Pathology Lab

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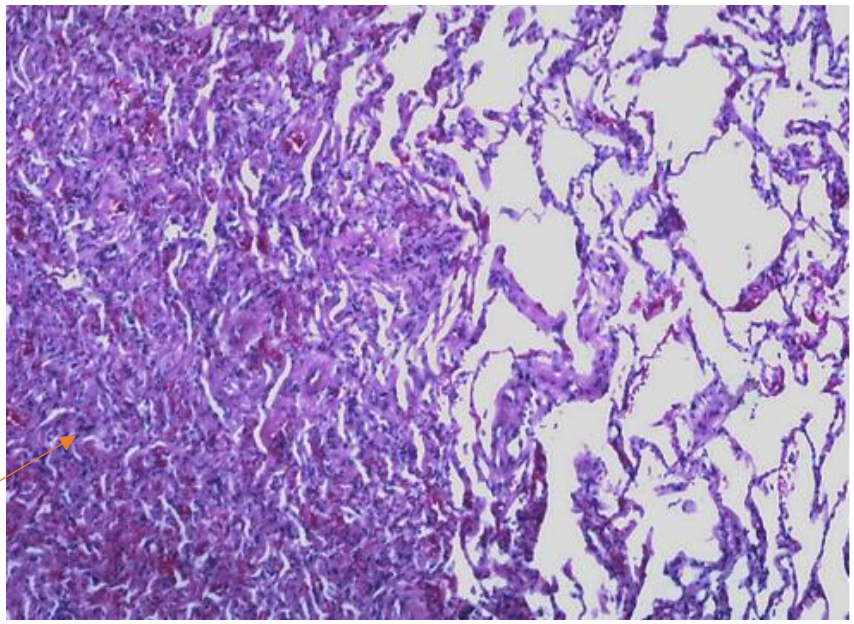
Lecture 1

Atelectasis

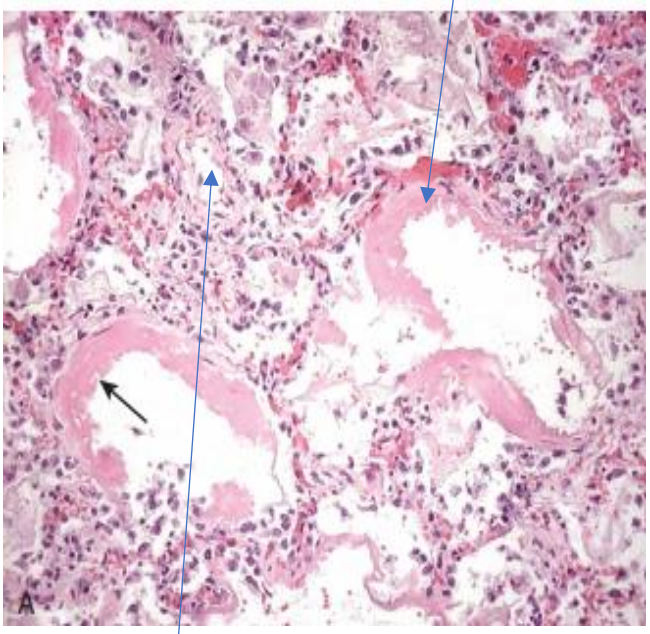


Notice air way collapse

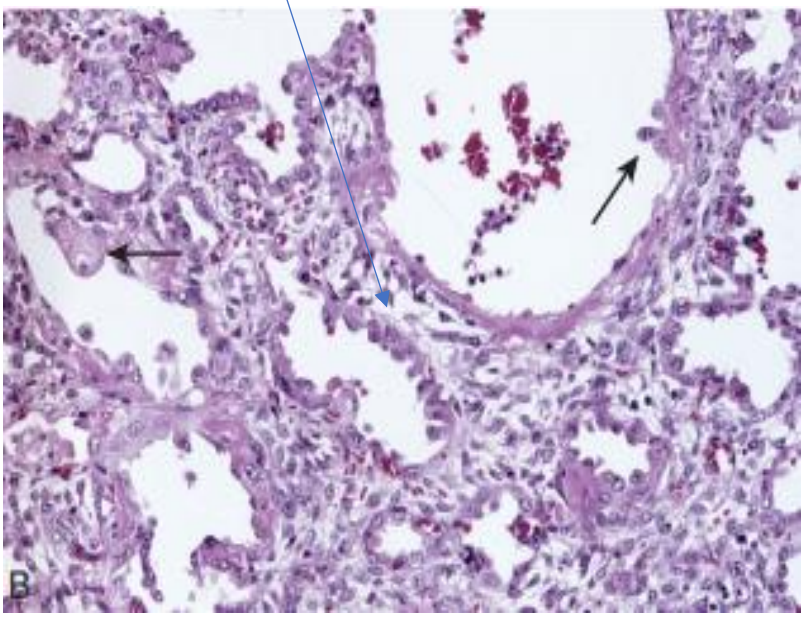
ARDS

Hyaline membrane

The healing stage is marked by resorption of hyaline membranes and thickening of alveolar septa by inflammatory cells, fibroblasts, and collagen.



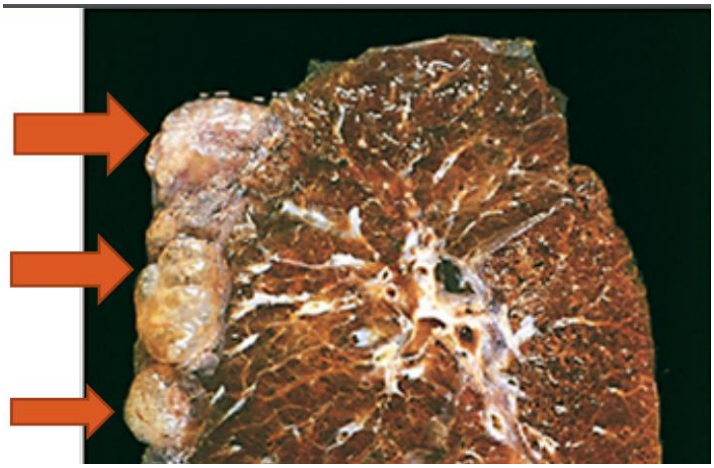
Collapsed alveoli



Numerous reactive type II pneumocytes also are seen at this stage (arrows), associated with regeneration and repair.

Lecture 2

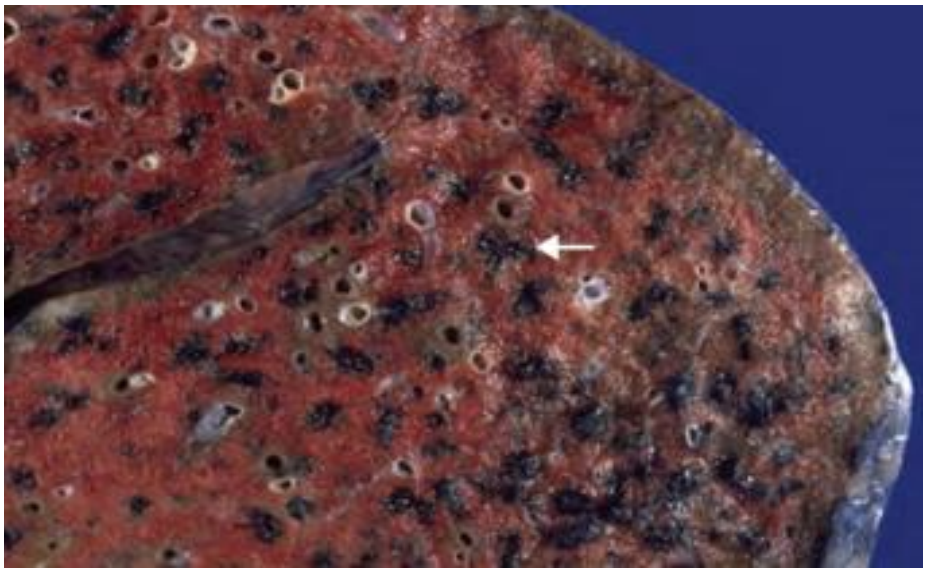
Bullous emphysema with large subpleural bullae



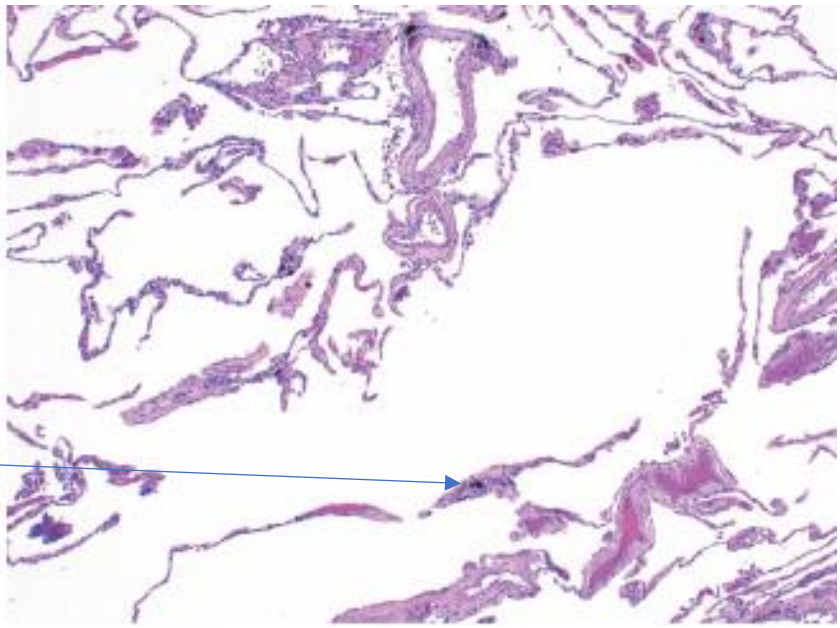
The central lobular emphysema

loss of lung tissue with intense black anthracotic pigmentation

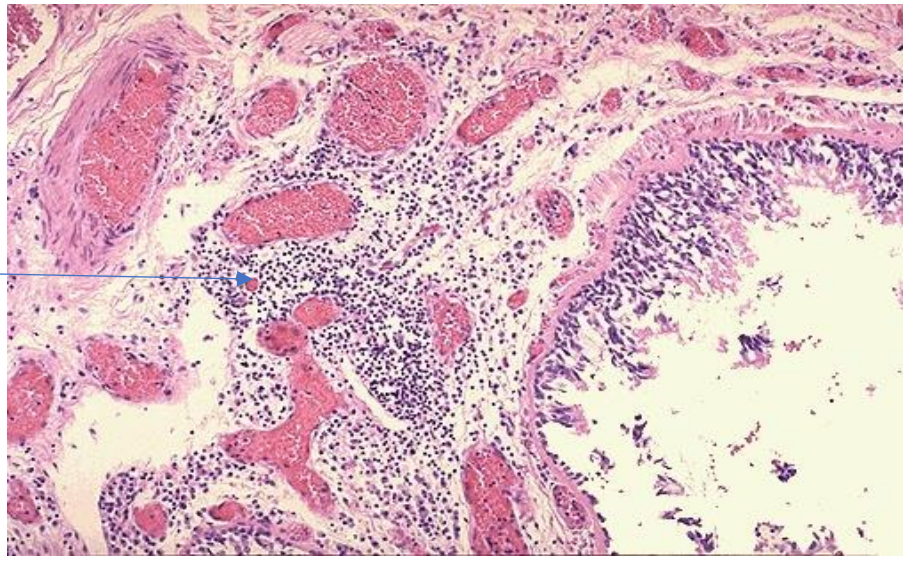
(arrow) is apparent here



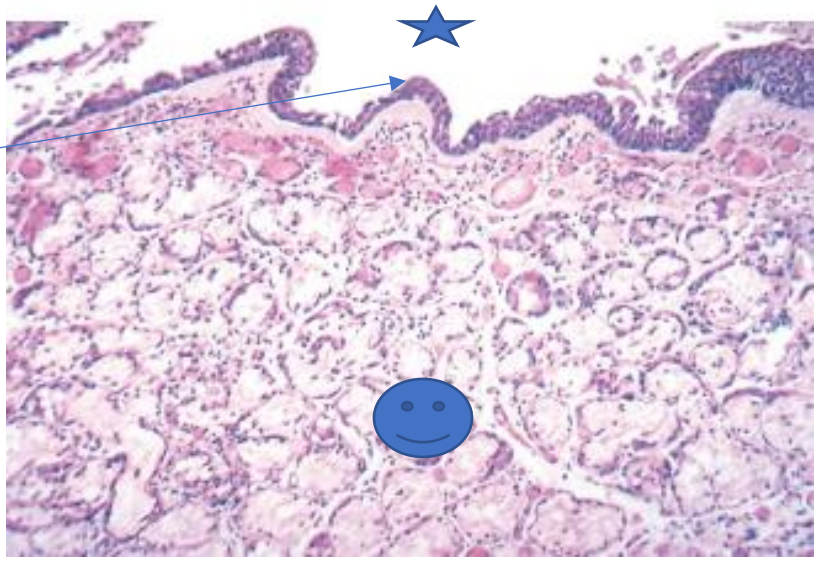
Pulmonary emphysema. There is marked enlargement of the air spaces, with destruction of alveolar septa but without fibrosis. Note the presence of black anthracotic pigment



Chronic bronchitis



Inflammatory cells



Lumen of bronchi.

