Restrictive Lung Diseases

OBJECTIVES:

- ✓ Understand the structure and constituents of the lung interstitium as well as the restrictive changes which occur in diseases of the interstitium (ILD).
- ✓ Know the symptoms of ILD : progressive breathlessness and cough .
- ✓ Know subtypes of ILD : acute and chronic .
- ✓ Discuss the causes, morphology and outcome of acute ILD .
- ✓ Appreciate the pathogenesis of chronic ILD regardless of their type.
- ✓ Become aware of the classification of interstitial lung diseases.
- ✓ Discuss examples of interstitial lung diseases including :
  - Idiopathic pulmonary fibrosis
  - Pneumoconiosis
  - Hypersensitivity Pneumonitis
  - Sarcoidosis

Editing File

Black: original content.
Red: Important.
Green: AlRikabi's Notes.
Grey: Explanation.
Blue: Only found in boys slides.
Pink: Only found in girls slides.

Restrictive Lung Disease :

Definition

- Group of diseases (of various etiologies) characterized by reduced expansion of lung parenchyma and decreased total lung capacity (decreased lung volume and compliance).
- Spirometry: FEV1 and FVC decreased (ratio is normal)
- Symptoms: chronic dry cough & dyspnea

Intrinsic lung diseases

- **Also called :** diseases of the lung parenchyma or Primary ILDs (Interstitial Lung Diseases).
- It causes inflammation or scarring of the lung tissue or result in filling of the air spaces with exudate and debris (pneumonitis).
- **They are characterized by :**
  - Inflammatory infiltrates in the alveolar interstitial space.
  - The interstitium becomes thickened and fibrotic (Due to increased release of elastic fibers and collagen from fibroblasts) which will lead to (Stiff lung*) and results in decreased oxygen-diffusing capacity.
  - They could be acute or chronic.

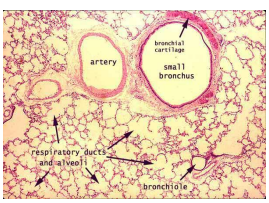
Extrinsic disorders

- **Also called :** Extraparenchymal diseases.
- They are related to the components of the respiratory pump (not the lung tissue itself):
  - Chest wall.
  - Pleura
  - Respiratory muscles. Ex:Guillain-Barré syndrome (weakens intercostal muscles).
- **Abnormalities of the chest wall include :**
  - bony abnormalities (kyphosis or kypho-scoliosis).*
  - massive pleural effusion .
  - morbid obesity
  - neuromuscular disease of respiratory muscles .

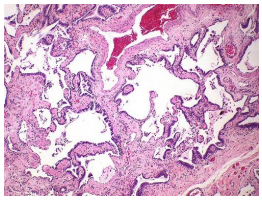
Honeycomb lung:

Definition: It's the condition when Both alveoli and bronchioles coalescence (يلتحمون) to form cysts lined with cuboidal or columnar epithelium and separated by inflammatory fibrous tissue .

Note : The final stage of all CHRONIC restrictive lung disease is extensive fibrosis with **honeycomb lung**.

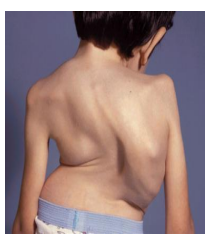


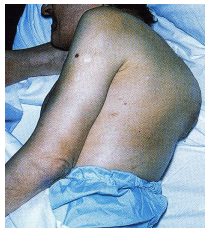
Normal lung


Honeycomb lung

Kyphoscoliosis :

Flexion of the spine → kyphosis
lateral deviation of the spine → scoliosis
Note: both reduce chest volume → compromises respiratory function and may cause restrictive lung disease.




Severe kyphoscoliosis of unknown etiology.

One of the complications of restrictive lung diseases, is cor pulmonale which happens due to pulmonary hypertension "the pressure is higher than 25 mmHg in the pulmonary artery"

Acute restrictive lung diseases (INTRINSIC TYPE)

1- Acute Respiratory Distress Syndrome (ARDS)

Definition

Respiratory failure occurring within one week of a known clinical insult with bilateral opacities on chest imaging.

