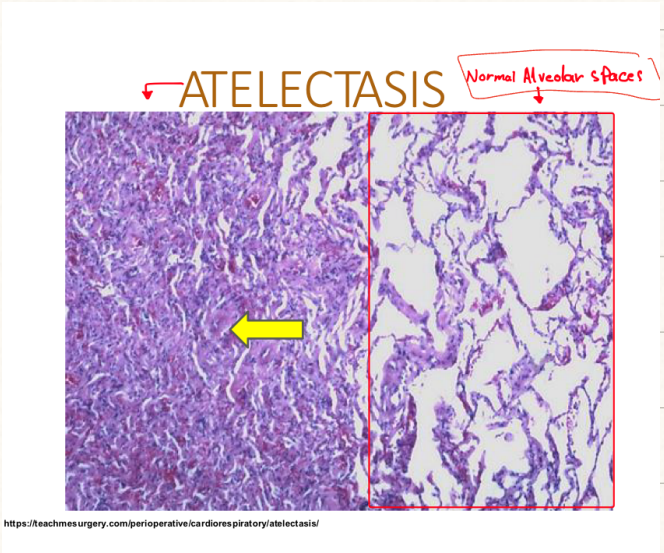
Pathology

Lab

لجنة الطب والجراحة

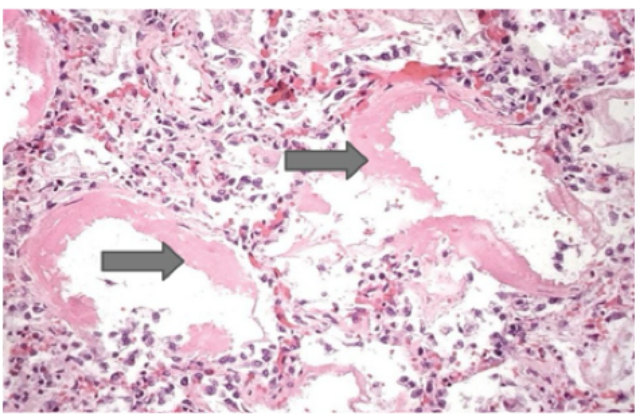
L1



https://teachmesurgery.com/perioperative/cardiorespiratory/atelectasis/

Atelectasis (except when caused by contraction) is potentially reversible and should be treated promptly to prevent:

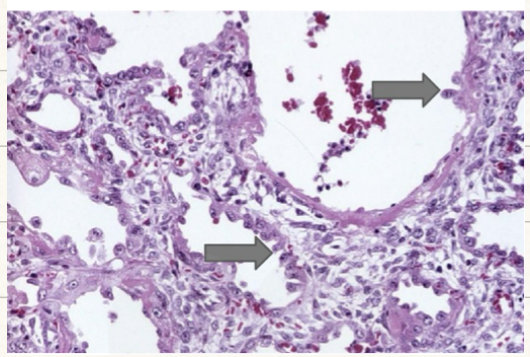
- Hypoxemia (low oxygen in blood)
- Superimposed infection of the collapsed lung.



Acute phase:

- Most characteristic finding: presence of hyaline membranes

ɣ composed of fibrin-rich edema fluid mixed with necrotic epithelial cell debris, (similar to neonatal respiratory distress syndrome)

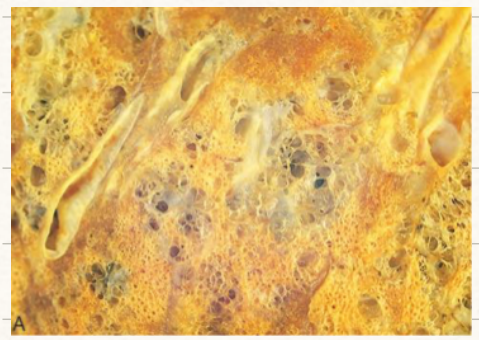


Organizing (late) stage:

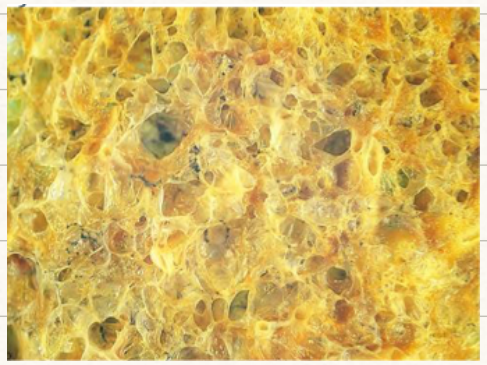
- Proliferation of Type II pneumocytes (for repair)
- Intraalveolar fibrosis due to organization of fibrin-rich exudates
- Marked thickening of alveolar septa because of:
- Proliferation of interstitial cells
- Collagen deposition