Focal squamous metaplasia

Which is one of the adaptive mechanisms to protect the respiratory epithelium in smokers.

Enlarged mucus gland approximately twice normal (diagnostic feature in the trachea and large bronchi).

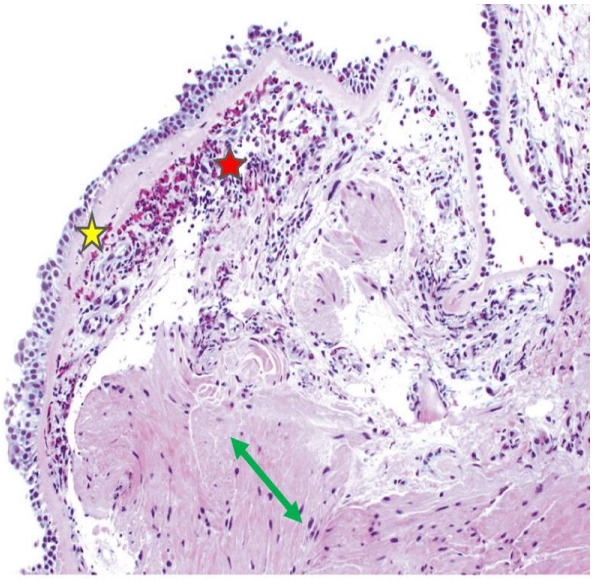
Lecture 3

Asthma

Sub-basement membrane fibrosis.

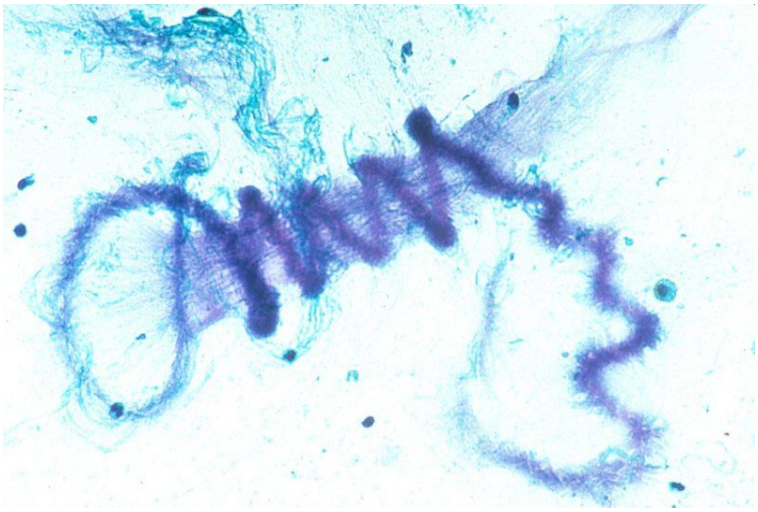
Eosinophilic inflammation.

Smooth muscle hypertrophy and hyperplasia.



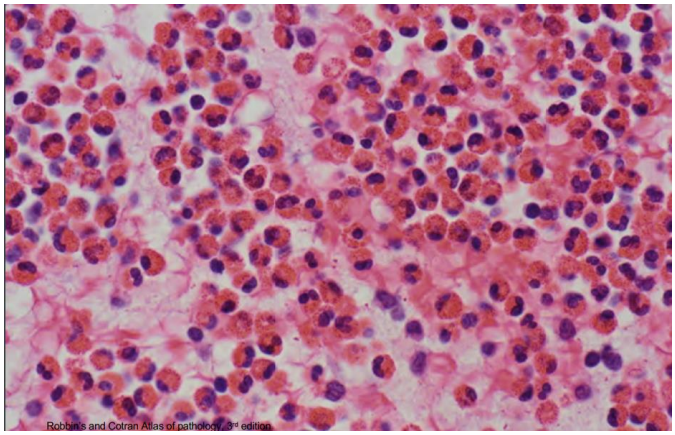
Asthma

mucous plugs contain whorls of shed epithelium called Curschmann spirals.



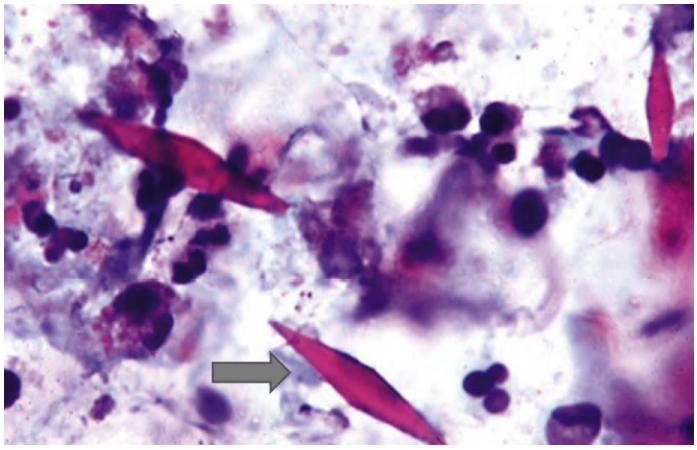
Asthma

Eosinophils are the characteristic cells in asthma



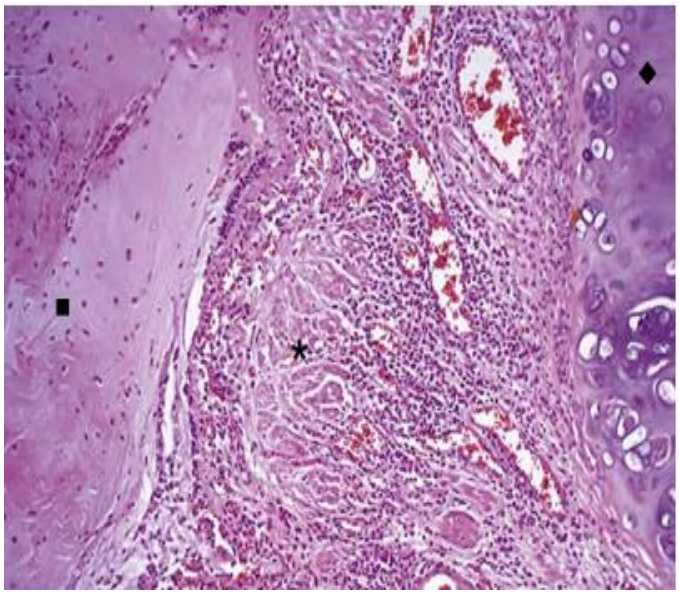
Asthma

Charcot-Leyden crystals: crystalloids made up of the eosinophil protein galectin-10.



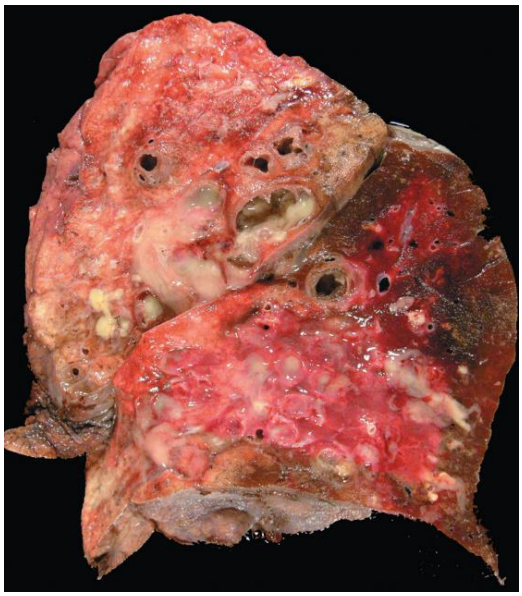
Asthma

Between the bronchial cartilage on the right (■) and the bronchial lumen filled with mucus on the left(♦) is a submucosa widened by smooth muscle hypertrophy, edema, and an inflammatory infiltrate with many eosinophils(★).



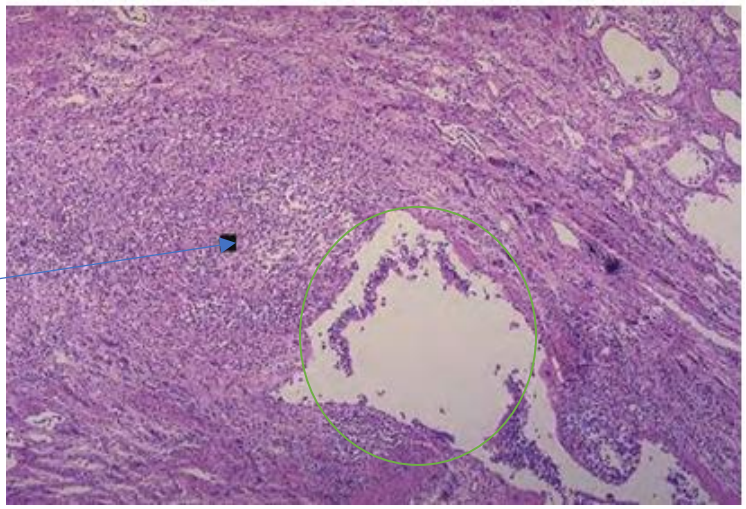
BRONCHIECTASIS

Cut surface of lung shows markedly dilated bronchi filled with purulent mucus that extend to subpleural regions (this patient has cyctic fibrosis).

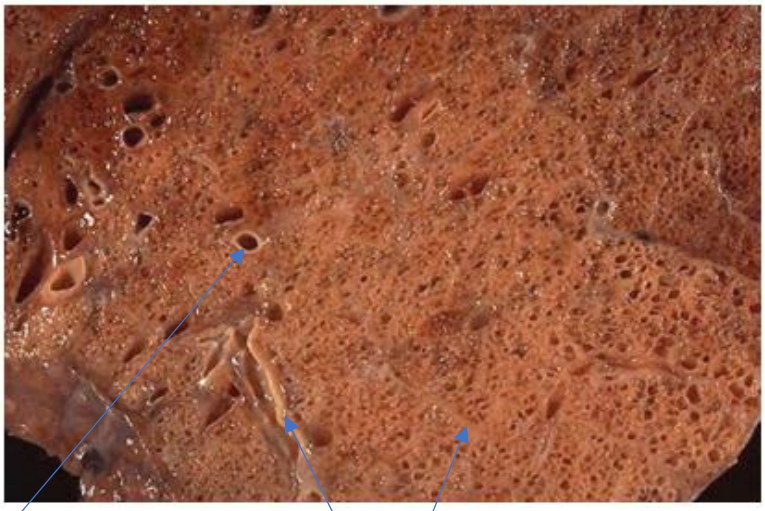


BRONCHIECTASIS

The mid and lower portion of this photomicrograph shows a dilated bronchus in which the mucosa and bronchial wall are not seen clearly because of the **necrotizing inflammation** with tissue destruction and mostly it's desquamated.



