



Pathology of the Respiratory System

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Objectives: You have to know the following items:

- **Upper respiratory tract dz:**

- Infectious rhinitis.
- Allergic rhinitis.
- Nasal polyp.
- Nasopharyngeal angiofibroma.
- Nasopharyngeal carcinoma.
- Larynx : - Laryngeal nodule.
 - Laryngeal carcinoma.

- **Lung**

- ***Atelectasis*** (Collapse).
- ***Diffuse pulmonary diseases:***
 - Obstructive lung dz:**
 - Asthma.
 - Chronic bronchitis.
 - Emphysema.
 - Bronchiectasis

-Restrictive lung dz:

-Acute (ARDS & Resp. distress syndrome of NB).

-Chronic : i. Without granuloma...

ii. With granuloma....

• *Lower respiratory tract infection (pneumonia):*

Classification: according to etiology & clinical settings.

• *Tumors :*

-Benign.

-Malignant : i-Primary.

ii- Secondary.

• *Pathology of pleura.*

Upper respiratory tract

- Infectious rhinitis: "common cold"

Common , caused by virus(s).

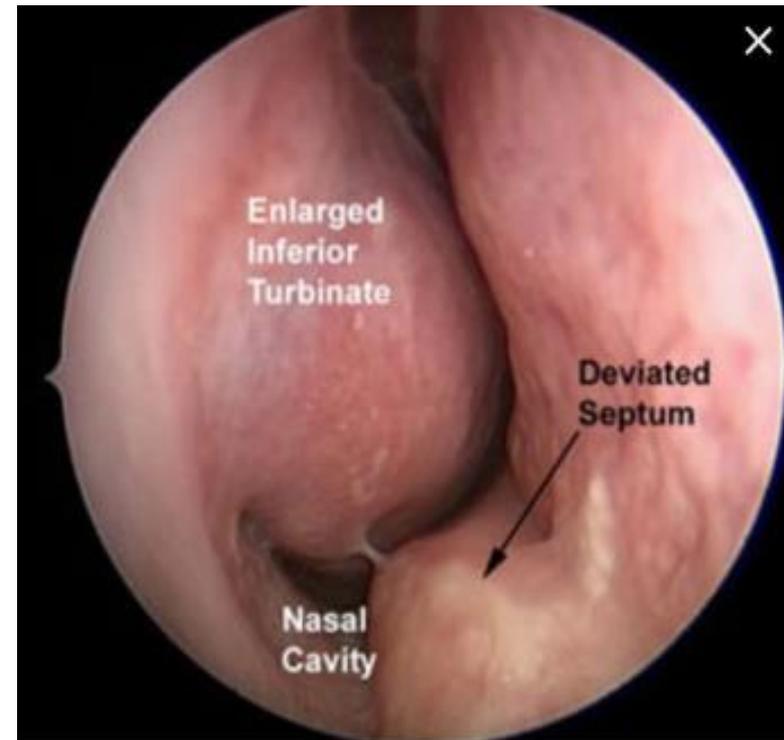
Evokes profuse catarrhal discharge ,initially the nasal mucosa is red thickened and edematous.

* without treatment clear up in a week if treated, but 7 days if ignored!.

-Secondary infection may occur...purulent secretion (AB is not a must).

- Allergic rhinitis : "hey fever"

Hypersensitivity-1 to some allergens, causing mucosal redness, edema and secretion with WBC (sp. eosinophil) infiltration, May be associated with turbinate hypertrophy \pm nasal obstruction.



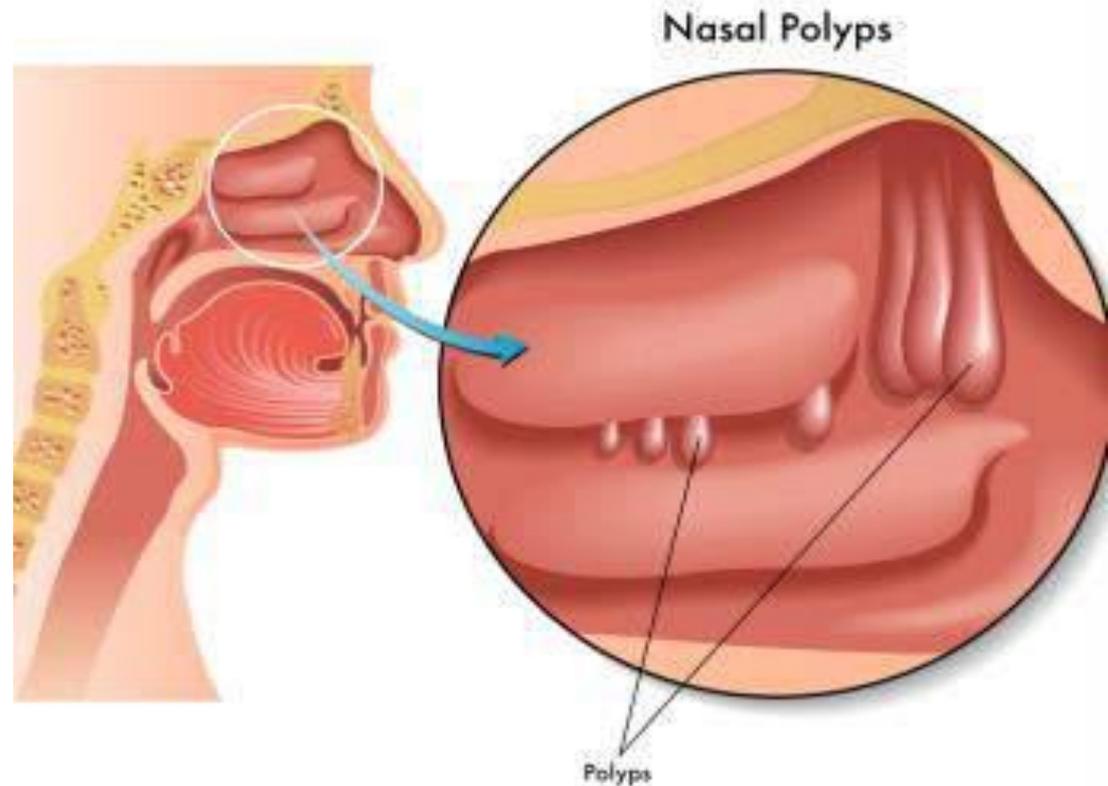
- **Nasal polyp:**

Recurrent attacks of rhinitis may leading to focal mucosa protrusion called polyp, ~ 2-4 cm. If occurs in child...CF is to be considered.

Histology:

Edematous & hyperplastic mucosa (± cystic glands) with loose stroma and infiltrated by inflammatory cells including eosinophils, plasma cells and lymphocytes. It can be multiple, may obstruct the airway & sinuses. In chronic cases there may be ulceration & infection.

Although allergy may be an etiologic factor; only 0.5% of atopic pt developed nasal polyp and most pt with polyps are not atopic.