L3

Emphysema



Centriacinar



Panacinar

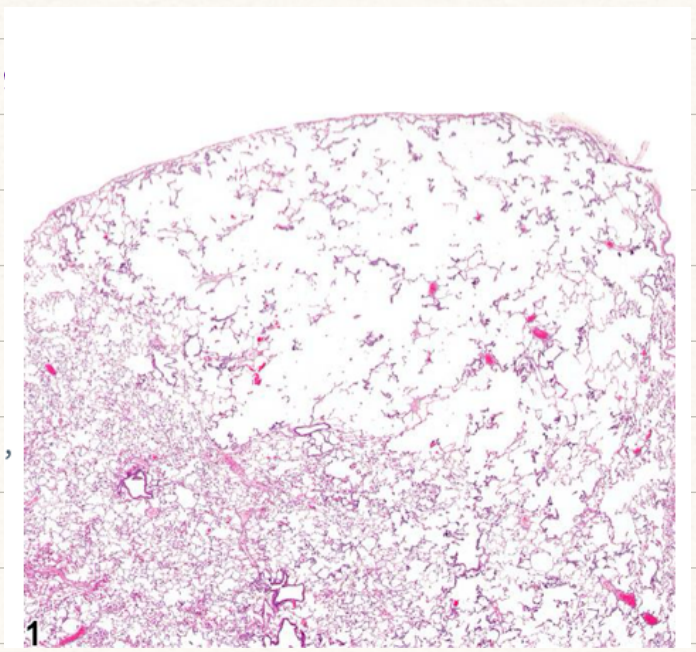
Centriacinar emphysema: in the upper lobes, most
common type in cigarette smokers(central or proximal parts of the acini, distal alveoli are spared.)

A less impressive. Until late stages, the lungs are a deeper pink than in panacinar emphysema and less voluminous

Panacinar emphysema: in the lower lung zones &
associated with α1-anti-trypsin deficiency. (the acini are
uniformly enlarged from the level of the respiratory
bronchiole to the terminal blind alveoli).

B pale, voluminous lungs that often obscure the heart.

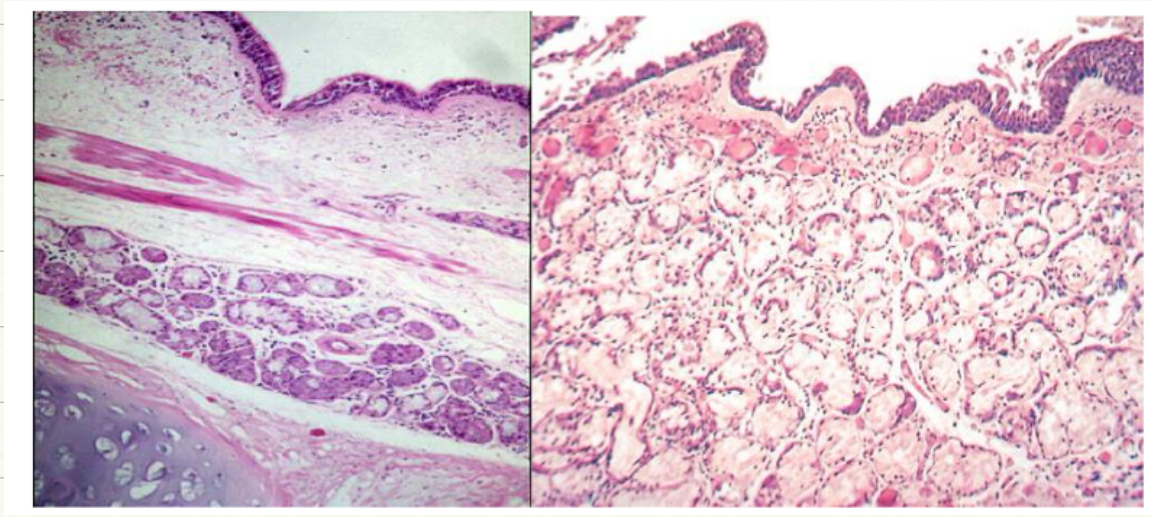
→ The color deep pink!



destruction of alveolar walls without fibrosis, leading to enlarged air spaces, alveolar capillaries is diminished.

