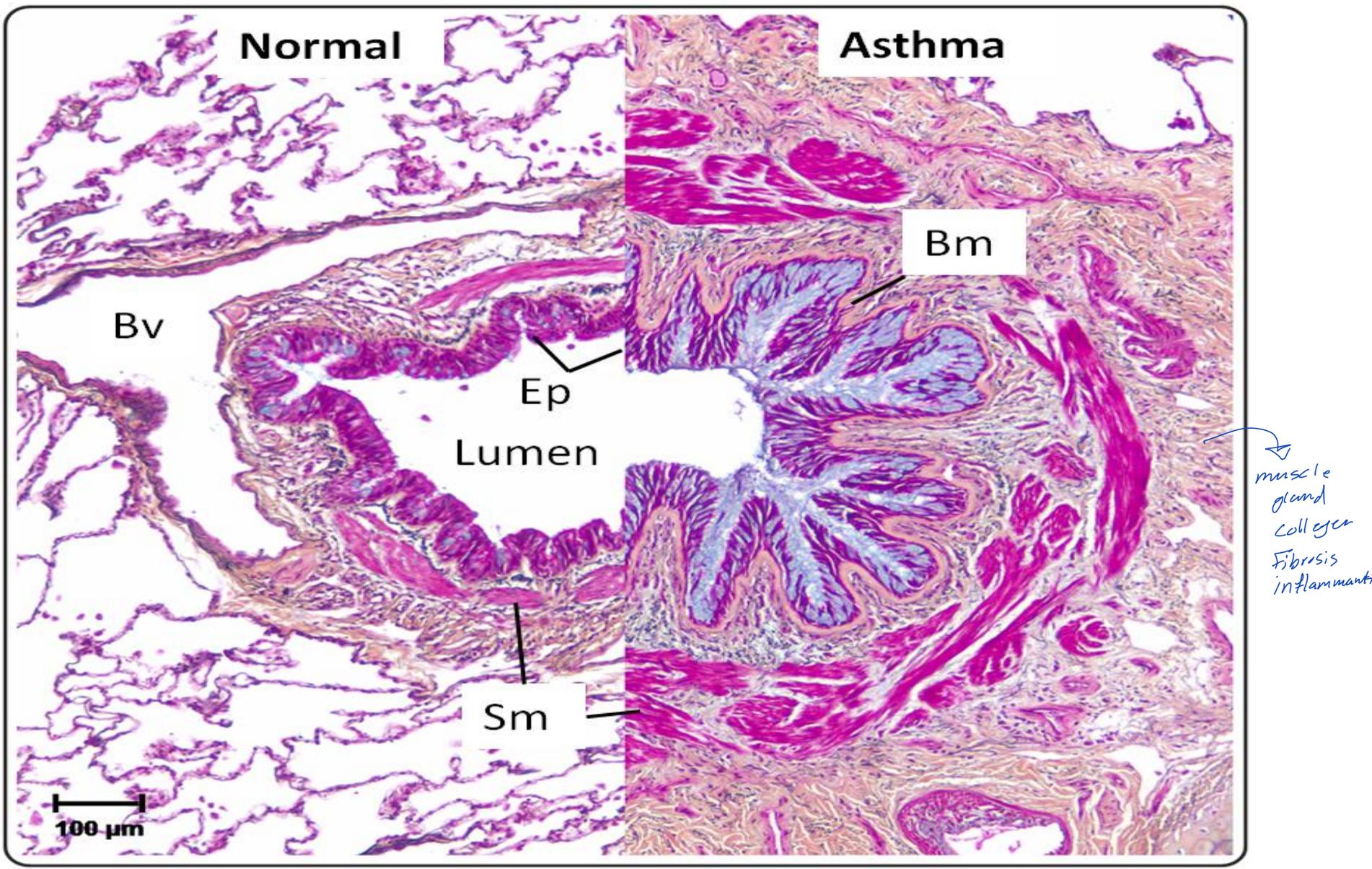
Airway remodeling:

Permanent changes in the airway

Repeated bouts of inflammation → structural changes in the bronchial wall called airway remodeling:

- ▶ Thickening of airway wall Narrower lumen → Lung obstruction
- ▶ Subbasement membrane fibrosis (deposition collagen)
- ▶ Increased vascularity
- ▶ ↑ in size of submucosal glands & number of goblet cells
- ▶ Hypertrophy and/or hyperplasia of bronchial wall muscle



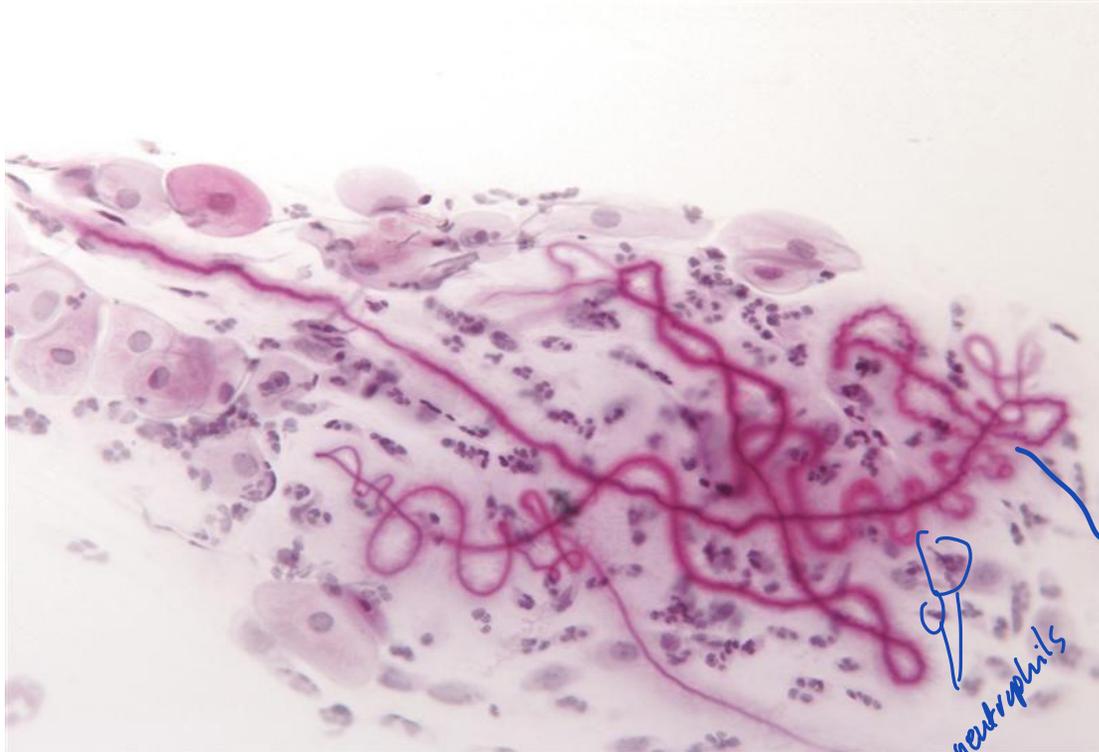


# Asthma – Morphology



The most striking finding is occlusion of bronchi and bronchioles by thick, tenacious mucous plugs