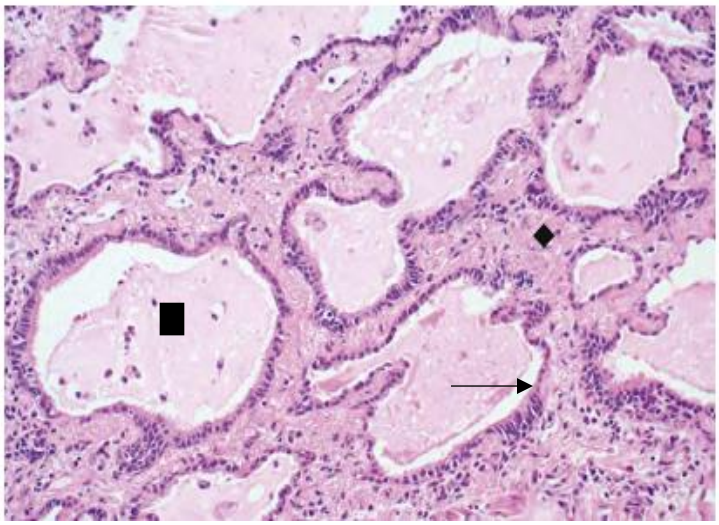
HONEYCOMB LUNG (End-stage lung) 'Gross appearance'



Regardless of the cause of restrictive lung diseases, the majority of cases show the same **gross and microscopic finding** (you can't differentiate between the underlying etiology) many eventually lead to extensive pulmonary interstitial fibrosis.

Irregular residual small dilated airspaces between bands of dense fibrous interstitial connective tissue.

HONEYCOMB LUNG 'Microscopic'



There is dense fibrous connective tissue (◆) surrounding residual airspaces filled with pink proteinaceous fluid(■) These remaining airspaces have become dilated and lined with metaplastic bronchiolar epithelium(→), This pro - duces marked diffusion block to gas exchange, resulting in abnormal ventilation-perfusion ratio and hypoxia.

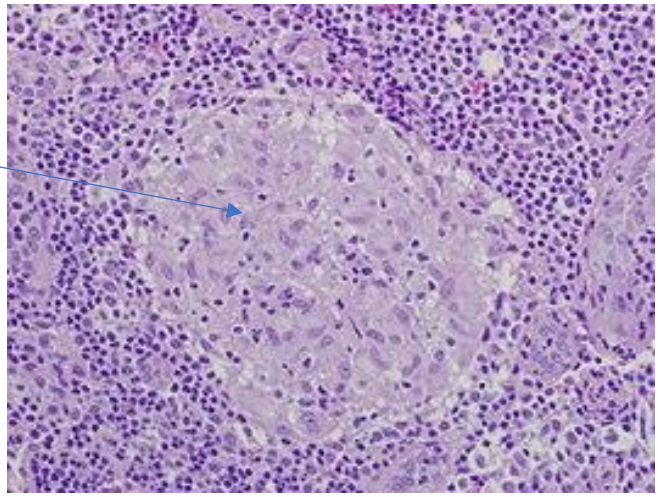
SARCOIDOSIS

Noncaseating epithelioid granuloma

Pale center

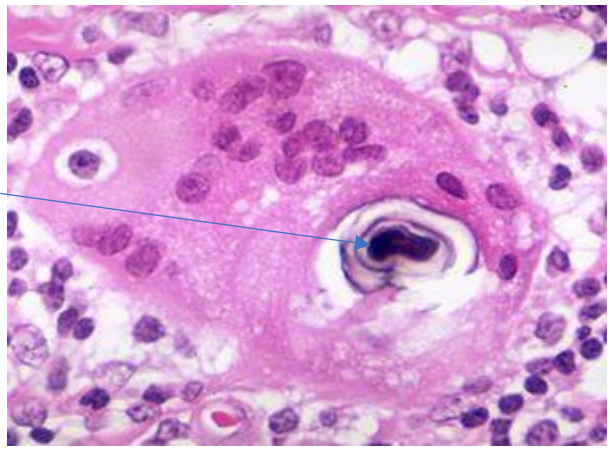
(activated Macrophages)

Dark blue at periphery(T-Lymphocyte).

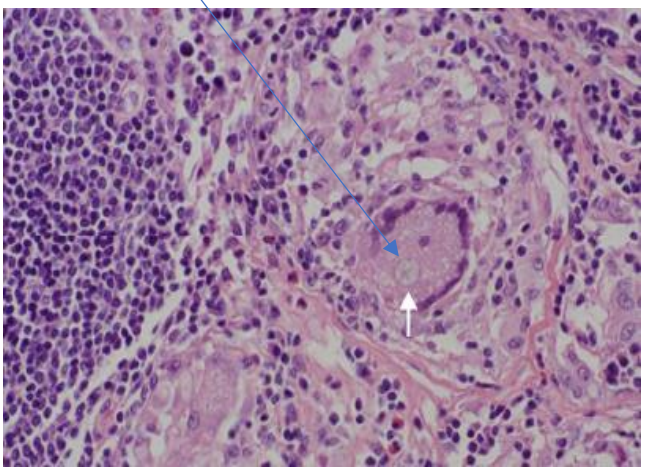


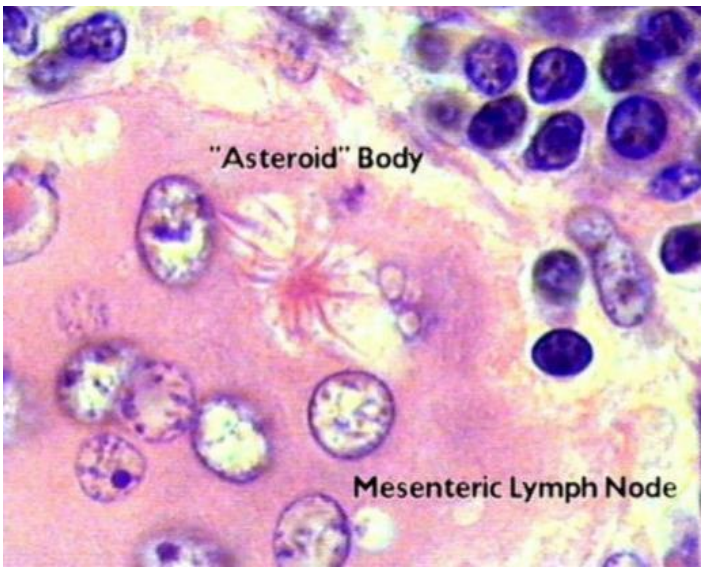
Multinucleated giant cell is engulfing (Schaumann body: laminated concretions composed of calcium and proteins).

***Note: This laminated appearance looks like the onion skin.**



Asteroid bodies: Multinucleated giant cell is engulfing star shaped structure.

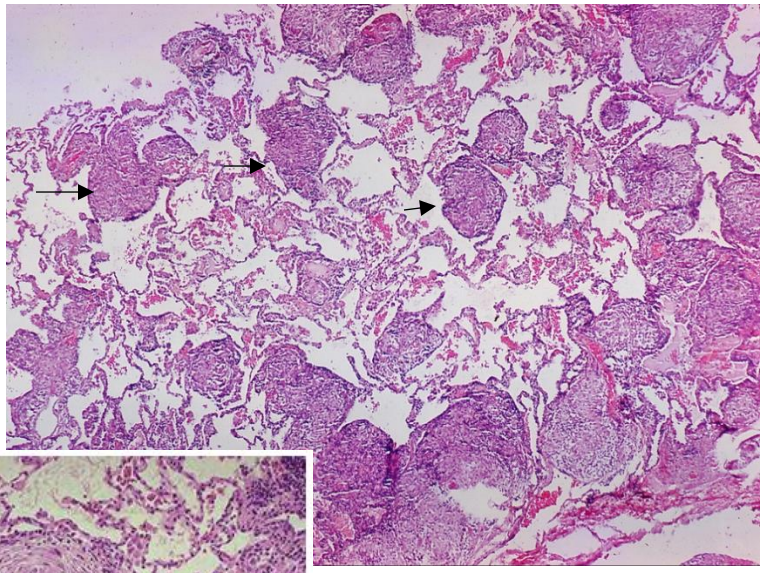




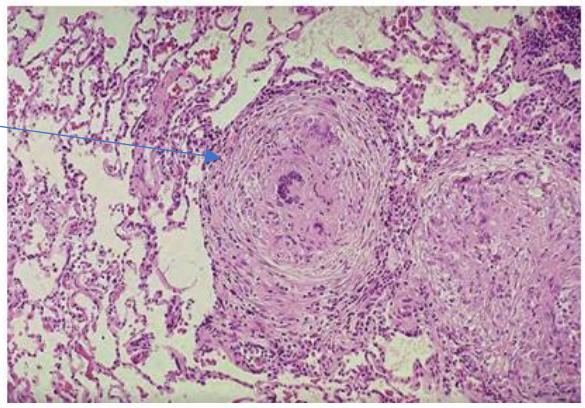
The presence of both bodies is not required for diagnosis of sarcoidosis, and they may also occur in granulomas of other origins.

→Granulomas in the wall of the alveoli (Interstitial) and they **aren't plugging** the alveolar spaces themselves.

Alveolar spaces still patent (we have gases exchange).

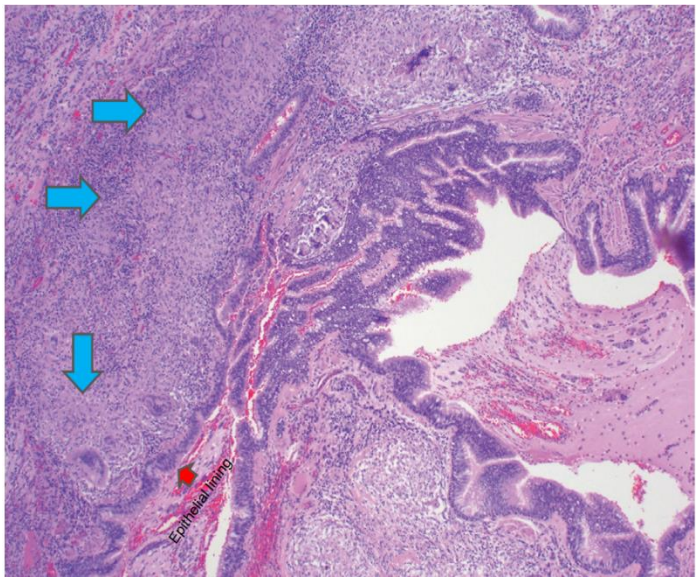


Higher magnification



Peribronchial noncaseating granulomas with many giant cells are present beneath the epithelial lining

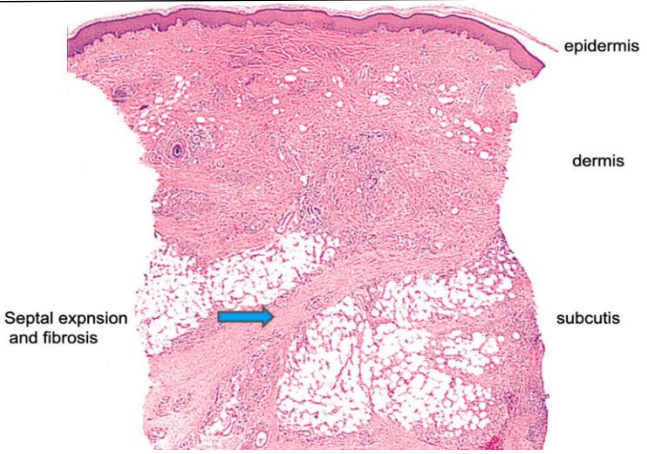
Note: these lesions have some tendency to be localized in the connective tissue around the bronchiole.