▷ With the loss of elastic tissue in the alveolar septa, radial traction on the small airways is reduced → collapse during expiration → chronic airflow obstruction functional

Chronic bronchitis:



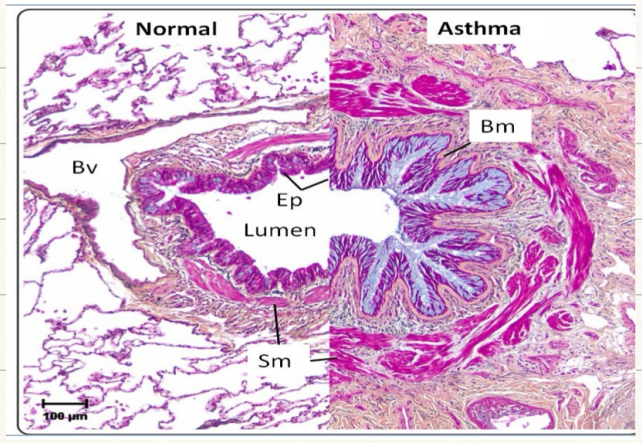
Enlargement of the mucus secreting glands in trachea and larger bronchi.

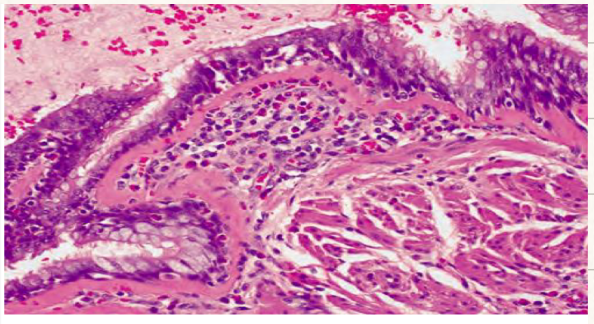
▷ assessed by the ratio of the thickness of the submucosal gland layer to that of the bronchial wall (the Reid index—normally 0.4).

	Predominant Bronchitis	Predominant Emphysema
Age (yr)	40-45	50-75
Dyspnea	Mild; late	Severe; early
Cough	Early; copious sputum	Late; scanty sputum
Infections	Common	Occasional
Respiratory insufficiency	Repeated	Terminal
Cor pulmonale	Common	Rare; terminal
Airway resistance	Increased	Normal or slightly increased
Elastic recoil	Normal	Low
Chest radiograph	Prominent vessels; large heart	Hyperinflation; small heart
Appearance	Blue bloater	Pink puffer

L4

Asthma:-

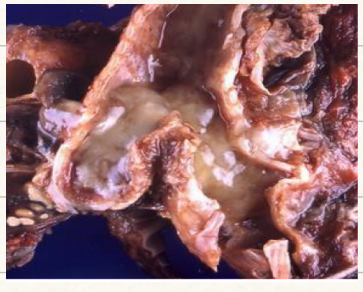


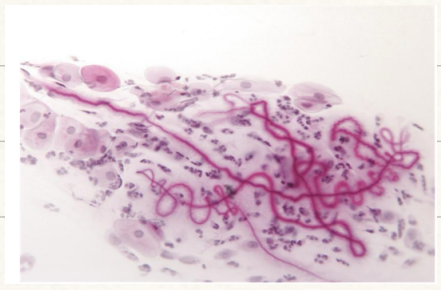


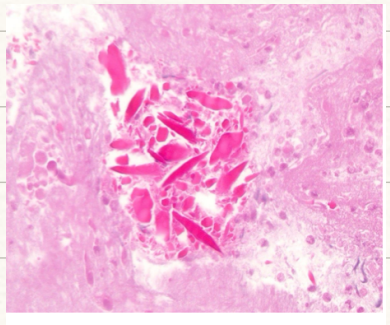
Airway remodeling:

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

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







tenacious mucous plugs

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

Curschmann Spirals

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

Charcot-Leyden crystals

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

Bronchiectasis:-

Two intertwined processes contribute to bronchiectasis: obstruction and chronic infection. Either may be the initiator:

▷ E.g., obstruction by a foreign body 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