• Acute epiglottitis

H. Influenza is the most common cause.

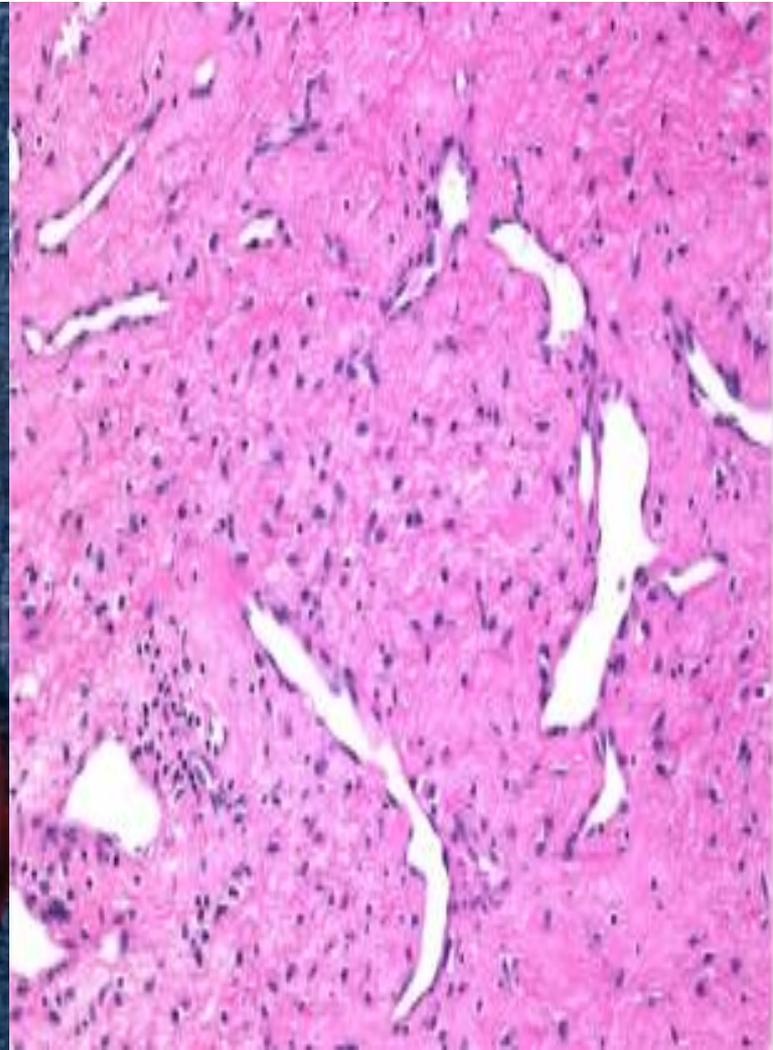
- Too much drooling, dysphagia, sore throat.
- Fever, muffled voice, inspiratory stridor.
- Thumb sign on x-ray.
- Risk of acute airway obstruction (medical emergency)



- **Nasopharyngeal angiofibroma:**

Polypoid richly vascularized spindle cell tumor, exclusively affects adolescent males.

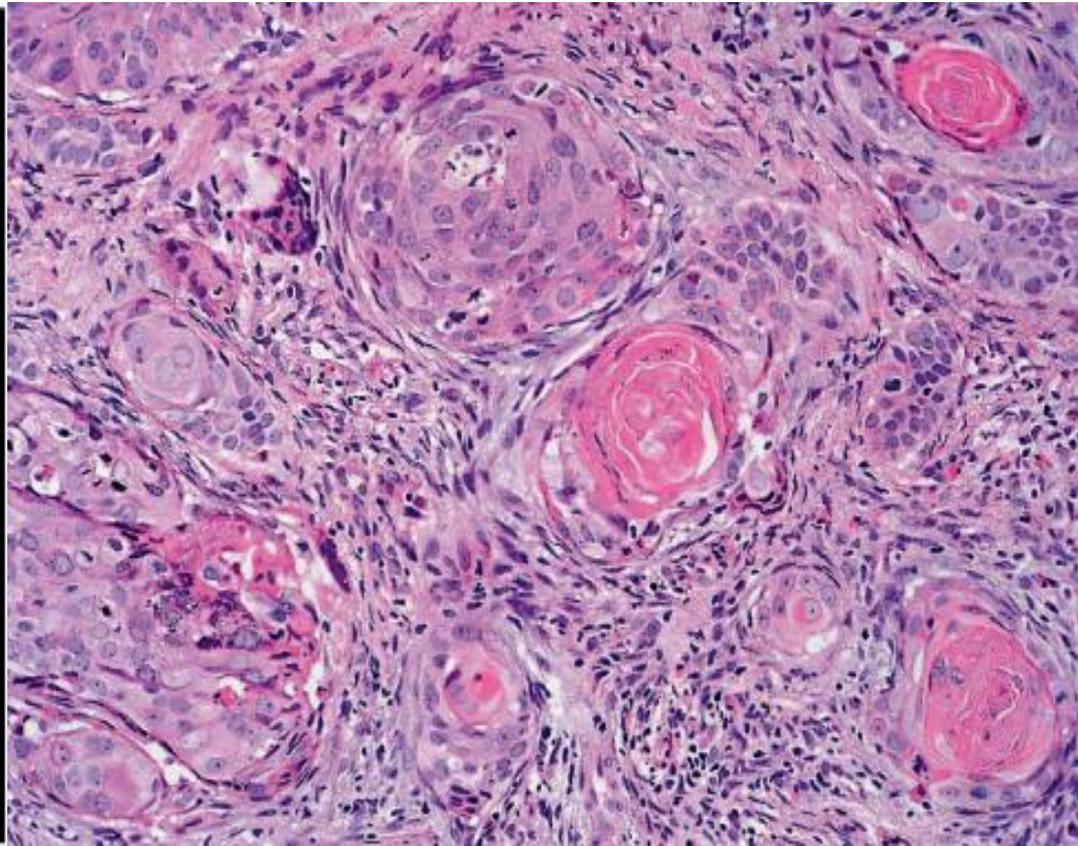
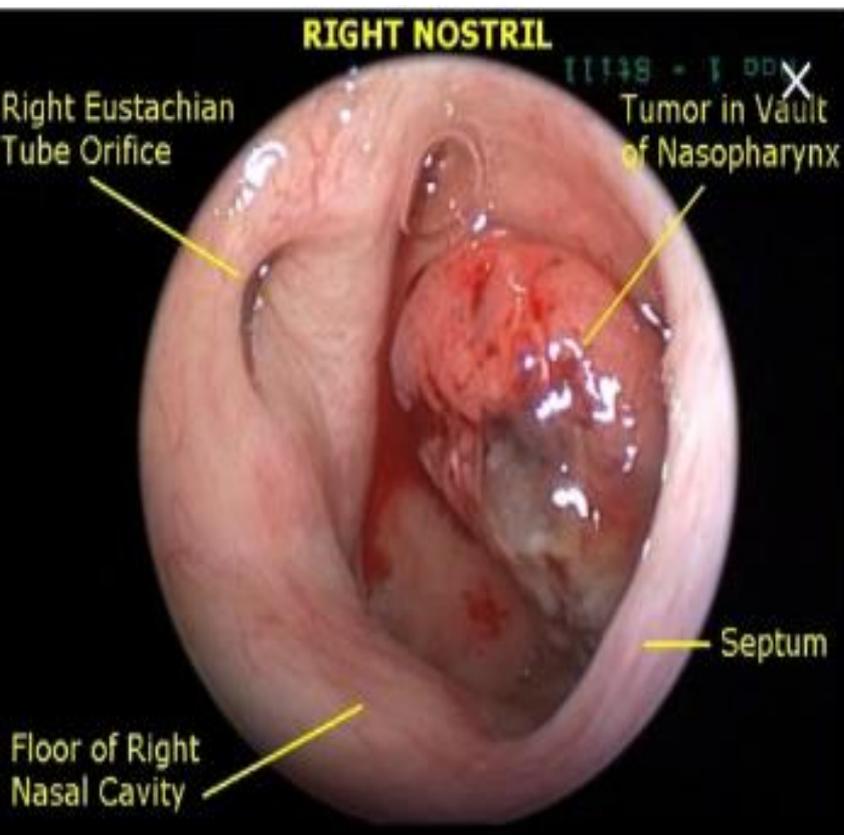
It is benign, but can cause severe bleeding specially during surgery.