Erythema nodosum

Septal expansion and fibrosis by inflammation in subcutaneous tissue (Septal panniculitis).

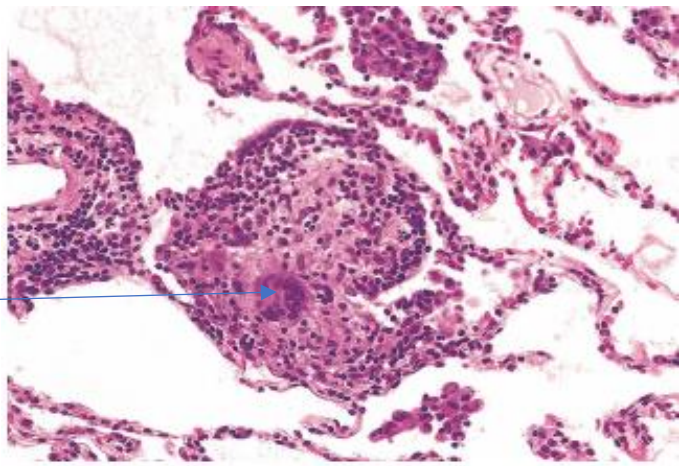
No histopathological finding in dermis and epidermis.



Hypersensitivity pneumonitis

Loosely formed interstitial granulomas surrounded by chronic inflammation and one multinucleated giant cell.

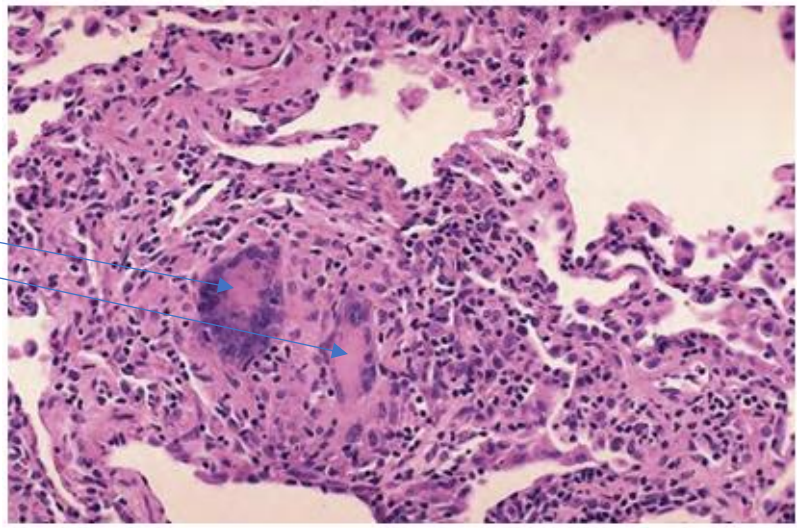
(alveolar spaces still patent)



Loosely formed interstitial granulomas

2 Giant cells surrounded by chronic inflammation

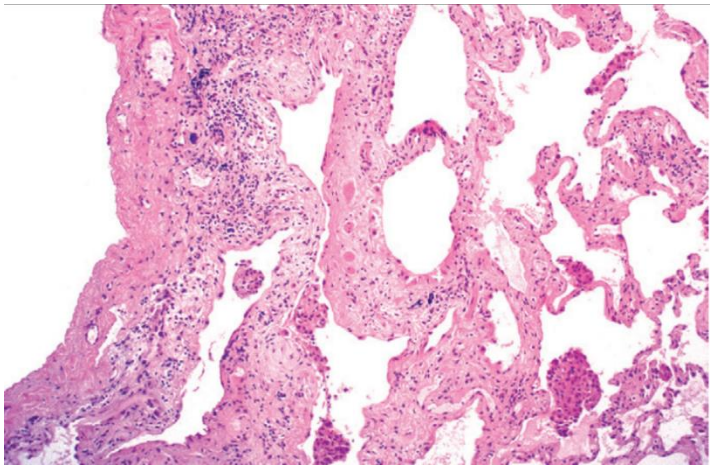
(alveolar spaces still patent)



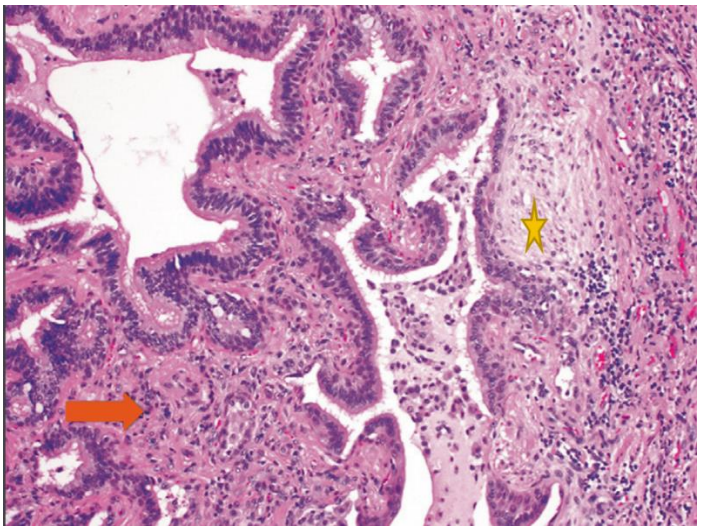
Lecture 5

Idiopathic Pulmonary Fibrosis

Usual interstitial pneumonia (UIP). The fibrosis, which **varies** in intensity, is more pronounced in the subpleural region.



Idiopathic Pulmonary Fibrosis



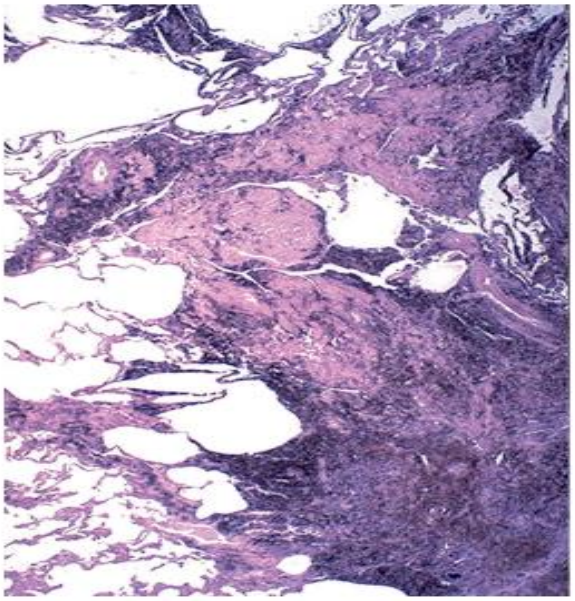
Usual interstitial pneumonia (UIP).

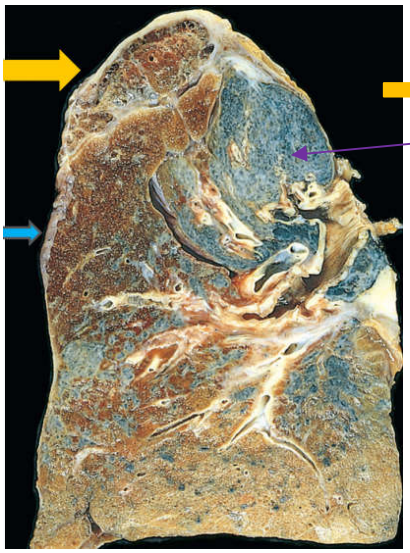
- Fibroblastic focus with fibers running parallel to surface and bluish myxoid extracellular matrix.
- Honeycombing is present.

Note: In advanced cases you may see secondary pulmonary hypertensive change such as intimal fibrosis and medial thickening in pulmonary artery.

Progressive massive fibrosis

A large amount of black pigment is associated with fibrosis.

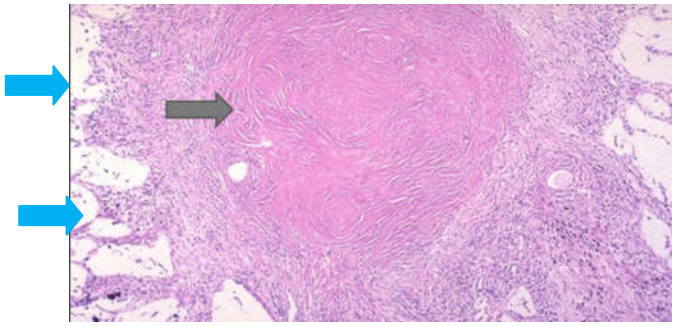




- **Advanced silicosis (gross appearance)**

Scarring has contracted the upper lobe into a small dark mass

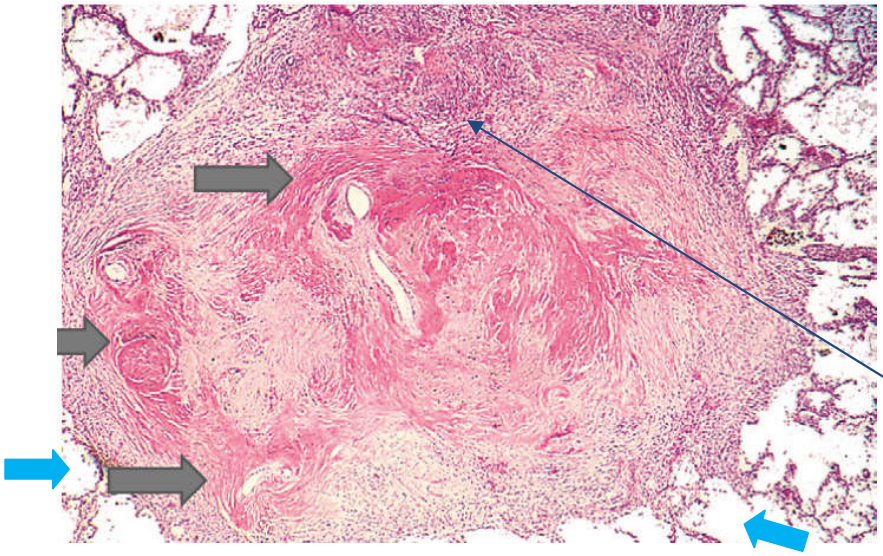
dense plural thickening



- **SILICOTIC NODULE**

Concentrically arranged hyalinized collagen fibers surrounding amorphous center.

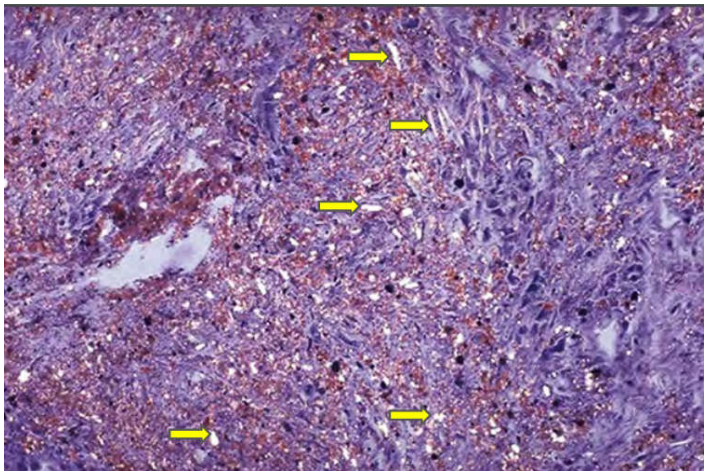
Alveolar spaces still patent.