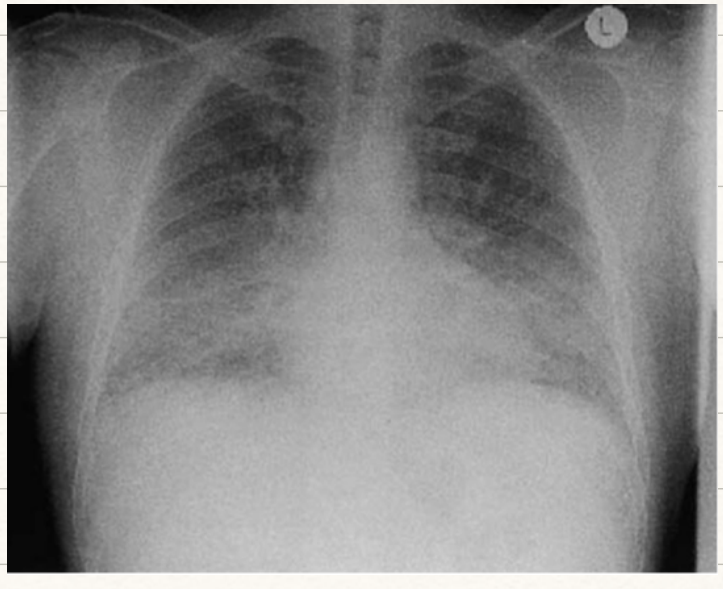
Usually affects lower lobes, particularly those that are most vertical (gravitational). The airways dilated up to four times their usual diameter → seen on gross examination almost out to the pleural surface (normally cannot be followed by eye beyond a point 2 to 3 cm from the pleura)

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 & **reversible** obstruction.	**Irreversible** 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	**Reversible** (intermittent).	**Irreversible.**
Complications	Status asthmaticus (fatal).	Brain abscess, amyloidosis, cor pulmonale.

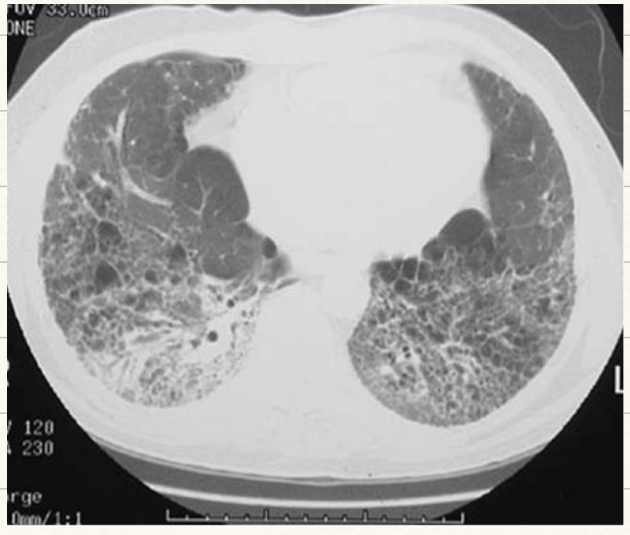
ممكن يساعد في حل الأسئلة النظرية بالأول (للمراجعة)

L5

CHRONIC INTERSTITIAL (RESTRICTIVE, INFILTRATIVE) LUNG DISEASES:

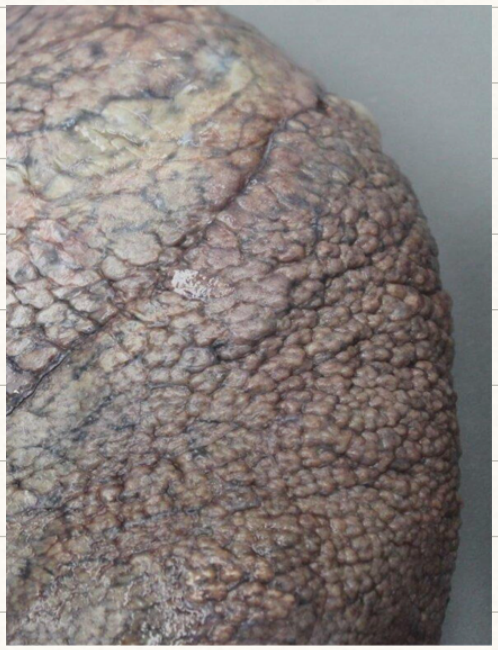


Chest radiographs: small nodules, irregular lines, or "ground-glass shadows.