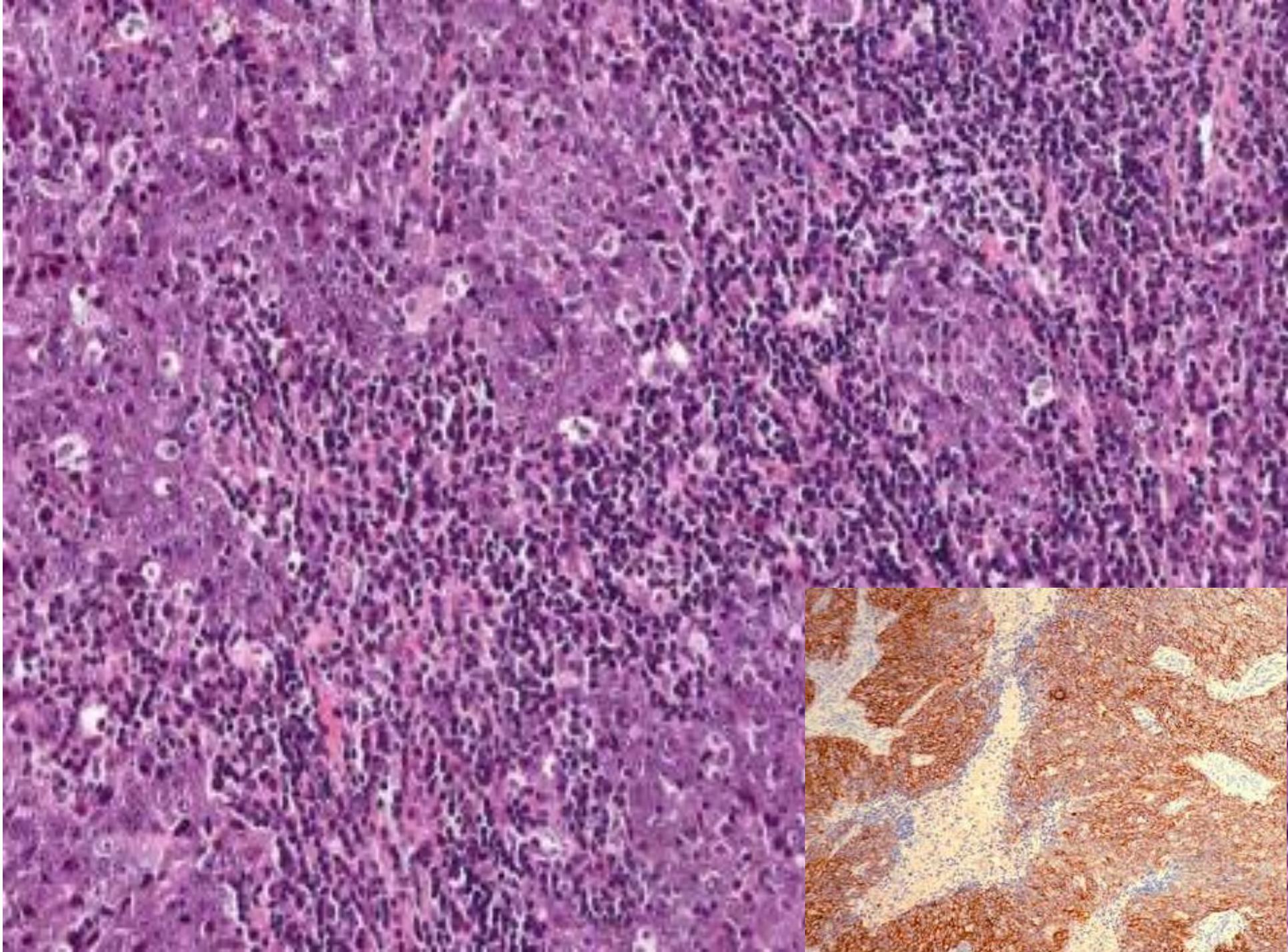


Nasopharyngeal carcinoma:

A type of carcinoma that has a special correlation to lymphoid tissue and EBV. It is of 3 subtypes:

- 1-Keratinizing sq. cell Ca.,
- 2-Non-keratinizing sq. cell ca.
- 3-Undifferentiated carcinoma (this type charact. by large epithelial cells with round vesicular nuclei and prominent nucleoli, admixed with abundant benign lymphocytes, so called (lymphoepithelioma)).