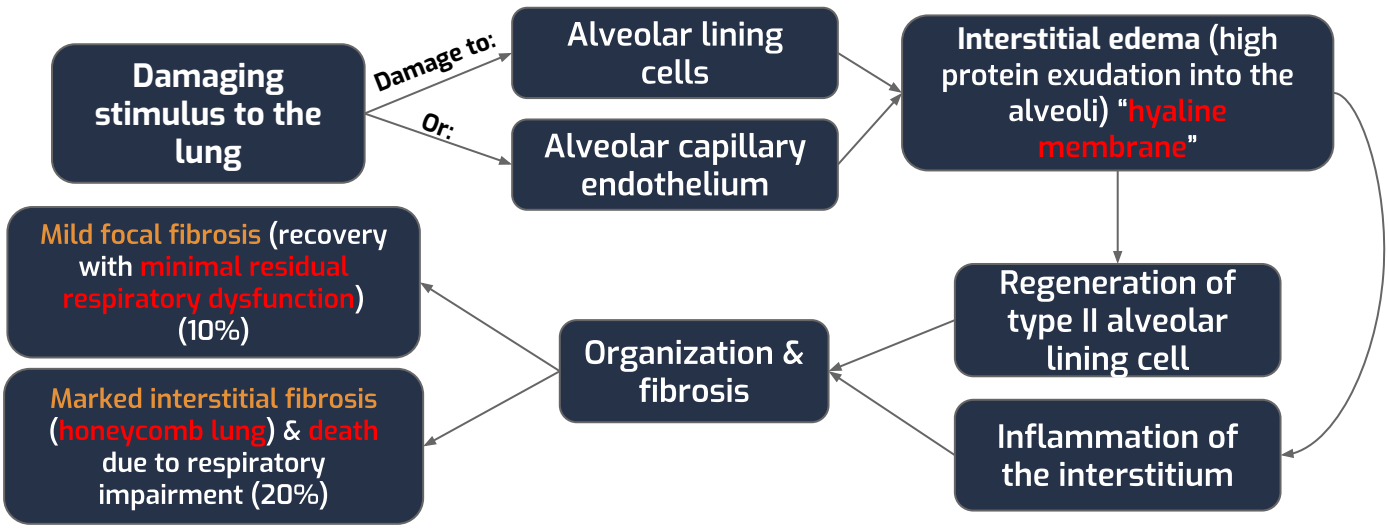
Etiology

Direct injury to lung	Indirect injury to lung
- **Pneumonia** . - Aspiration of gastric contents . - Pulmonary trauma, Fat embolism . - Post lung transplant, Near drowning . - Toxic inhalation injury (irritants such as chlorine,O2 toxicity). - **Severe acute respiratory syndrome (SARS)**: The virus is a coronavirus that destroys the **type II** pneumocytes and causes diffuse alveolar damage.	- **Sepsis**, Shock, Transfusion, Uremia. - Severe trauma (e.g. bone fractures, head injury, burns, radiation) - Cardiopulmonary bypass, Acute pancreatitis. - Overdose with street drugs such as heroin . - Therapeutic drugs such as bleomycin . - Hematologic conditions e.g multiple transfusion, coagulation disorders.

Pathogenesis and outcomes:

In ARDs, the integrity of the alveolar-capillary membrane is compromised by endothelial and epithelial injury.

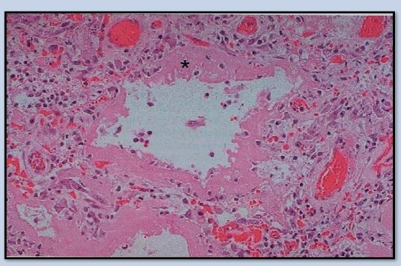
Leads to: Death in acute phase (70%)



Histology

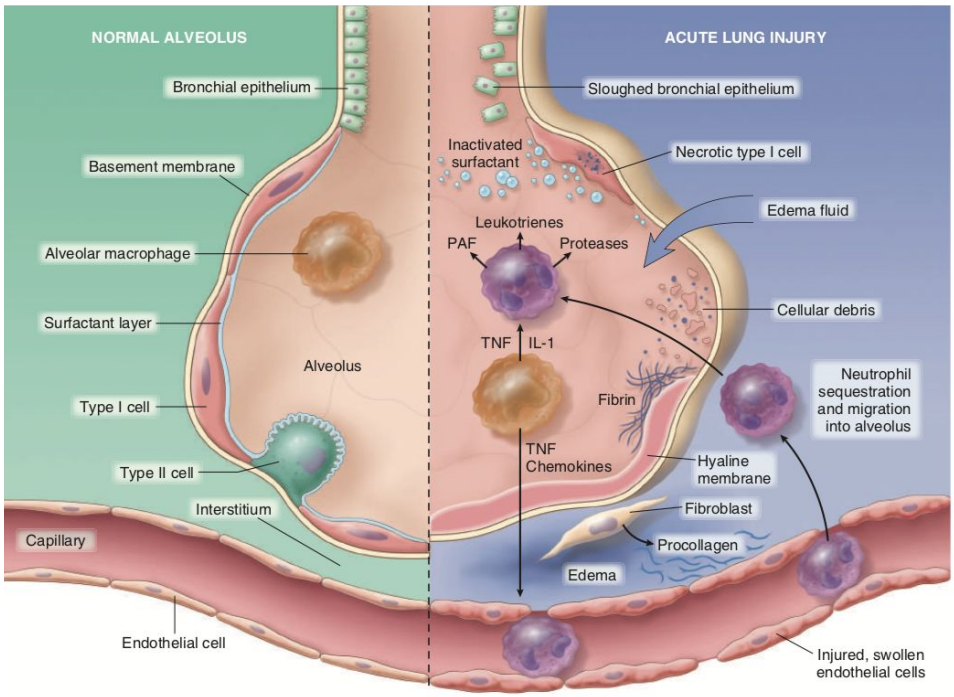
Diffuse Alveolar Damage(DAD):