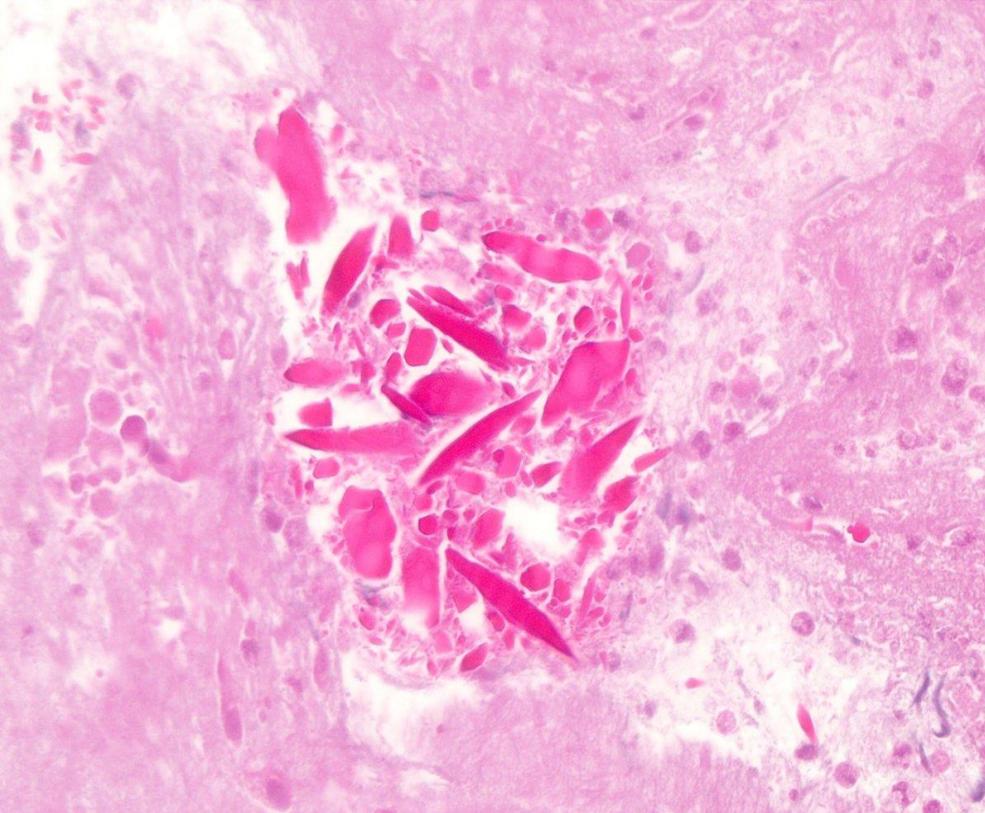
# Asthma – Morphology



**Curschmann Spirals** → result from extrusion of mucus plugs from subepithelial mucous gland ducts

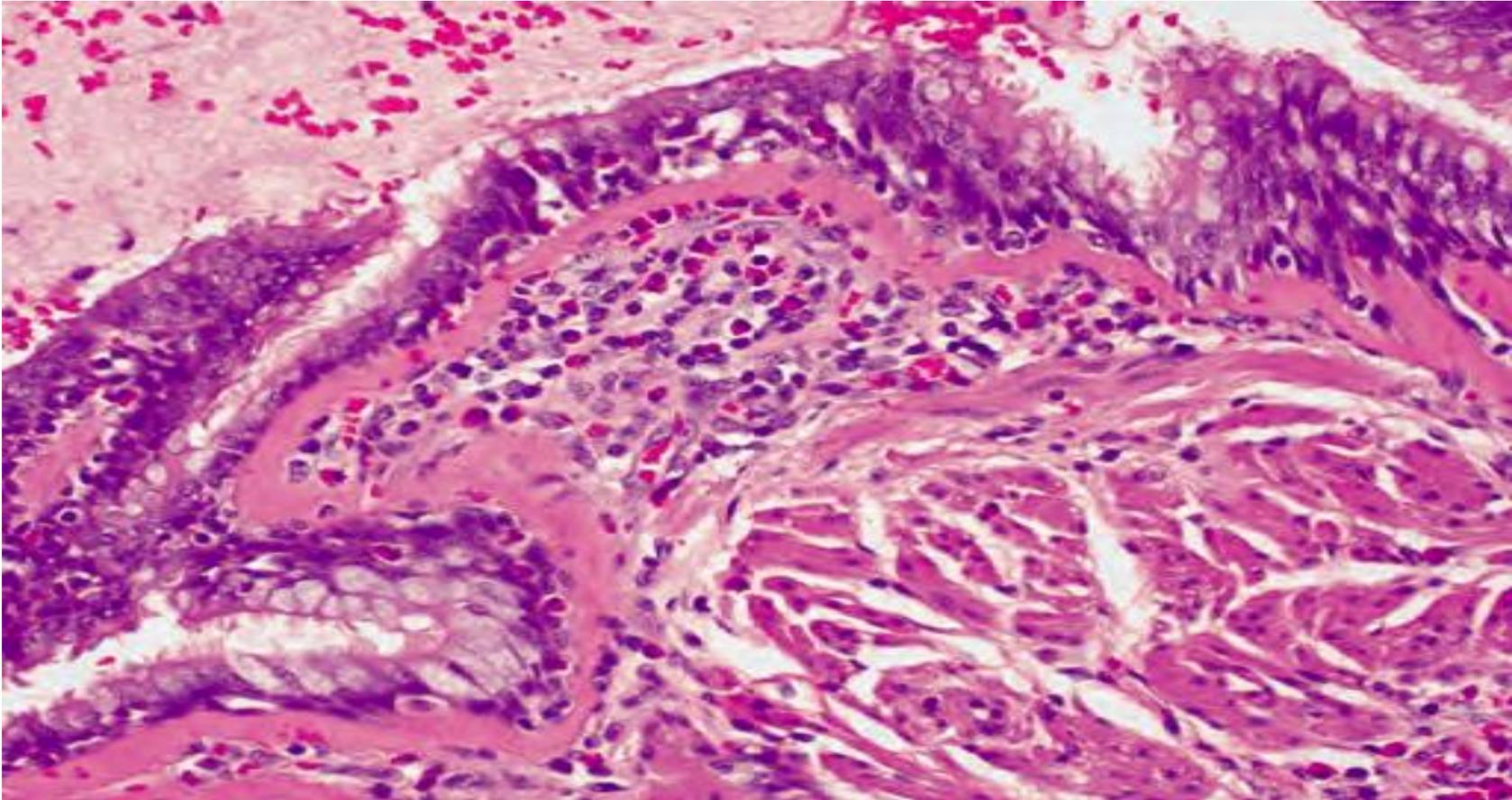
mucous gland ducts

# Asthma – Morphology



Numerous eosinophils and **Charcot-Leyden crystals** (crystalloids made up of the eosinophil protein galectin-10)

# Asthma – Morphology



} this thickness is never normal..

# Asthma – Clinical features

- ▶ **Classic asthmatic attack:** chest tightness, dyspnea, wheezing, & coughing (with or w/out sputum) due to bronchoconstriction & mucus plugging → trapping of air in distal airspaces & progressive hyperinflation of lungs. ↳ like emphysema
- ▶ Intervals between attacks are characteristically free from overt respiratory difficulties. (early disease)
- ▶ Occasionally a severe paroxysm occurs that does not respond to therapy and persists for days and even weeks (*status asthmaticus*) ass/w hypercapnia, acidosis, and severe hypoxia → may be fatal.