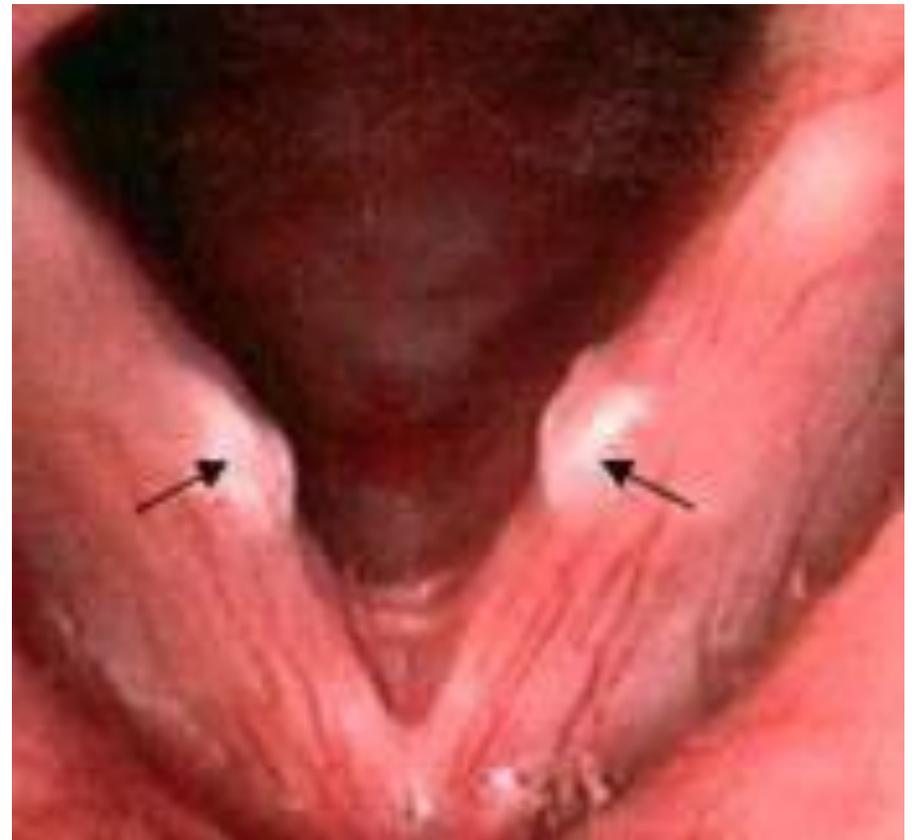


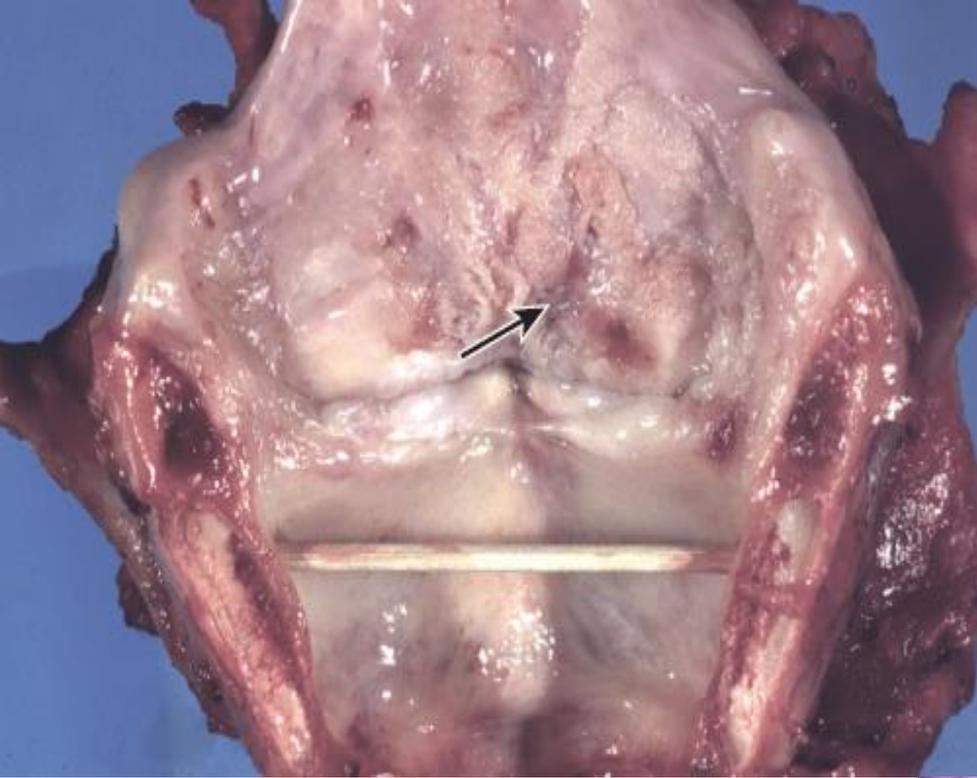
- Reactive laryngeal nodule “singer nodule”

Usually bilateral smooth vocal cord nodules, ~ few mm
Affecting heavy smokers and those using great strain on
their vocal cords. Classically presented with hoarsening voice.

Histology:

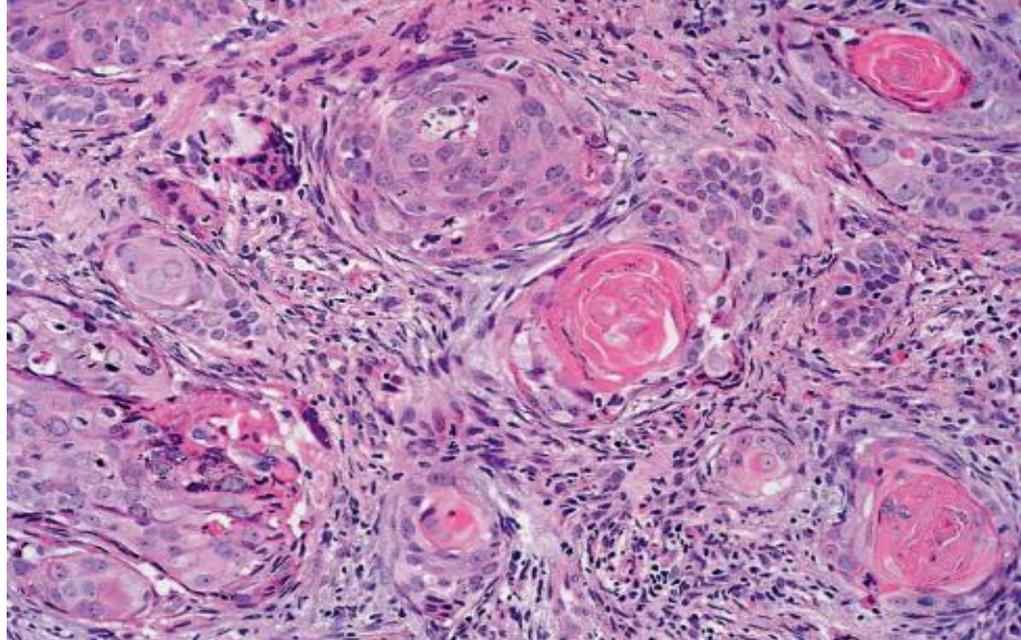
Small nodules covered by sq. epithelium overlying loose
vascularized myxoid core.





Carcinoma of larynx: (95% is Sq. cell carcinoma). Usually follows a spectrum of hyperplasia..dysplasia..ca in situ...and then invasive ca.

Risk factors: **smoking**, alcohol.. Asbestoses, HPV..





Pathology of the Respiratory System- 2

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Lung pathology

Atelectasis (Collapse).