- Fine granularity
- Lung edema
- Atypical pneumonia



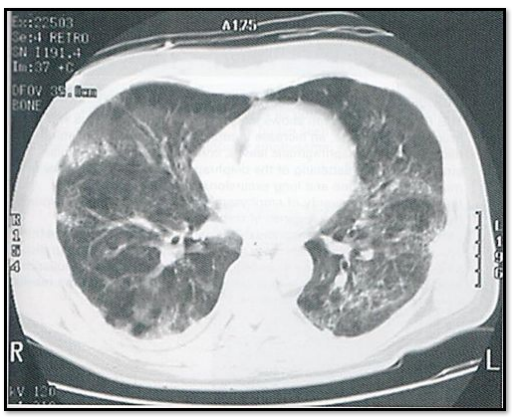
Acute restrictive lung diseases (INTRINSIC TYPE)

1- Acute Respiratory Distress Syndrome (ARDS)



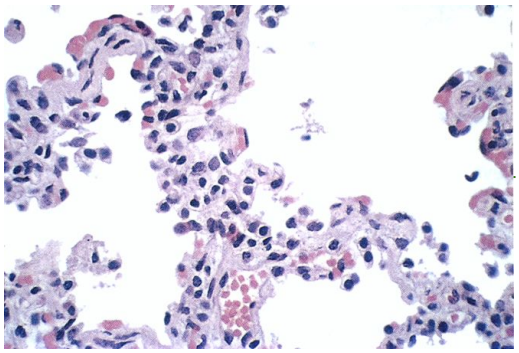
(from robbins)
Cytokines released during acute lung injury:
IL-8
IL1
TNF
Potent for neutrophil chemotaxis and activation
Neutrophils release:
Leukotrienes
PAF
Proteases

Macrophages-derived factors that stimulate the growth of fibroblasts and collagen deposition in healing process:
TGF-Beta
PGDF



Diffuse alveolar damage, CT image

CT scan→White lung syndrome Fibrin and debris→form a hyaline membrane around the alveoli



Atypical pneumonia
- Could lead to interstitial pneumonitis
- Usually caused by Influenza virus
- Edema in the interstitium and chronic inflammatory infiltration
-Inflammatory cells are lymphocytes (viral infection), and not neutrophils

Acute restrictive lung diseases (INTRINSIC TYPE)

2- Neonatal Respiratory Distress syndromes (NRDS)

Definition

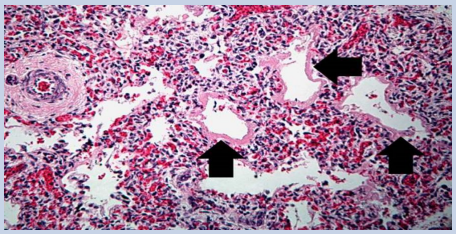
Also called : Hyaline membrane disease.(Due to the formation of 'membranes' in the peripheral air spaces observed in infants who succumb to this condition.)

· It is the most common cause of respiratory distress in the newborn and is the most common cause of death in premature infants.