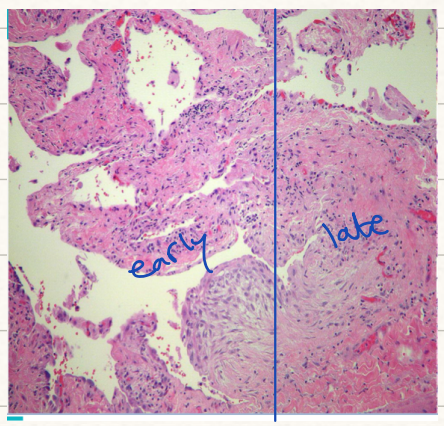


honeycomb

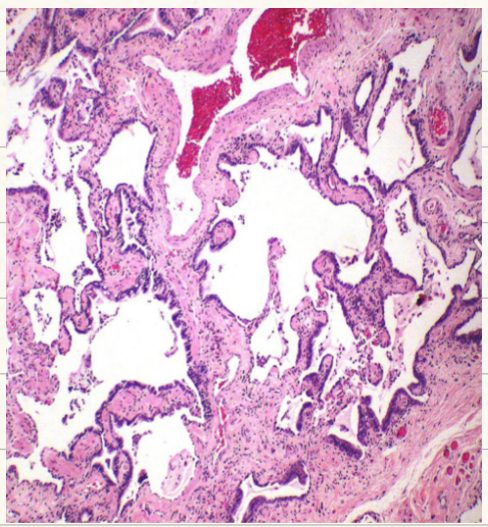
IPF:-



, the pleural surfaces of the lung are cobblestoned due to retraction of scars along the interlobular septa. The cut surface shows firm, rubbery, white areas of fibrosi

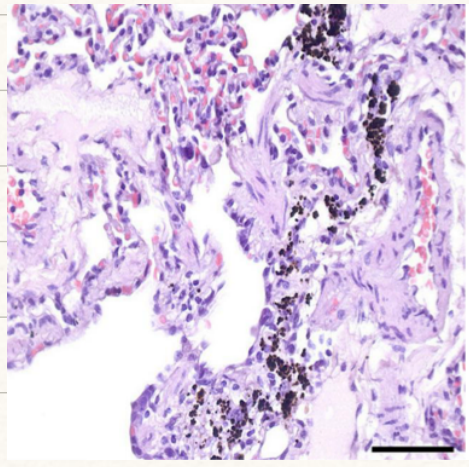


typical finding:
coexistence of both early & late lesions.

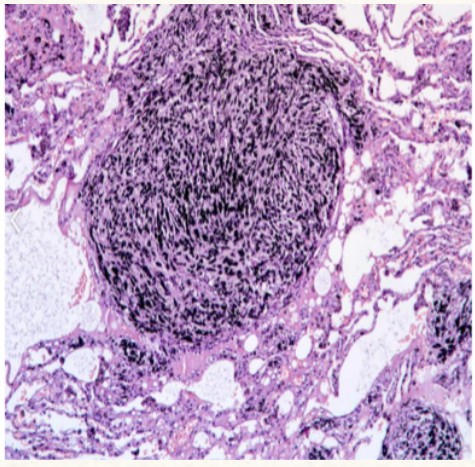


▷ Dense fibrosis causes collapse of alveolar walls and formation of cystic spaces (honeycomb fibrosis) lined by hyperplastic type II pneumocytes or bronchiolar epithelium. ▷ The interstitial inflammation consists of alveolar septal infiltrates of lymphocytes and occasional plasma cells, mast cells, and eosinophils.

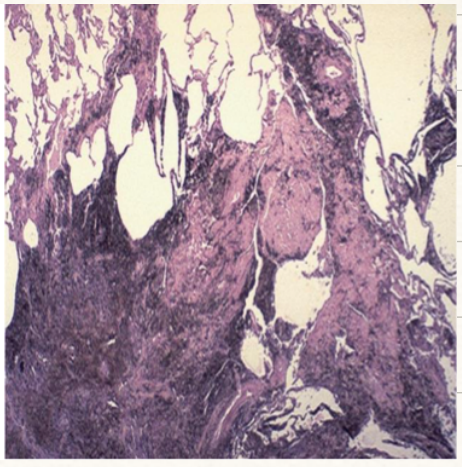
Coal Workers' Pneumoconiosis



Pulmonary anthracosis: Inhaled carbon pigment is engulfed by alveolar
or interstitial macrophages → accumulate in the connective tissue
along the pulmonary & pleural lymphatics & in draining lymph nodes.