Diffuse pulmonary diseases:

-Obstructive lung dz:

- Asthma.
- Chronic bronchitis.
- Emphysema.
- Bronchiectasis

-Restrictive lung dz:

- Acute (ARDS , Resp distress synd. of NB).
- Chronic : i. Without granuloma...
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Lower respiratory tract infection (pneumonia):

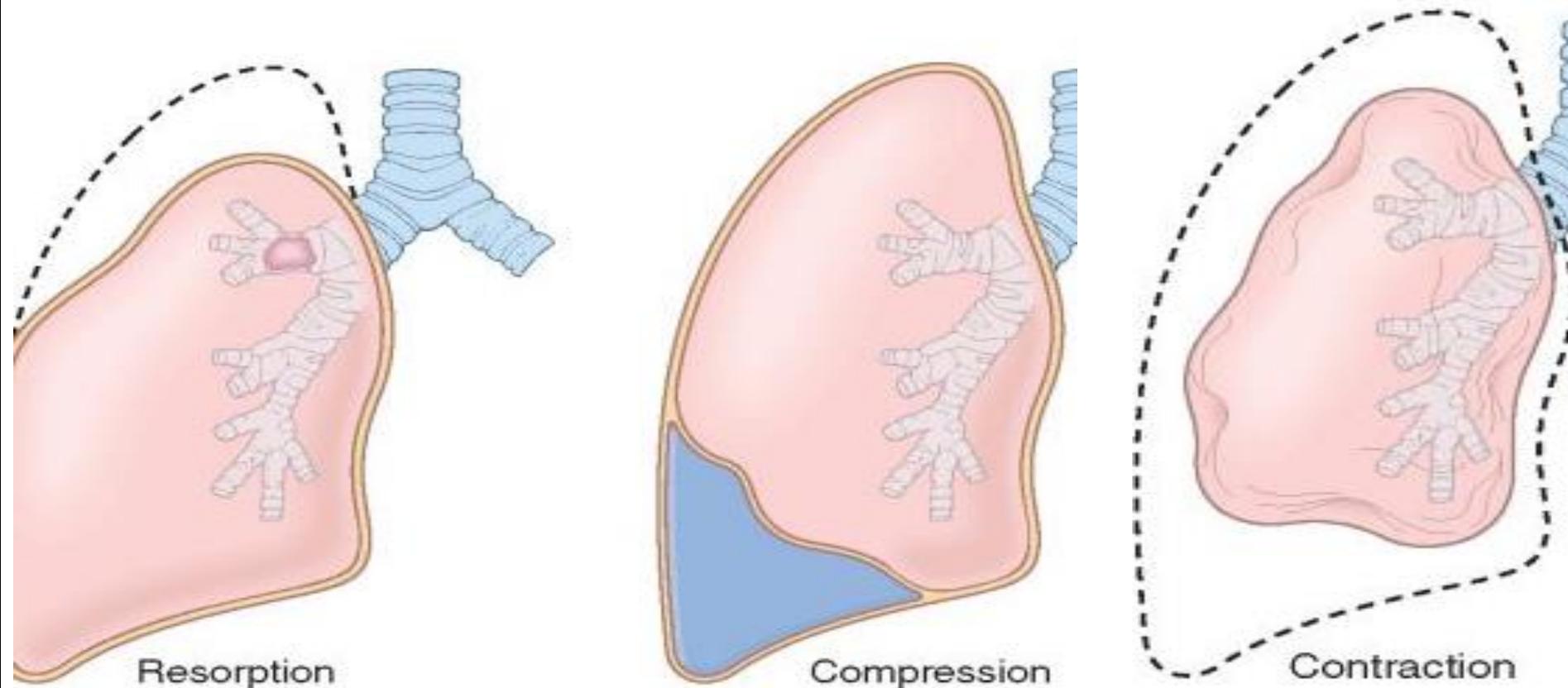
Classification: according to etiology & clinical settings.

Tumors

Atelectasis (Collapse):

Loss of lung volume caused by inadequate expansion of air spaces, results in shunting of inadequately oxygenated blood from pulmonary arteries into veins, thus giving rise to a ventilation perfusion imbalance and hypoxia, **Types:-**

- 1-Consequence of complete obstruction of an airway
- 2-By fluid , tumor , air.
- 3-Local or generalized fibrotic changes



OBSTRUCTIVE LUNG DISEASES:

- ↑ air out flow resistance at the level of bronchial passages (obstruction to getting air out of lungs).
- Decreased Forced Expiratory Volume (FEV at 1 sec.),
- They include:
 - Asthma,
 - Chronic bronchitis.
 - Bronchiectasis.
 - Cystic fibrosis.
 - Bronchiolitis.

Asthma

Episodic multifactorial dz in which there is **hyper-responsiveness** of airways to a variety of stimuli (in genetically susceptible pt), leading to **inflammatory reaction**, and then to episodes of bronchial **obstruction** that is relieved either spontaneously or by drugs.

- **Status Asthmaticus**: unrelieved prolonged attack.

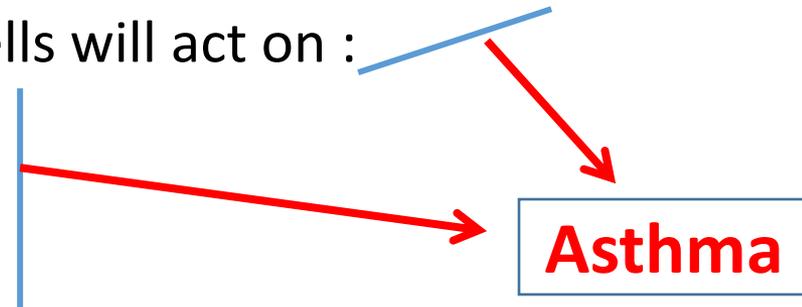
Pathogenesis

T-helper1 secretes interferon that **inhibits** T-helper2.

They found that T-helper1 cells from asthmatic pts have a genetic defect, and incompetent to inhibit T-helper 2.

T-Helper 2 secretes interleukins that stimulate eosinophils & IgE secretion with activation of mast cells, these mast cells will act on :

- influx of more eosinophils ,
- proliferation of bronchial smooth muscle .
- parasympathetic stimulation.



Phases of asthma:

i- Acute phase (starts within ~30 minutes)

1- Allergen binds to IgE on mast cells on surface epithelium.