الأعراض التي  
عندها

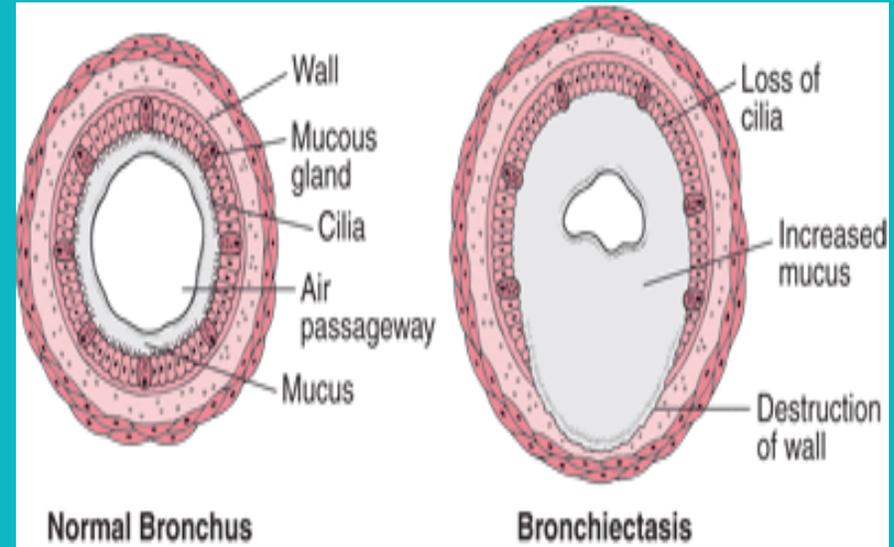
تكون بحاجة  
ICU

No response to normal  
treatment (bronchodilators,  
and corticosteroids)

# 4.

# Bronchiectasis

It is not a primary disease what is associated with another disease  
(Secondary disease)



The permanent dilation of bronchi & bronchioles caused by destruction of smooth muscle & the supporting elastic tissue; it typically results from or is associated with chronic necrotizing infections

# Bronchiectasis

← كل disorders التي اخذناهم قبل كانوا primary

- ▶ Not a primary disorder → always occurs secondary to **persistent infection or obstruction** caused by a variety of conditions.
- ▶ **Characteristic symptom complex:** cough & expectoration of copious amounts of purulent sputum.
- ▶ **Diagnosis:** appropriate history & radiographic demonstration of bronchial dilation.

# Conditions predispose to Bronchiectasis

- ▶ **Bronchial obstruction:** caused by tumors, foreign bodies, & impaction of mucus. (localized to the obstructed lung segment).
- ▶ **Congenital or hereditary conditions:**
  1. **Cystic fibrosis:** widespread severe bronchiectasis results from obstruction caused by abnormally viscid mucus and secondary infections.
  2. **Immunodeficiency states:** develops because of recurrent bacterial infections.

# Conditions predispose to Bronchiectasis

↳ autosomal recessive

3. **Primary ciliary dyskinesia:** (immotile cilia syndrome): Rare AR disorder ass/w bronchiectasis & sterility in males. Caused by inherited abnormalities of cilia → impair mucociliary clearance of the airways → persistent infections.
- ▶ **Necrotizing, or suppurative, pneumonia:** particularly with virulent organisms such as *Staphylococcus aureus* or *Klebsiella spp.*, predispose affected patients to development of bronchiectasis.

↳ accumulation of neutrophils

# Bronchiectasis - Pathogenesis

- ▶ **Two intertwined processes contribute to bronchiectasis: obstruction and chronic infection.** Either may be the initiator:
- ▶ E.g., obstruction by a foreign body <sup>+ Tumors</sup> impairs clearance of secretions → a favorable substrate for superimposed infection → inflammatory damage to bronchial wall & accumulating exudate distend the airways → irreversible dilation.
- ▶ E.g., Persistent necrotizing infection in bronchi or bronchioles → poor clearance of secretions, obstruction, & inflammation with peribronchial fibrosis and traction on the bronchi, culminating again in full-blown bronchiectasis → *associated with scarring*  
 ← بهير واسع و كبير

# Bronchiectasis - Morphology

Usually affects lower lobes, particularly those that are most vertical (gravitational).

**The airways dilated up to four times their usual diameter** → seen on gross examination almost out to the pleural surface (**normally** cannot be followed by eye beyond a point 2 to 3 cm from the pleura)