- **SEVERAL COALESCENT COLLAGENOUS SILICOTIC NODULES**
- The silicotic nodules shown here are composed mainly of :

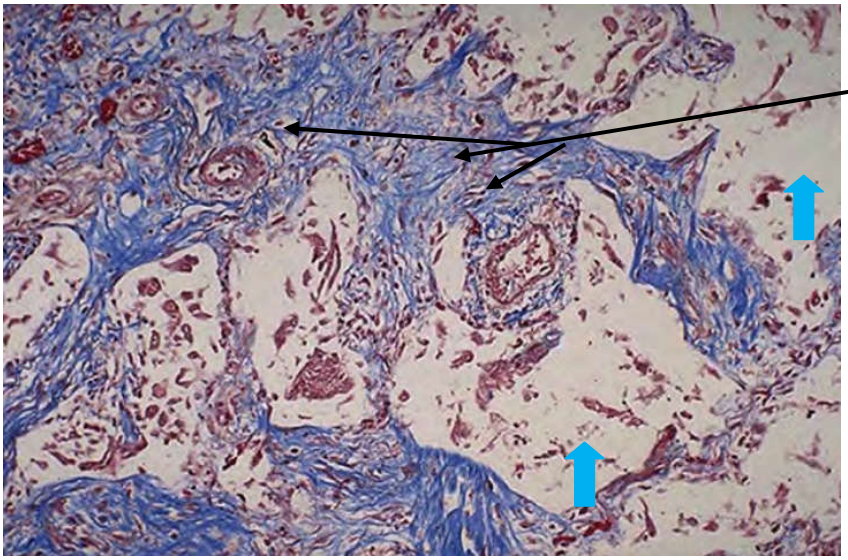
Bundles of interlacing pale pink collagen, and there is a **surrounding inflammatory reaction**.

Alveolar spaces



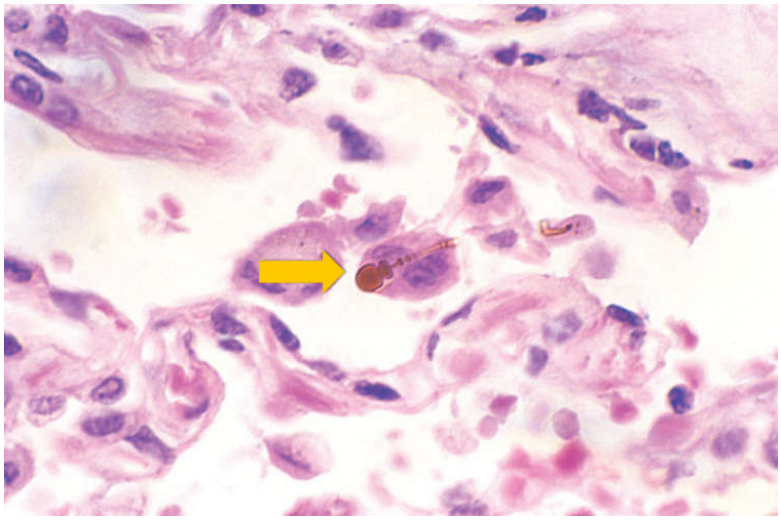
- **Silicosis**
  - Silica crystals under polarized light microscopy

➡ Bright white polarizable crystals of varying sizes are shown here.



- **Asbestosis**
  - diffuse pulmonary interstitial fibrosis (which is the first characteristic feature of asbestosis)
  - The tissue section in this figure is stained by **trichrome stain** which highlights collagen in **blue**, so all the blue areas of the Interstitium are expanded and distorted by **fibroblast proliferation and collagen** deposition which is called pulmonary interstitial fibrosis.

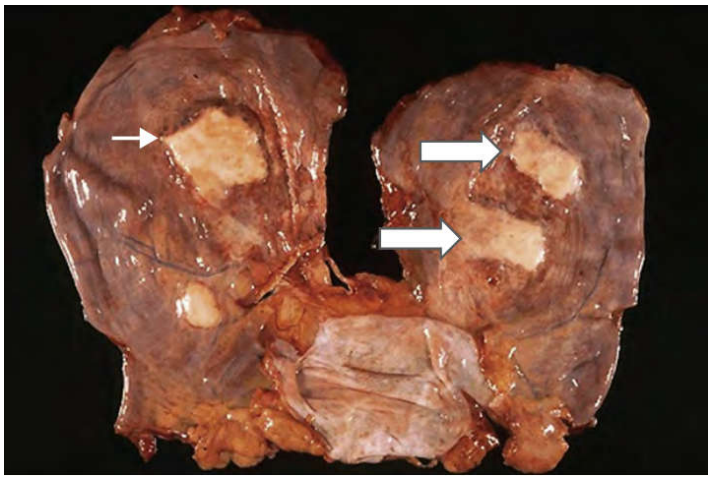
➡ Normal alveolar spaces.



- **Asbestosis**

➡ Asbestos body with beading and knobbed ends, engulfed by pulmonary macrophage (characteristic feature of asbestosis)

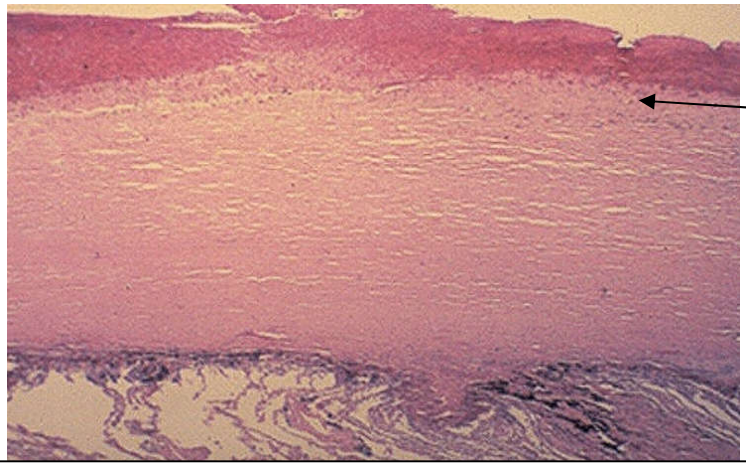
- Asbestos bodies are seen as **golden brown**, fusiform or beaded rods with translucent center.



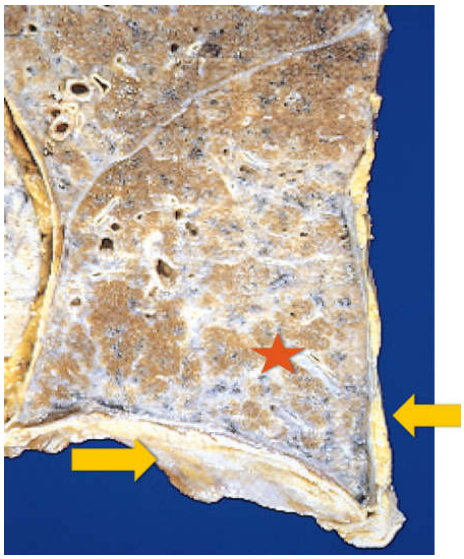
- **Asbestosis**

⇨ Pleural plaques(most common manifestation of asbestos exposure) (gross appearance)

⇨ tan-white multiple pleural plaques on the **pleural aspects of the diaphragm**,they develop most frequently on the anterior and posterolateral aspects of the parietal pleura and over the domes of the diaphragm.



- **Asbestosis**
- Pleural plaques (histologically)
- pleural plaque is composed of dense laminated layers of collagen.



- **Asbestosis**
- Gross appearance of two important findings:

1- ➡ markedly thickened area of the visceral pleura covering the lateral and diaphragmatic surface of the lung.

2- ★ the area under the red star shows sever interstitial fibrosis diffusely affecting the lower lobe of the lung

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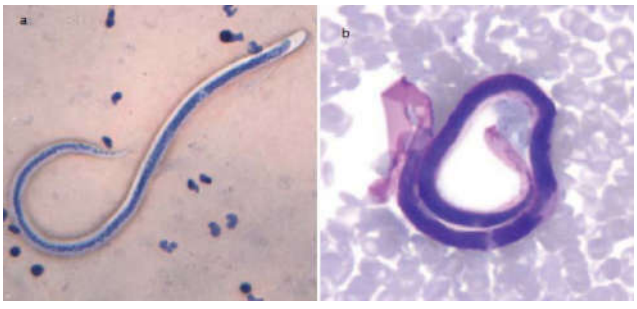
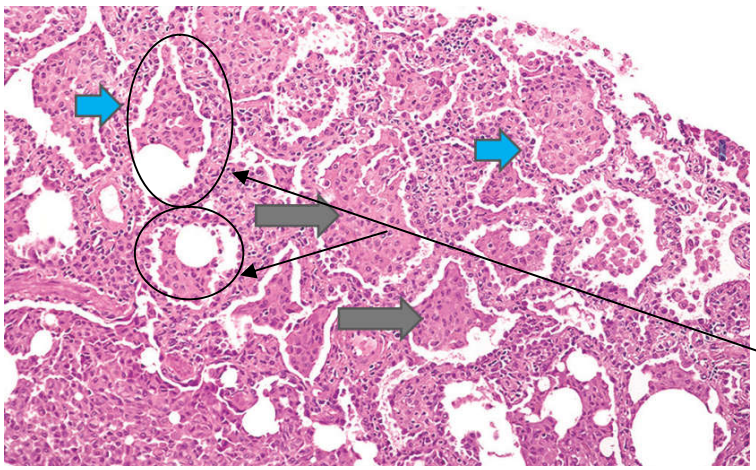


- **seen in Tropical Eosinophilia**
- Two types of microfilaria that can cause Tropical eosinophilia.



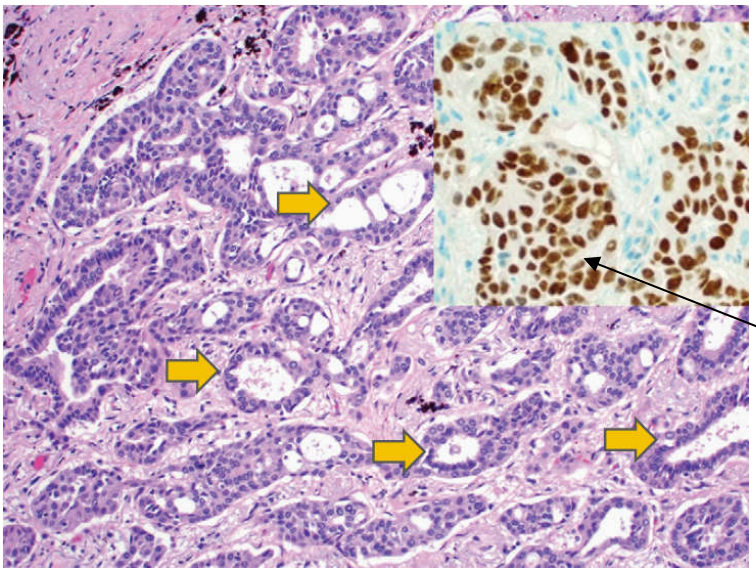
- **Desquamative Interstitial Pneumonia**

➡ Collections of large numbers of smoker's macrophages(pigmented macrophages) within the air spaces.

➡ Mildly expanded alveolar septa by lymphocytes and mild fibrosis

- You can see both a small air space and collections of smoker's macrophages IN each circle .

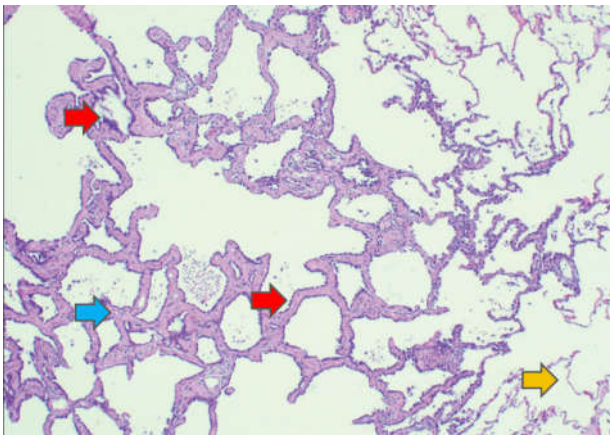
Lecture 7



- **Adenocarcinoma**

➡ Many proliferating gland like structures, these glands (acini) are surrounded by desmoplastic reaction.