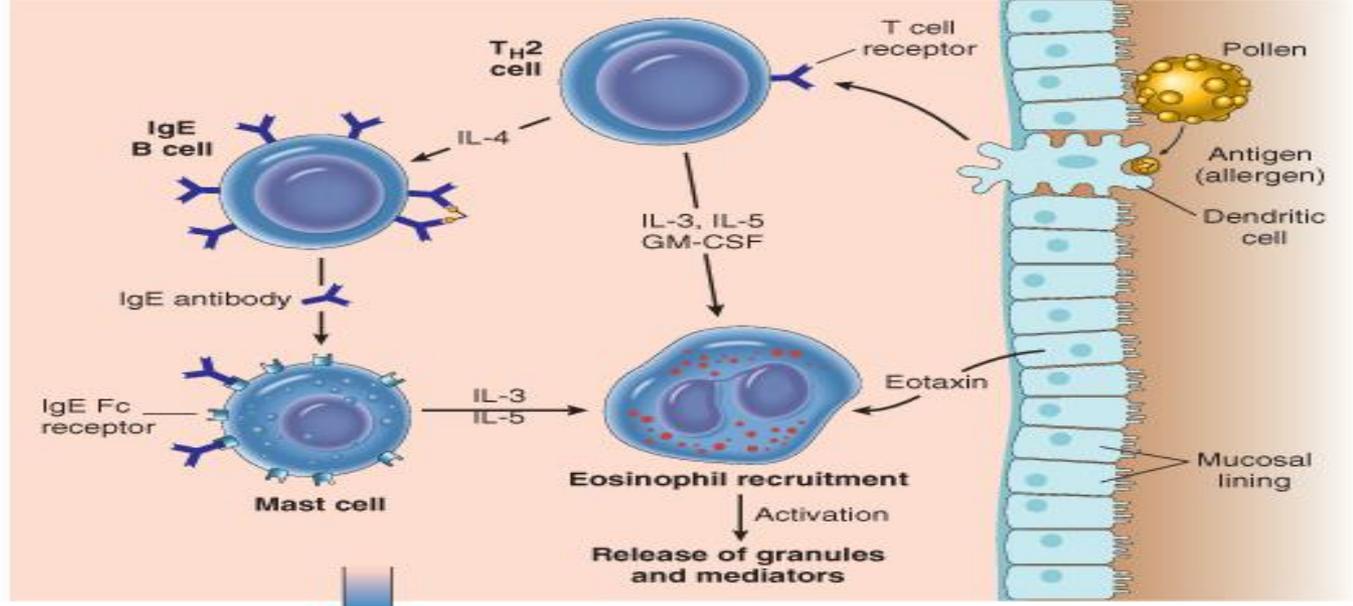
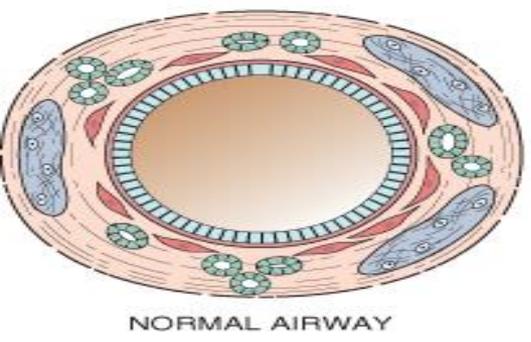
2- Degranulation of mast cells and release mediators that open the junctions and permits Ag to reach the subepithelial mast cells and parasympathetic nerves, so as a result there will be:

-bronchoconstriction , edema , mucus secretion and setting the cells (eosinophils, neutrophils, macrophages ..) for late phase.

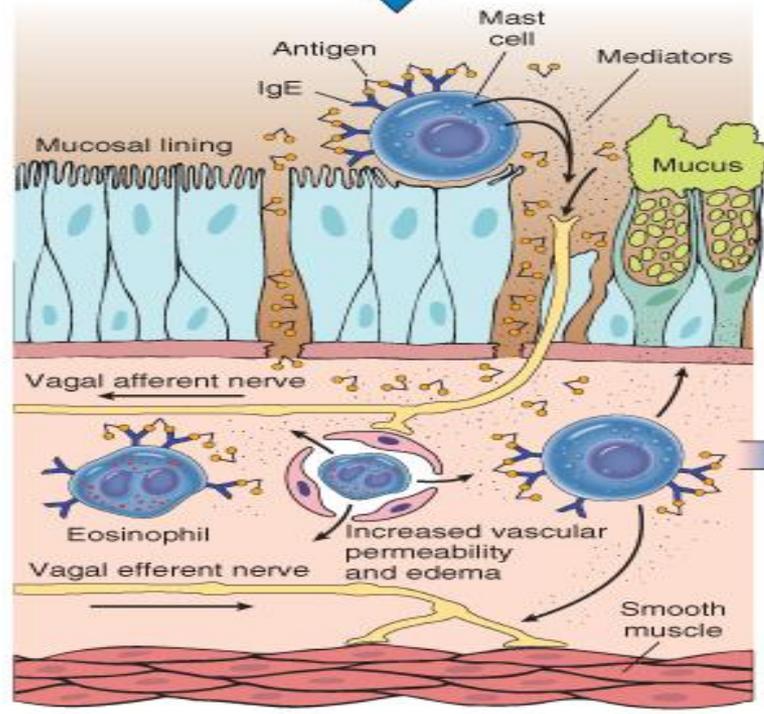
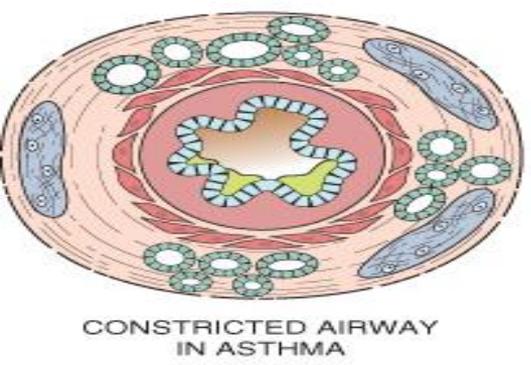
ii- Late phase (12-24 hrs):

Causes epithelial cells damage especially by eosinophils mediators (major basic protein and eosinophil cationic protein) in addition to bronchoconstriction , edema and mucus secretion.

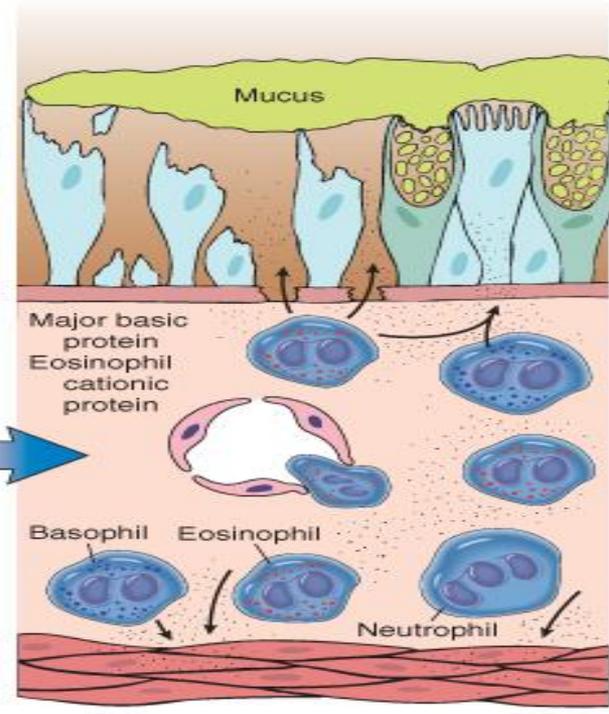
A. SENSITIZATION TO ALLERGEN



B. ALLERGEN-TRIGGERED ASTHMA



IMMEDIATE PHASE (MINUTES)