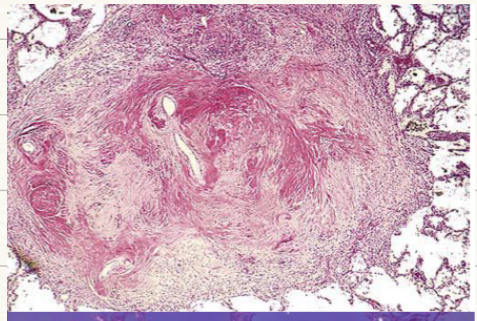


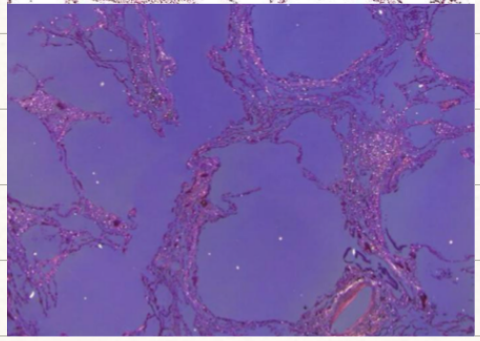
Simple CWP: coal macules & larger coal nodules.
The coal macule consists of dust-laden macrophages & small amounts of collagen fibers arrayed in a delicate network.
(upper lobes and upper
zones of the lower lobes are more heavily involved).



Complicated CWP (PMF): coalescence of coal nodules & generally develops over many years.
Multiple, dark black scars larger than 2 cm consist of dense collagen and pigment.

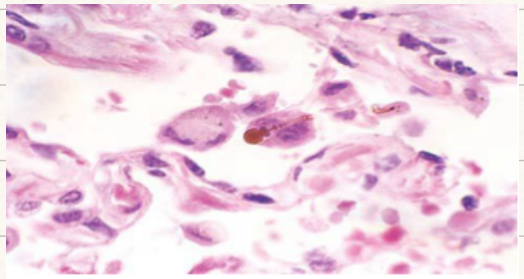
Silicosis

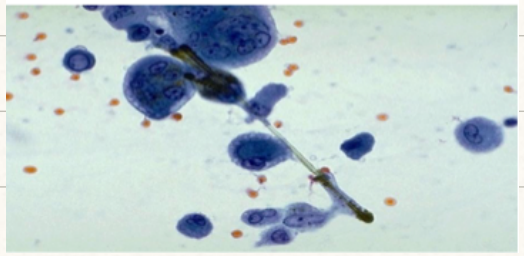




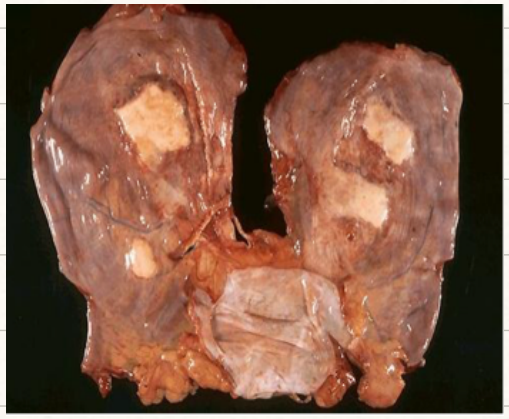
▷ Silicotic nodules in early stages are tiny, barely palpable, discrete, pale-to-black (if coal dust is present) nodules in the upper zones of the lungs.
▷ Microscopically: Silicotic nodule demonstrates concentrically arranged hyalinized collagen fibers surrounding an amorphous center.
▷ The "whorled" appearance of collagen fibers is quite distinctive for silicosis.
▷ Nodules on polarized microscopy: weakly birefringent silica particles. .

Asbestosis

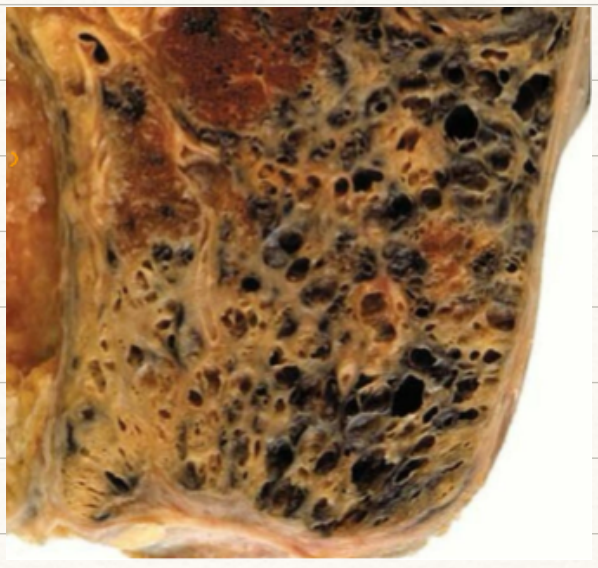




Marked by diffuse pulmonary interstitial fibrosis, characterized by the presence of asbestos bodies→ golden brown, fusiform or beaded rods with a translucent center.
▷ Asbestos bodies formed when macrophages attempt to phagocytose asbestos fibers; the iron "crust" is derived from phagocyte ferritin.