- The small box at the right corner shows thyroid transcription factor 1(TTF-1) positivity(brown nuclear staining)
- Usually we use TTF-1 immune stain in histopathology lap to highlight tumors of lung origin , and it shows positive expression in the majority of pulmonary adenocarcinoma.

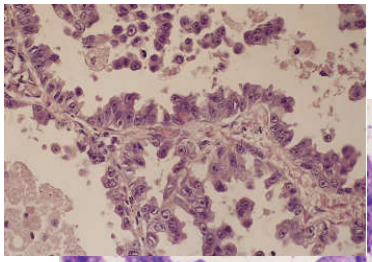


- **Atypical adenomatous hyperplasia (AAH)**
  - Precursor lesion for ADENOCARCINOMA

➡ Almost normal alveolar **walls** (look how thin it is)

➡ Proliferation of hyperchromatic cuboidal epithelium lining with some degree of cytologic atypia, this epithelia lining lines up the alveolar walls.

➡ Mild interstitial fibrosis.



- **Adenocarcinoma in situ (AIS) -mucinous subtype.**
  - Atypical adenomatous hyperplasia (AAH) progress into adenocarcinoma in situ (AIS) in a stepwise fashion

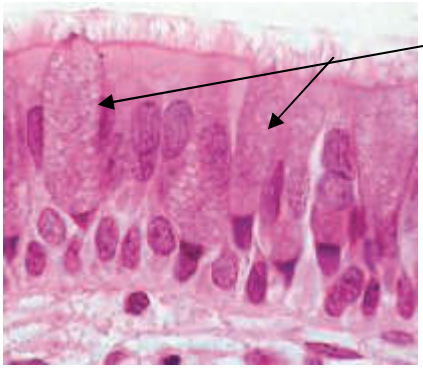
➡ Monolayer proliferation of atypical cells, these atypical cells are proliferation along the alveolar septa with (- no destruction, no desmoplasia,no invasion-) of the preexisting alveolar septa.

➡ The preexisting alveolar septa.

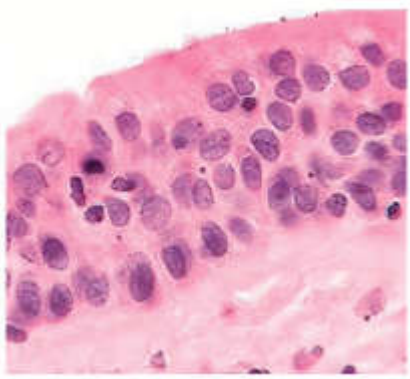
- Atypical cells (nuclear enlargement , hyperchromasia)

➡ Apical mucin

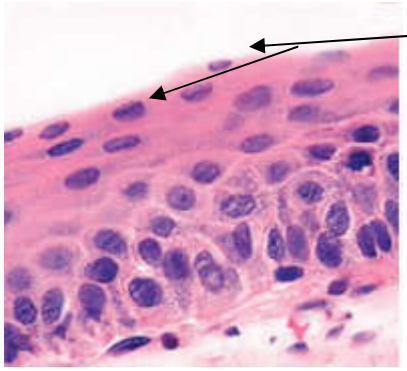
- So if you see this slide in the exam the first thing you can notice is the(★alveolar spaces) but the alveolar septa in this section is somehow abnormal (you can notice hyperchromasia and nuclear enlargement) so you are looking at atypical cells that is only of one layer (Monolayer proliferation) and the alveolar septa is not destructed(no invasion). SO it is adenocarcinoma in situ , and as you can see apical mucin it is mucinous subtype.



- **Goblet cell hyperplasia**
  - One of the earliest and mild change in **smoking-damaged respiratory epithelium**.

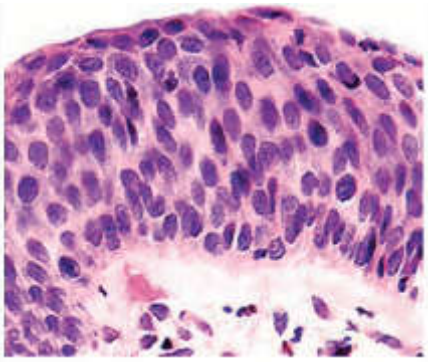


- **Basal cell (or reserve)hyperplasia**
- Another **smoking related** adaptive response

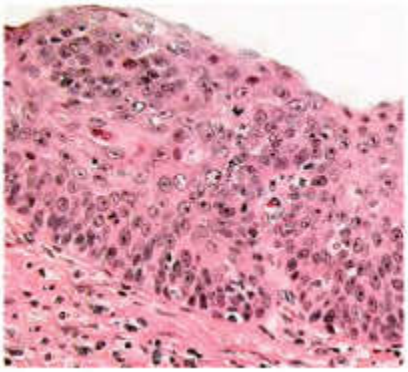


- **Squamous metaplasia**
  - The normal respiratory epithelium (ciliated, pseudostratified columnar **epithelium)** is replaced by squamous epithelium.

-can be mild, moderate or sever.



- **Squamous dysplasia.**
  - Characteristic by the presence of disorder squamous epithelium , with loss of nuclear polarity ,nuclear hyperchromasia ,pleomorphism and mitotic figures.



- **Carcinoma in situ (CIS)**
  - Full thickness of Squamous epithelium showing cytologic atypia an lacking the basement membrane destruction , happened immediately before invasive squamous cell carcinoma

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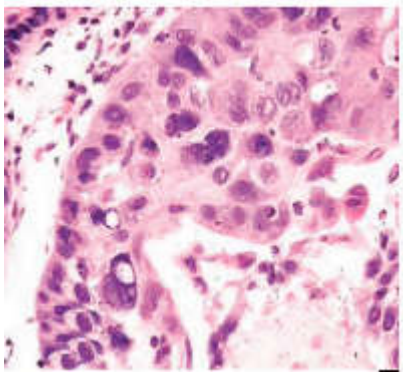
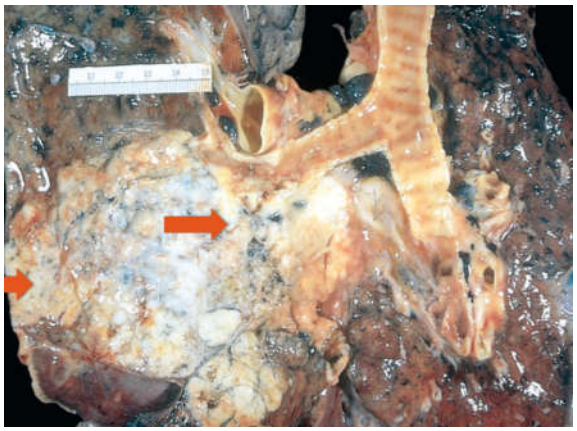


- **Invasive squamous cell carcinoma**

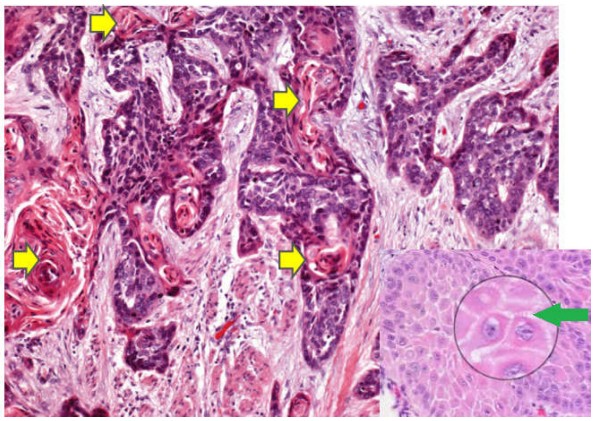
Invasive squamous cell carcinoma lesions show cytologic atypia and basement membrane invasion.

And these lesions can be classified into well-differentiated, moderately differentiated and poorly differentiated according to the cytologic feature and the squamous cell differentiation in each type.



- **squamous cell carcinoma (gross appearance)**

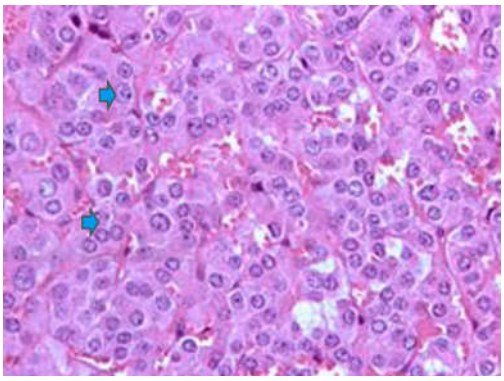
➡ Pale yellow white central area accounting for lung carcinoma, that start centrally and then grows to peripheral lung parenchyma.



- Well differentiated **squamous cell carcinoma** showing keratin pearls and intercellular bridges.

⇨ Keratin pearls.

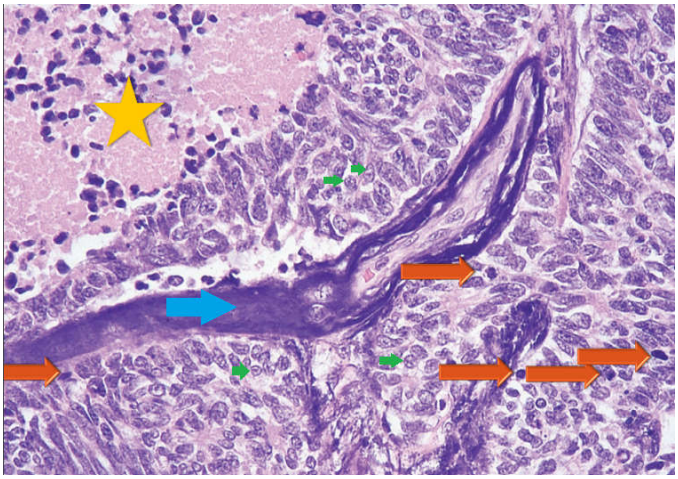
⇨ Intercellular bridges(desmosomes)



Lecture 8

- **Small Cell Carcinoma.**

➡ Monomorphic proliferation of a relatively small calls with finely granular chromatin with salt and pepper appearance.



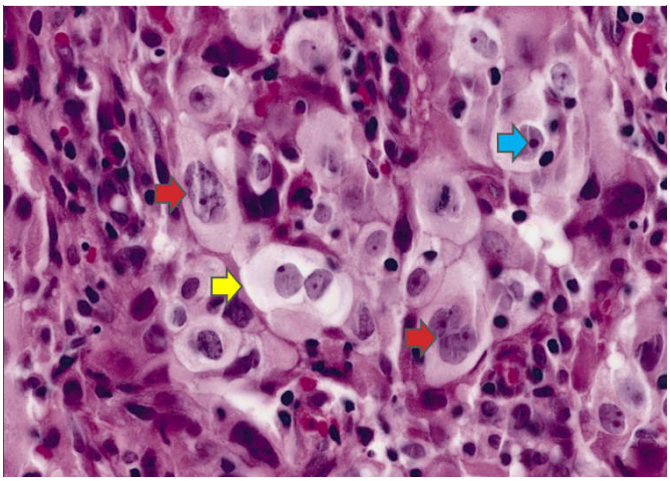
- **Small Cell Carcinoma**

⇨ Small round to oval blue cell with salt and pepper nuclei.

➡ Mitotic figures.

★ Area of extensive necrosis

➡ Azzopardi effect: basophilic staining of vascular walls due to encrustation by and from necrotic tumor cells.