C. LATE PHASE (HOURS)

Stages of asthma

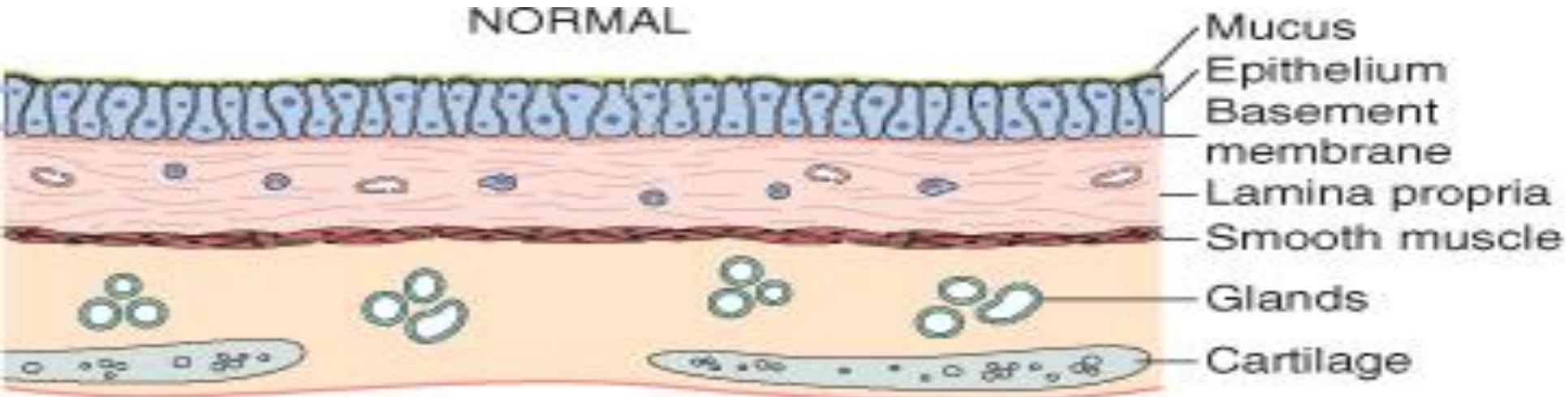
Pathology

- Mucosal edema, congestion + inflammatory cells (~ eosinophils.), lumen of bronchi & bronchioles contain thick mucous that contain:
 - * *Charcot Leyden crystals*: Eosinophil protein (~ eosinophilia).
 - * *Curshmann's spirals* : Whorls of necrotic epithelial cells.
- Necrosis of surface epithelium.
- Increased basement membrane collagen deposition
- Hypertrophy & hyperplasia of mucus gland with goblet cell metaplasia
- Hypertrophy & hyperplasia of smooth muscle.

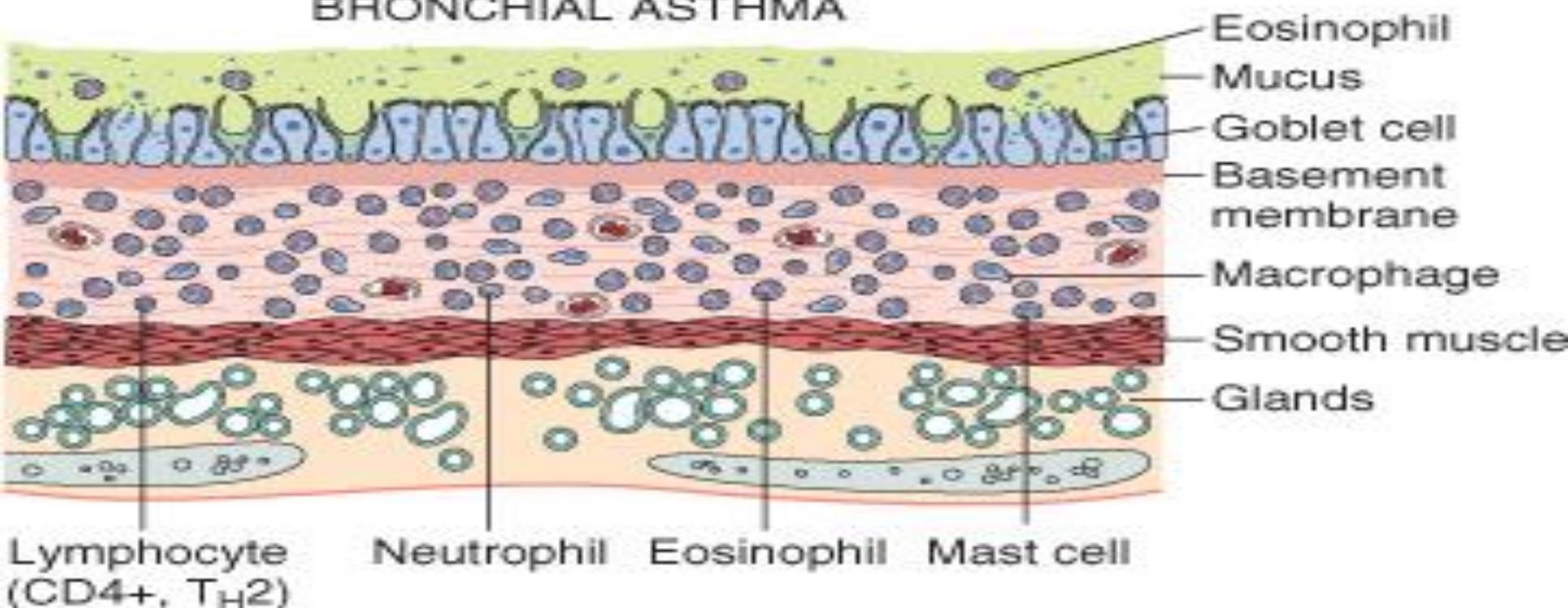
Clinical picture :