# Bronchiectasis – Morphology

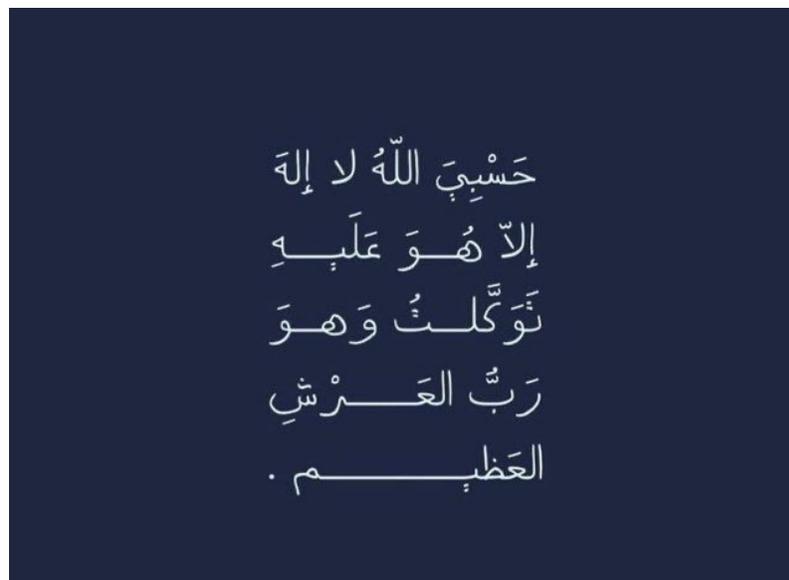
- ▶ The histological changes vary with the severity and duration of the disease.
- ▶ In an active full-blown disease: an intense acute & chronic inflammatory exudates within the wall of the bronchi & bronchioles seen & desquamation of lining epithelium cause extensive ulceration .
- ▶ In severe cases abnormal dilatation occurs due to necrosis & fibrosis of muscle coat with associated peribronchial fibrosis .
- ▶ In severe cases lung abscesses may develop .

*necrosis* → *fibrosis*  
*healing*

# Bronchiectasis – Clinical features

- ▶ **Symptoms:** severe, persistent cough ass/w expectoration of mucopurulent sputum.
- ▶ Other symptoms: **dyspnea, rhinosinusitis, and hemoptysis.** (precipitated by upper respiratory tract infections). ↗ Cough with blood
- ▶ Severe, widespread bronchiectasis → significant obstructive ventilatory defects → hypoxemia, hypercapnia, pulmonary hypertension & cor pulmonale.
- ▶ Current treatment outcomes improved → severe complications of bronchiectasis (brain abscess, amyloidosis & cor pulmonale) occur less frequently now than in the past.

Feature	Asthma	Bronchiectasis
Definition	Chronic airway inflammation → episodes of wheezing, dyspnea, and cough (esp. at night).	Permanent dilation of bronchi due to destruction of smooth muscle & elastic tissue.
Nature	Intermittent & <b>reversible</b> obstruction.	<b>Irreversible</b> airway dilation.
Pathogenesis	TH2 → IgE, eosinophils, mast cells → airway hyperreactivity.	Obstruction + chronic infection → wall destruction & fibrosis.
Main Causes / Triggers	Atopic (allergens), Non-atopic (viruses, pollutants), Drug (aspirin), Occupational (fumes, dusts).	Bronchial obstruction, Cystic fibrosis, Ciliary dyskinesia, Immunodeficiency, Necrotizing pneumonia.
Inflammatory Cells	Eosinophils, mast cells.	Neutrophils (chronic infection).
Airway Wall Changes	Thick wall, fibrosis, ↑vascularity, goblet & muscle hypertrophy.	Destruction of wall + peribronchial fibrosis, dilation esp. lower lobes.
Morphology	Mucus plugs, Curschmann spirals, Charcot-Leyden crystals.	Ulceration, necrosis, fibrosis, abscesses.
Clinical Features	Episodic wheeze, dyspnea, chest tightness, air trapping; may progress to status asthmaticus.	Persistent purulent cough, dyspnea, hemoptysis; may lead to cor pulmonale.
Reversibility	<b>Reversible</b> (intermittent).	<b>Irreversible.</b>
Complications	Status asthmaticus (fatal).	Brain abscess, amyloidosis, cor pulmonale.



Questions?  
Thanx!

*'Things get bad for all of us, almost continually, and what we do under the constant stress reveals who/what we are.'*

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