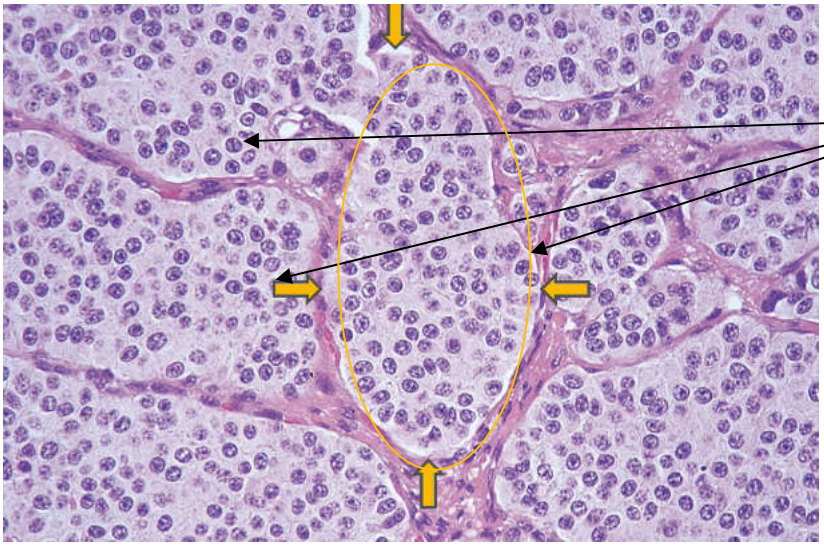
- **LARGE CELL CARCINOMA**

➡ Large cells

➡ Large pleomorphic nuclei that is different in size and shape.

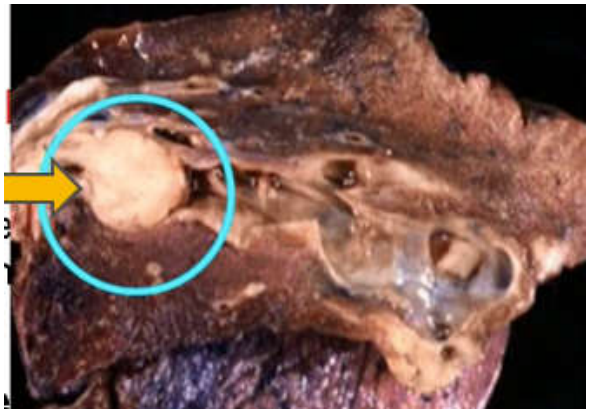
➡ Prominent nucleoli

- There is no glandular or squamous cell differentiation.



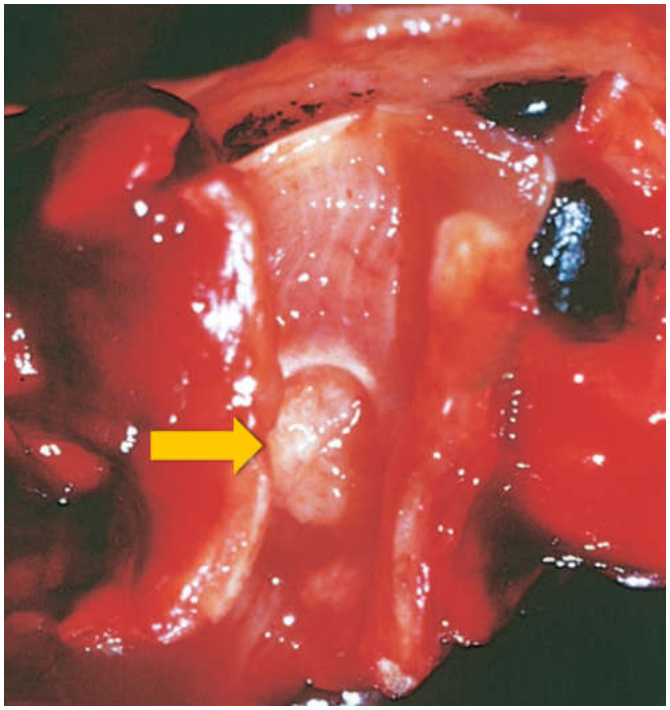
- **Typical CARCINOID**
- Multiple nests each contain uniform cells that have regular round nuclei with "salt and pepper "chromatin , no increase mitotic activity and no necrosis

➡ One nest.



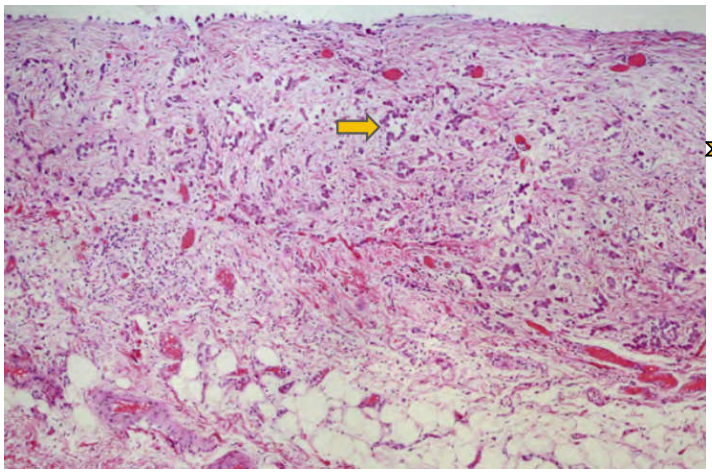
➡ • **obstructing polypoid tumor with the lumen of a bronchus .**

- **It is a growth pattern of CARCINOID tumors.**



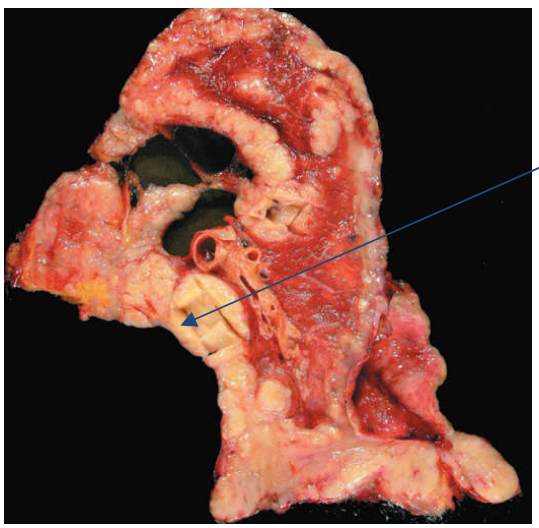
- **CARCINOID TUMOR (Bronchial carcinoid)**

- Carcinoid tumor growing as a spherical mass protruding into the lumen of the bronchus.



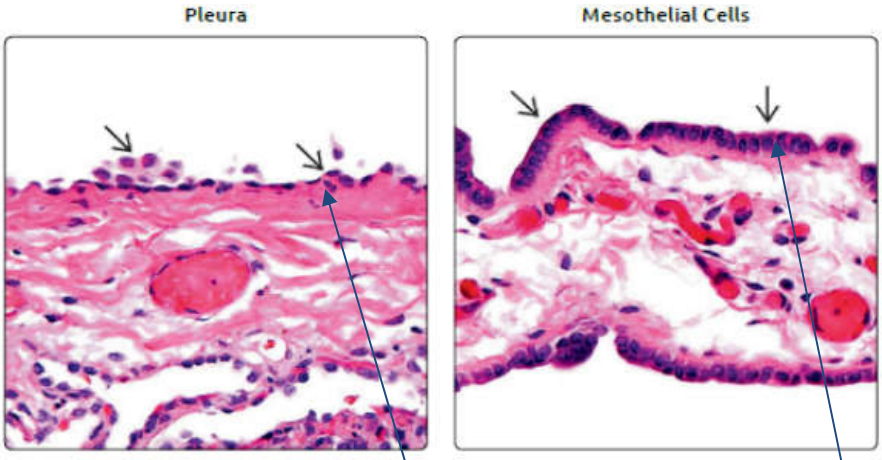
MALIGNANT MESOTHELIOMA

plump, rounded cells forming gland like configurations.

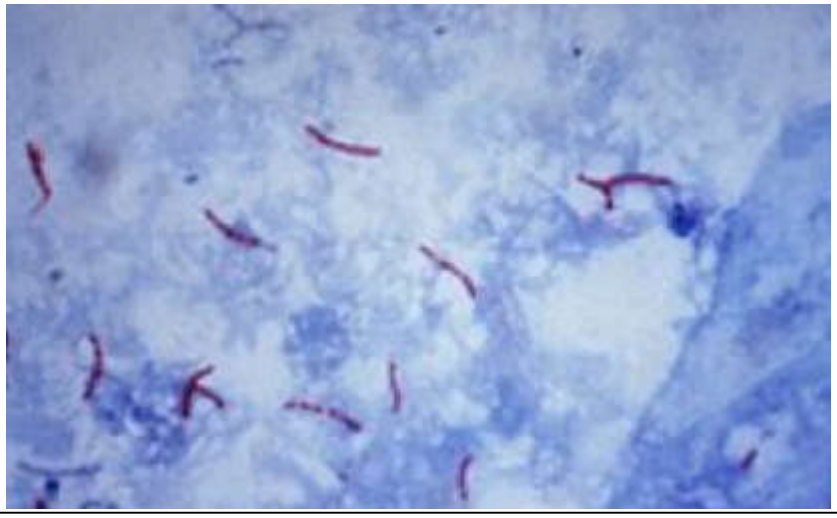


Autopsy of MALIGNANT MESOTHELIOMA

At autopsy, the affected lung typically is ensheathed by a layer of yellow-white, firm, variably gelatinous tumor that obliterates the pleural space.



- **Normal mesothelial cells**
- Pleura is lined by single layer of normal mesothelial cells these cells are almost flat cells (you may descrip them as small cuboidal cells) with eosinophilic cytoplasm And indestinct nucler features.
- Same mesothelial lining but the cells are much more cuboidal.



Lecture 9

this figure shows Ziehl Neelsen stain tissue sections, you can see cylindrical rod stained with purple colour,those are the bacilli of mycobacterium tuberculosis

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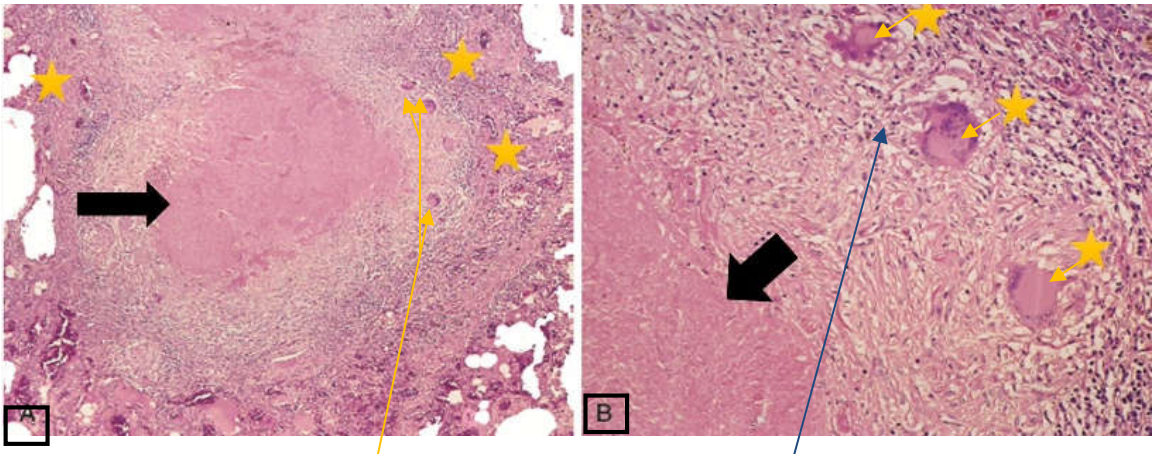
Primary Tuberculosis gross appearance

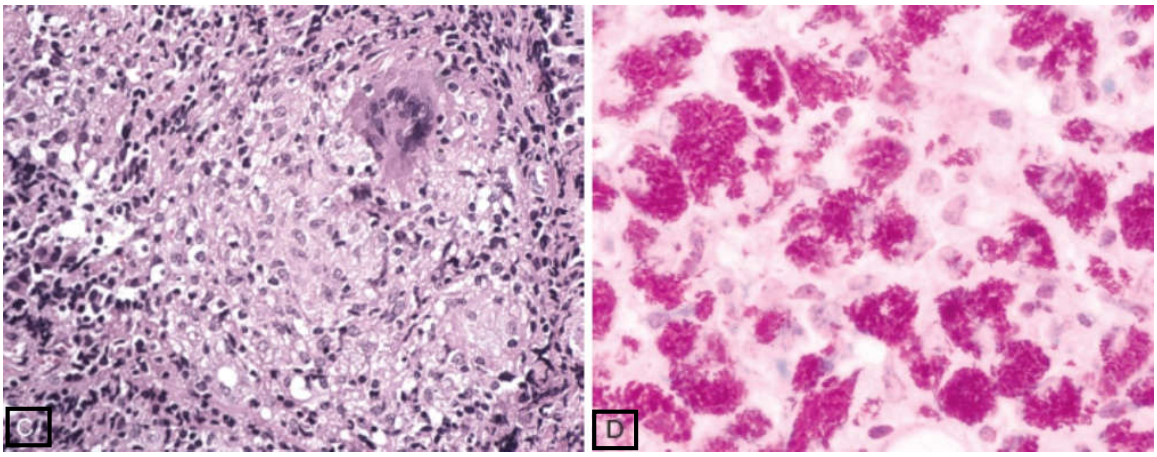
Ghon focus (in the lower part of the upper lobe) : a 1-cm to 1.5-cm area of gray-white inflammatory consolidation emerges during the development of sensitization,and in the majority of cases the center of this focus undergoes necrosis.