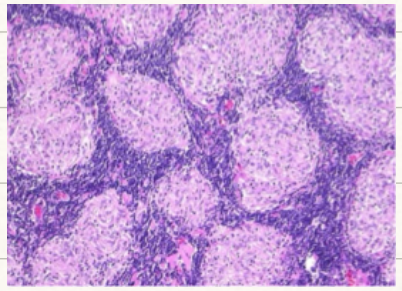


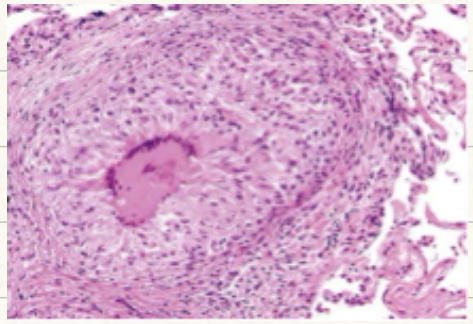
Pleural plaques: the most common manifestation of asbestos exposure.
▷ A well-circumscribed plaques of dense collagen often containing calcium.
▷ At the anterior & posterolateral aspects of the parietal pleura & over the domes of the diaphragm.



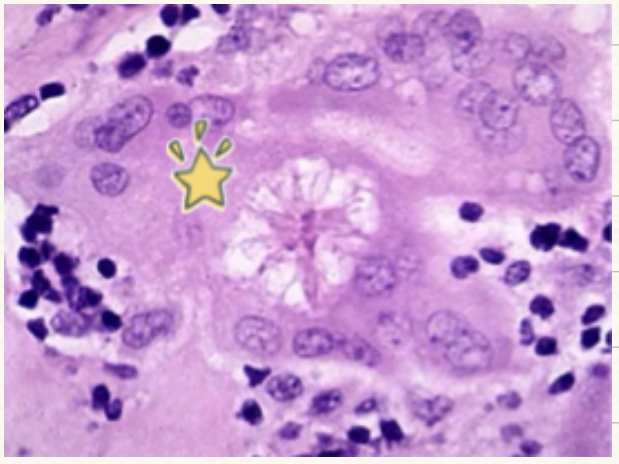
Asbestosis begins in the lower lobes and subpleurally, spreading to the middle and upper lobes of the lungs as the fibrosis progresses.
▷ Contraction of the fibrous tissue distorts the normal architecture, creating enlarged air spaces enclosed within thick fibrous walls; eventually, the affected regions become honeycombed

Sarcoidosis

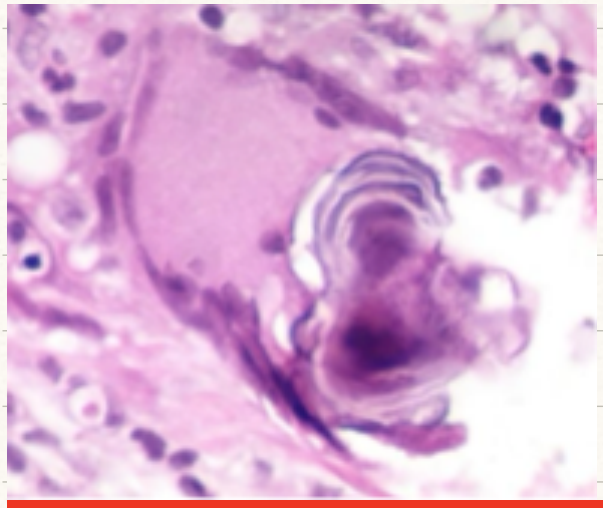




▷ The cardinal histopathologic feature of sarcoidosis is the nonnecrotizing epithelioid granuloma.
▷ This is a discrete, compact collection of epithelioid macrophages rimmed by an outer zone rich in CD4+ T cells.
▷ It is not uncommon to see intermixed multinucleate giant cells formed by fusion of macrophages.