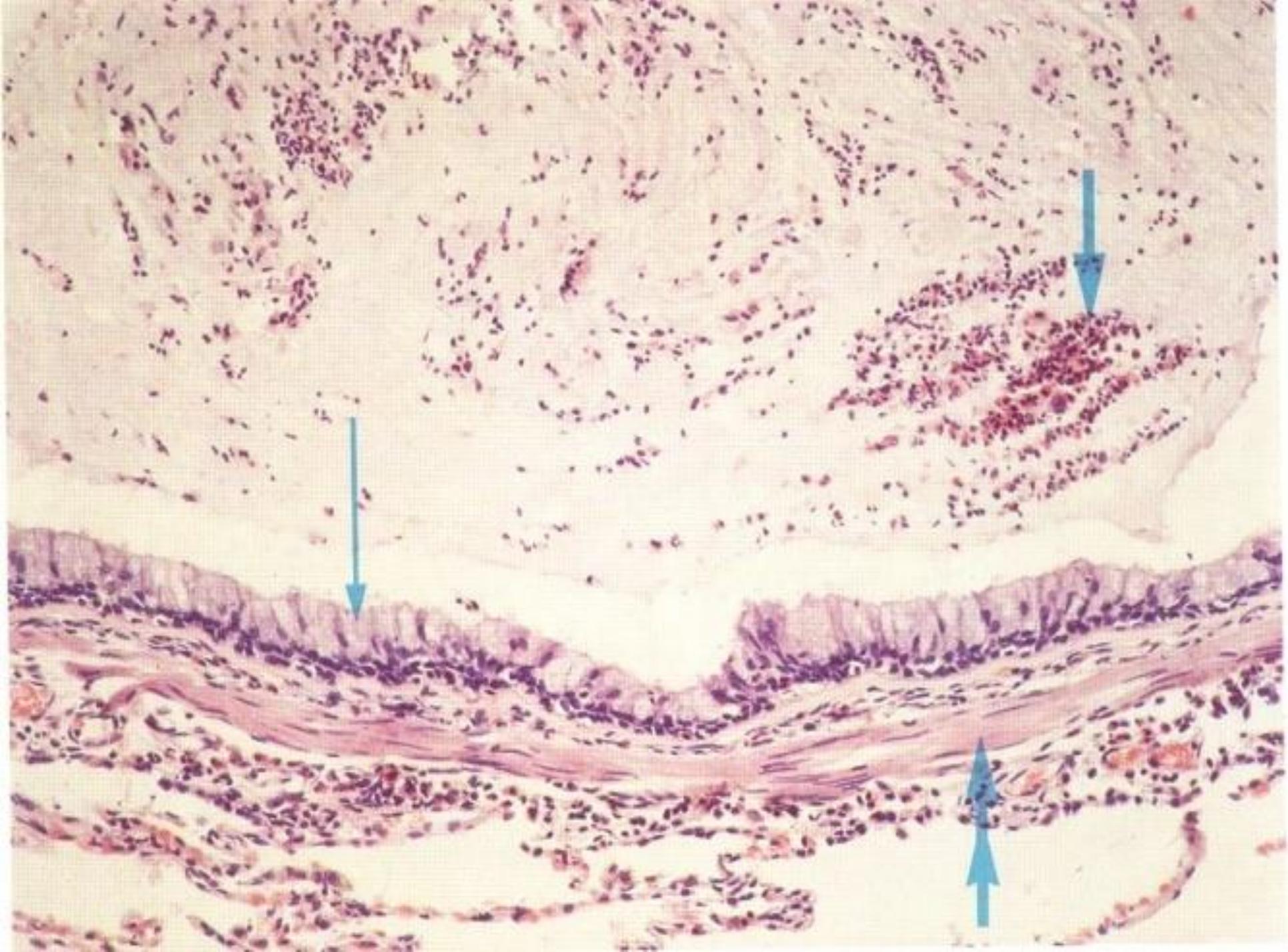
- 1- Reversible severe dyspnea ,wheezing & cough.
- 2- Lung hyperinflation
- 3- Bronchospasm lasting several hours or ***Status Asthmaticus***.

NORMAL



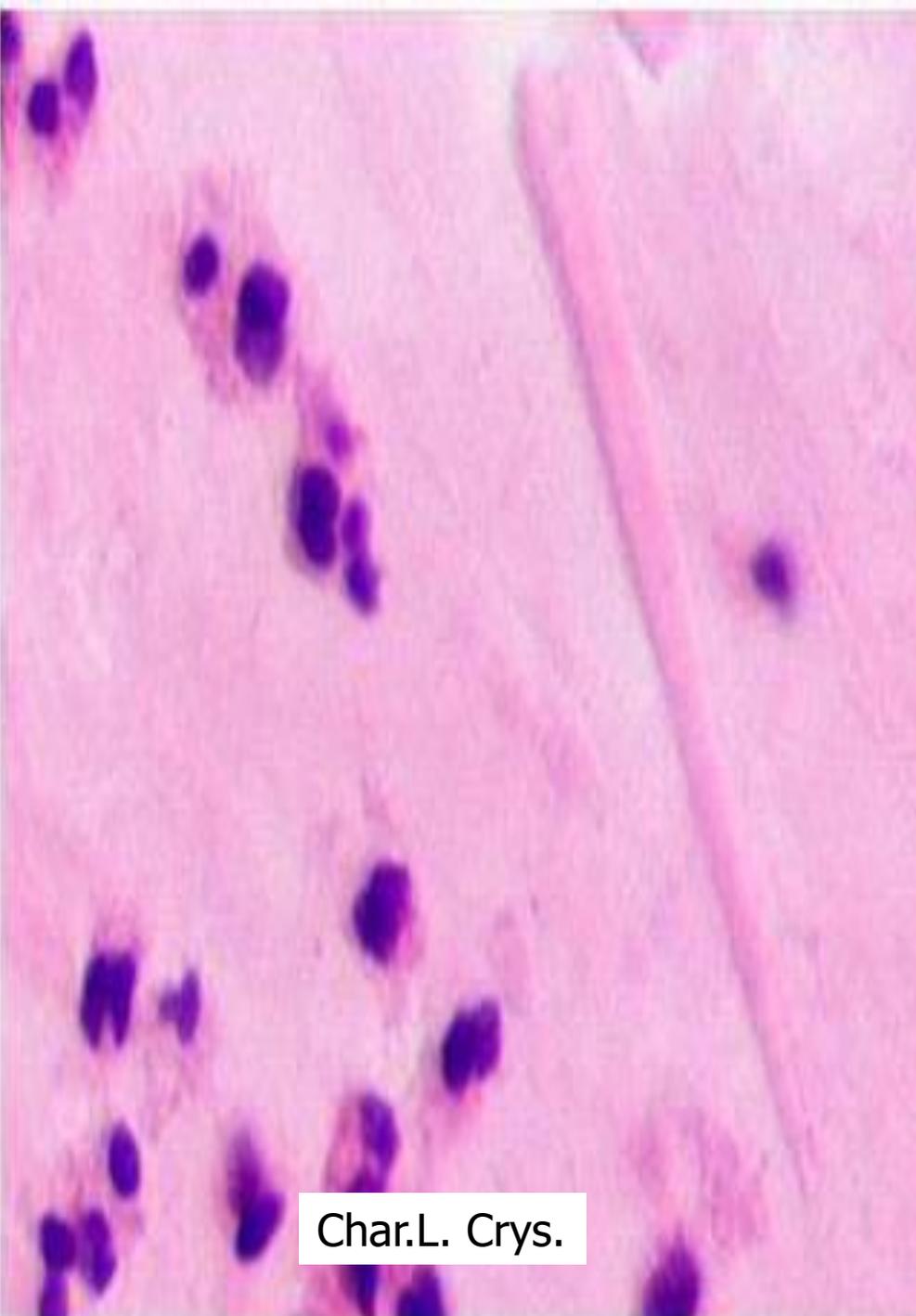
BRONCHIAL ASTHMA







Mucus bronchial cast



Char.L. Crys.



Types of asthma:

Clinically :mild, moderate and severe asthma, but according to presence or absence of an **underlying immune disorder** it divided in to extrinsic & intrinsic asthma.

A- Extrinsic Asthma :

- Immune mediated resulting from exposure to:
 - a- Allergen (Atopic)
 - b- Occupational agents.
 - c- Aspergillosis.
- Early age
- Increased IgE
- Positive family history for asthma or others.
- ± Other allergic disorders.

B- Intrinsic Asthma (non atopic)

- ? Viral infection, ? Aspirin, ? Air pollution , ? Exercise (these factors increase the respiratory hypersensitivity in both normal and asthmatic persons , but in the latter, the response is more severe and sustained).
- Family history for asthma and other allergies are uncommon.
- ~ no increase of IgE.



asthma-histo.mp4