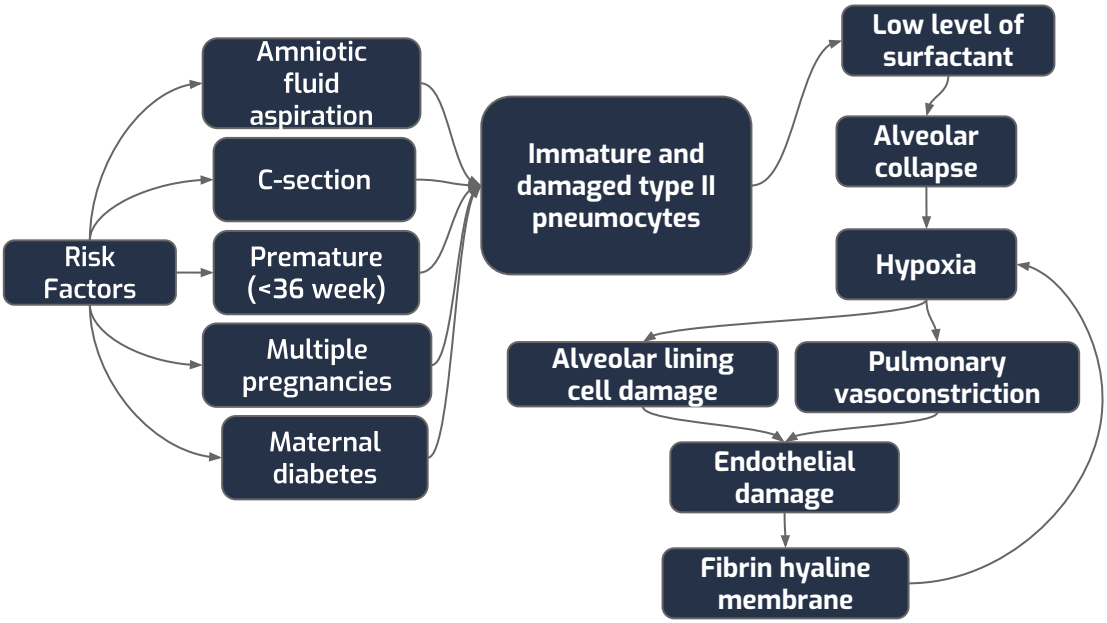
· There are also strong thought not invariable associations with male gender, maternal diabetes, and delivery by cesarean section.

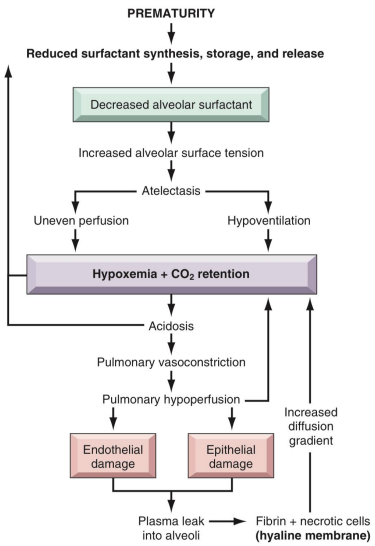
Etiology

- **Inability of the immature lung to synthesize sufficient surfactant.***
- It is the same as **ARDS** except that it is caused by a deficiency of pulmonary surfactants in newborns, most often as a result of immaturity.



Pathogenesis and outcomes





*Surfactant is a complex of surface-active phospholipids, principally dipalmitoylphosphatidylcholine (lecithin) and at least two groups of surfactant-associated proteins. The importance of surfactant-associated proteins in normal lung function can be gauged by the occurrence of severe respiratory failure in neonates with congenital deficiency of surfactant caused by mutations in the corresponding genes. Surfactant is synthesized by type II pneumocytes

Chronic restrictive lung disease (INTRINSIC TYPE)

Definition

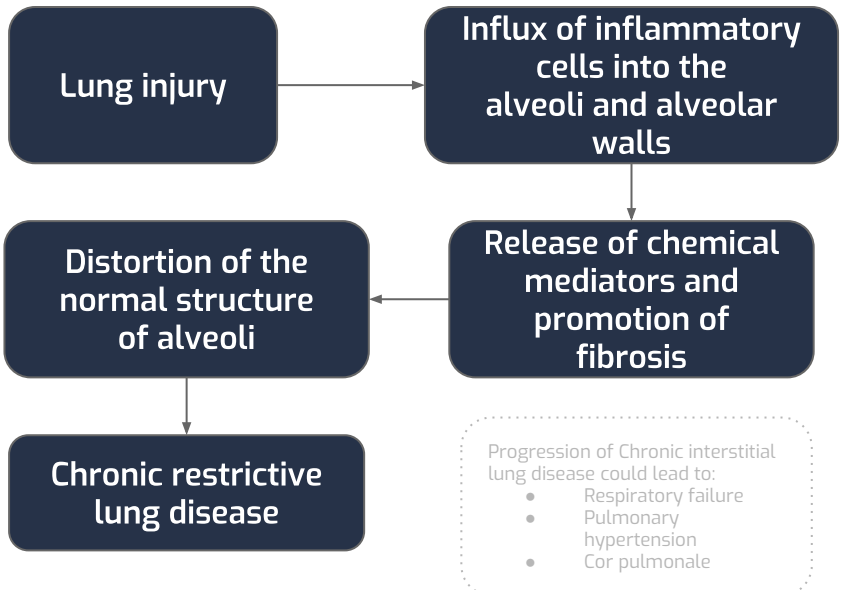
Heterogenous group of disorders characterized by bilateral, often patchy pulmonary fibrosis mainly affecting the walls of the alveoli.