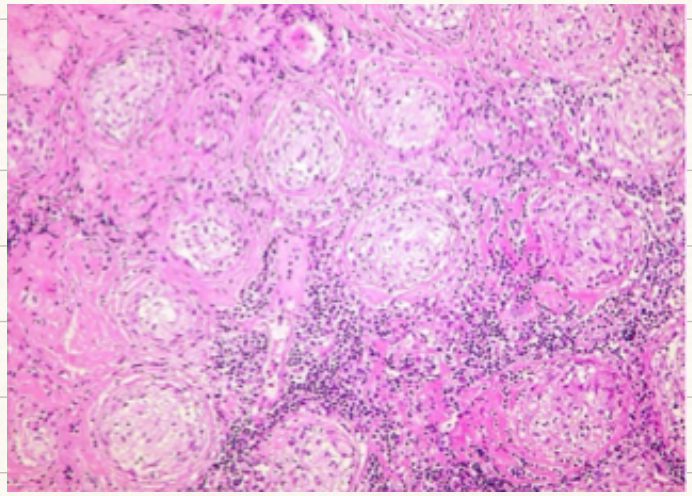


Asteroid bodies, stellate inclusions enclosed within giant cells.

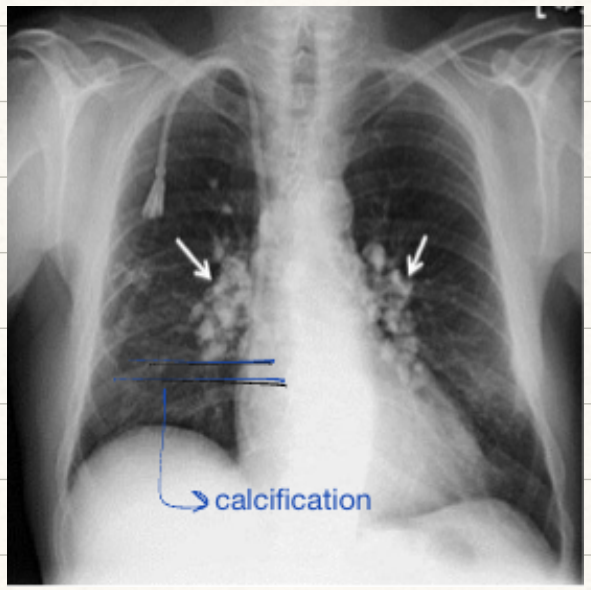


Schaumann bodies, laminated concretions composed of calcium & proteins.

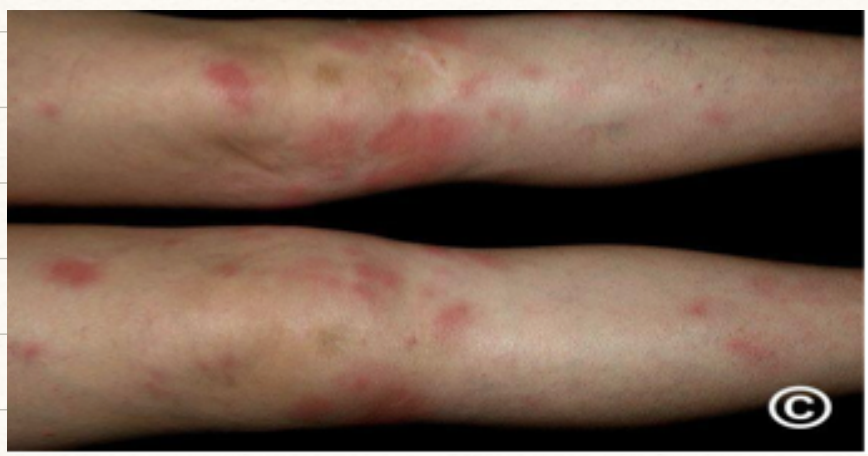
not required for diagnosis of sarcoidosis, & may be found in granulomas seen in other disorders



Early on, a thin layer of laminated fibroblasts is found peripheral to the granuloma; over time, these proliferate and lay down collagen that replaces the entire granuloma with a hyalinized scar.



Intrathoracic hilar and paratracheal lymph nodes are enlarged in 75-90% of patients; nodes are painless and have a firm, rubbery texture. Unlike in tuberculosis, lymph nodes in sarcoidosis are "nonmatted" (nonadherent) & do not undergo necrosis.



Skin lesions are encountered in approximately 25% of patients: Erythema nodosum, a hallmark of acute sarcoidosis, presents as bilateral raised, red, tender nodules on the anterior aspects of the legs. It is a form of panniculitis.