Hilar lymph node shows caseation,as tubercle bacilli, free or within phagocytes, travel via the lymphatic vessels to regional lymph nodes.

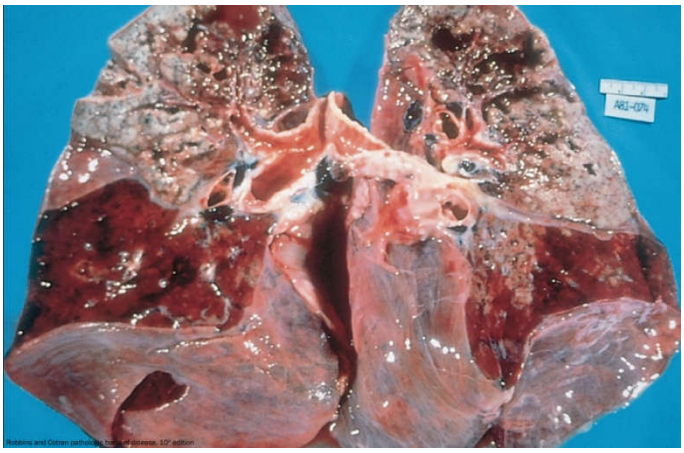
- **Ghon complex** :Combination of parenchymal and nodal lesions.



- **Tuberculosis**
- a. Low magnification
- b. Higher magnification for the same focus.
- ➡ **Central granular caseation** (pink amorphous tissue representing caseous necrosis)surrounded by epithelioid and **multinucleated giant cells (Langhans giant cells) (yellow stars)** this is the usual response in individual who develop cell mediated immunity to the organism.



- **Tuberculosis**
- C. TB granuloma but with no central caseation (occasionally in immune competent patient).
- D.Acid fast stain shows sheets of macrophages packed with mycobacteria,this specimen is from an immunocompromised patient.



-Secondary pulmonary tuberculosis

-The upper parts of both lungs are riddled with gray-white areas of caseation and multible areas of softening and cavitations.

-Secondary pulmonary tuberculosis classically localized to the apex of one or both upper lobes

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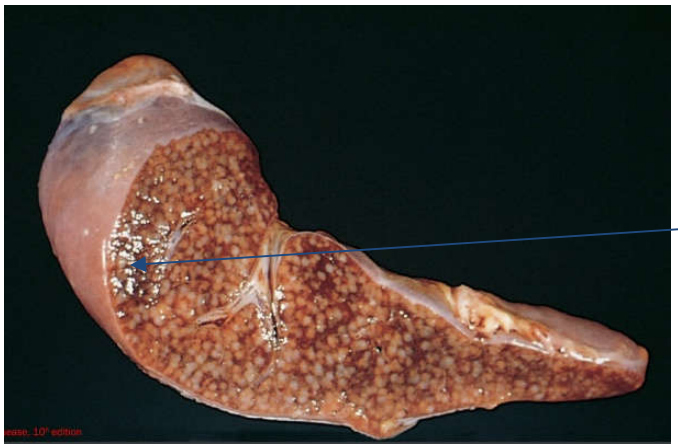
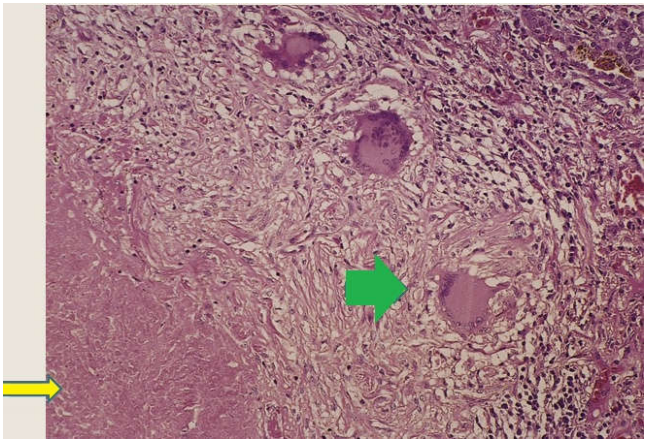


Systemic miliary tuberculosis of the spleen

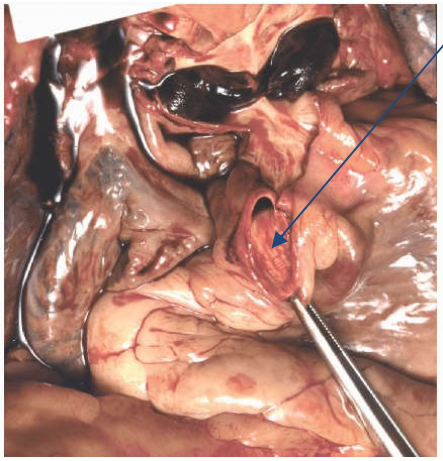
The cut surface shows numerous gray-white granulomas .



Tuberculosis.

⇨ pink amorphous tissue representing caseous necrosis.

⇨ epithelioid cells and Langhans giant cells.



Large saddle embolus from the femoral vein lying astride the main left and right pulmonary arteries

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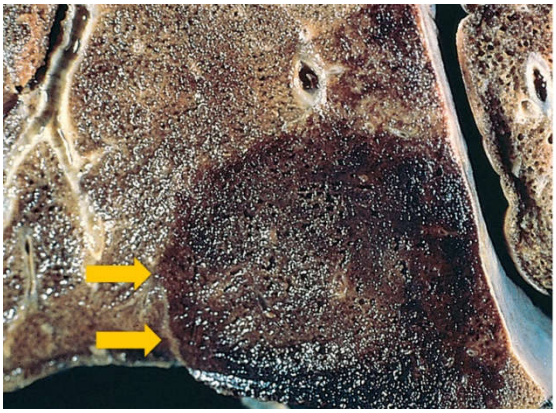
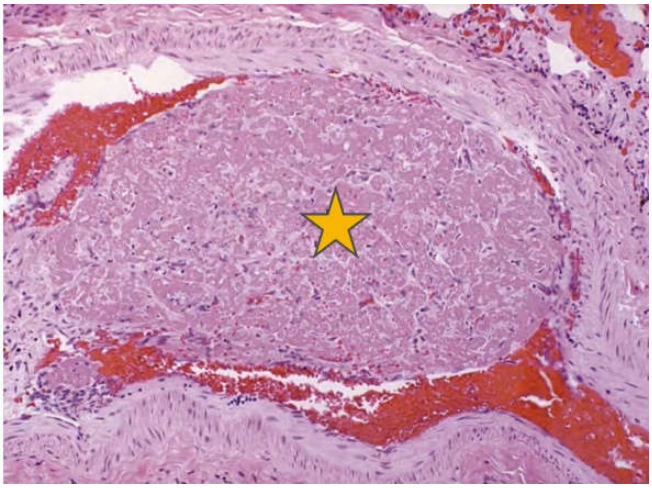
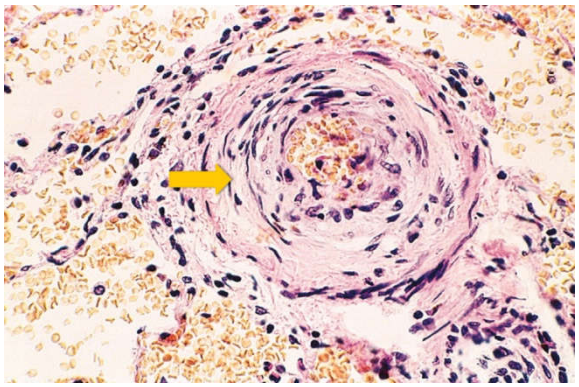


A small, roughly wedge-shaped hemorrhage pulmonary infarct of recent occurrence.

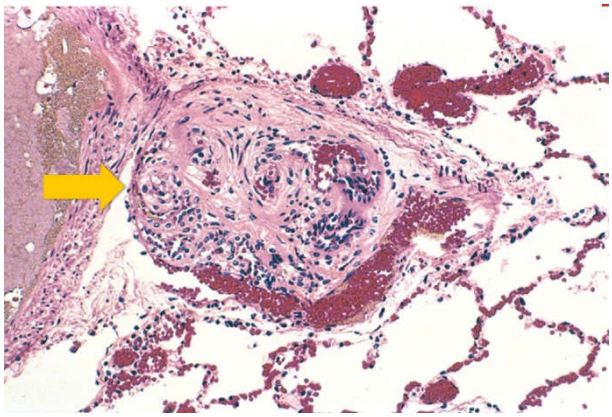
Gross appearance .



Thromboembolism In a peripheral pulmonary arterial branch



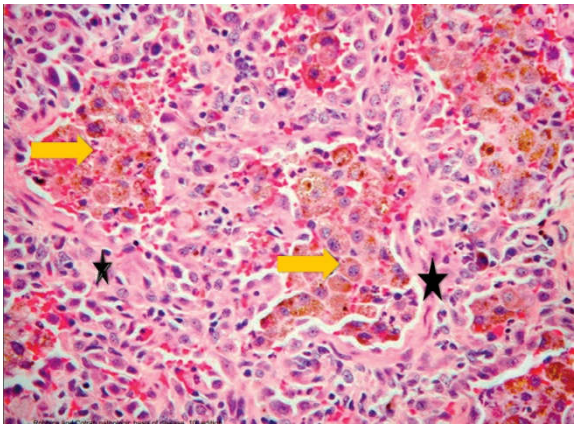
Medial hypertrophy affecting an arteriole



Plexiform lesion seen in small arteries

A tuft of capillary formations is present, producing a network that spans the lumens of dilated thin-walled small arteries.

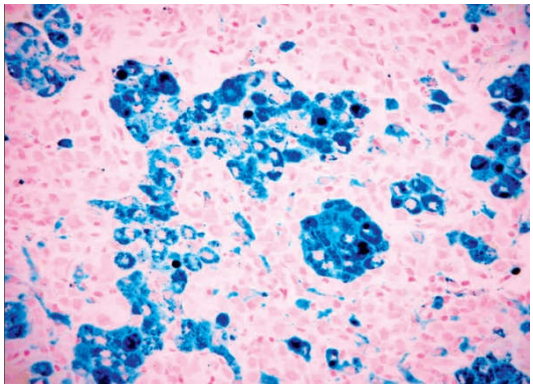
1



Diffuse alveolar hemorrhage syndrome

Intraalveolar hemosiderin-laden macrophages.

★ Background of thickened fibrous septum.



Diffuse alveolar hemorrhage syndrome

The same tissue**(1)** but has been stained with **Prussian blue**, an iron stain that highlight the abundant intracellular hemosiderin.