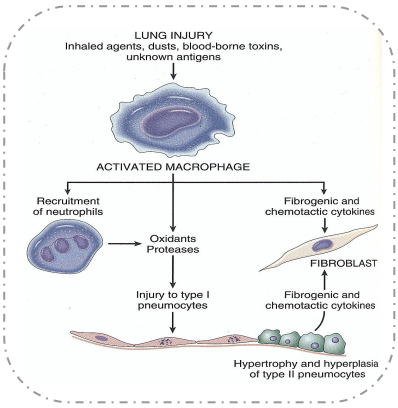
- **Major categories of chronic interstitial lung diseases :**

They are categorized based on clinicopathologic features and characteristic histology

Fibrosing	Granulomatous	Eosinophilic	Smoking-Related
▪ Usual interstitial pneumonia (idiopathic pulmonary fibrosis). ▪ Nonspecific interstitial pneumonia. ▪ Cryptogenic organizing pneumonia. ▪ Collagen vascular disease-associated. ▪ Pneumoconiosis. ▪ Therapy-associated (drugs, radiation).	▪ Sarcoidosis. ▪ Hypersensitivity pneumonia.	▪ Loeffler syndrome. ▪ Drug allergy–related. ▪ Idiopathic chronic eosinophilic pneumonia.	▪ Desquamative interstitial pneumonia. ▪ Respiratory bronchiolitis.

- **Pathogenesis of chronic interstitial lung diseases :**





Idiopathic pulmonary fibrosis (Usual interstitial pneumonia)

Idiopathic pulmonary fibrosis

Epidemiology

- **Unknown** . (Idiopathic means : unknown cause)
- MUCB4-rikabi (MUC5B-robbins) gene mutation (chromosome 9)→higher tendency to develop fibrosis
- The resulting injury to alveolar epithelial cells set in motion event that lead to increase local production of fibrogenic cytokines such as TGF-β
- Most patients are 55-75 years.

Pathogenesis

- The injured epithelial cells are the source of profibrogenic factors such as **TGF-βI** secondary to down regulation of caveolin

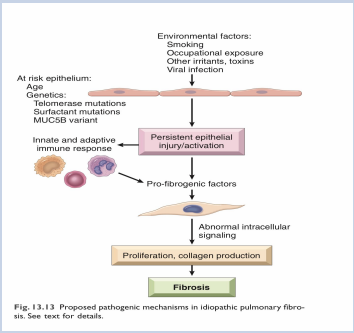


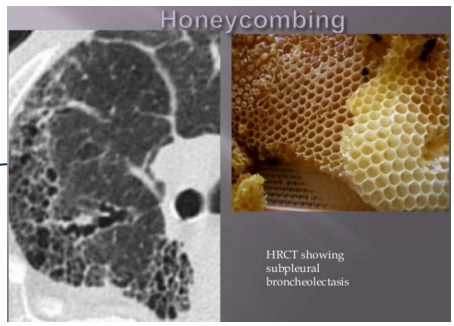
Fig. 13.13 Proposed pathogenic mechanisms in idiopathic pulmonary fibrosis. See text for details.

Prognosis

- **Poor**, the median survival is about 3 years

Characteristics:

1. Reduced lung compliance
2. Subpleural patchy, bilateral interstitial fibrosis
3. Fibroblastic foci
4. Formation of cystic space (honeycomb lung)
5. Affecting the interstitium of the lung/alveolar wall
6. Does not affect the air spaces themselves, but the tissues around them
7. Temporal heterogeneity fibrotic distribution



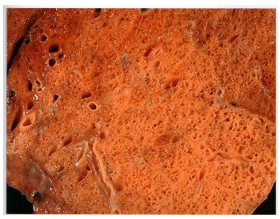
Idiopathic pulmonary fibrosis (Usual interstitial pneumonia)

Clinical features

- Gradually increasing dyspnea on exertion
- Dry cough
- On physical examination, most patients have characteristic “dry” or “Velcrolike” crackles during inspiration.
- Associated with usual interstitial pneumonia
- X ray:
  Early: ground glass fine granularity.
  Advanced: honeycomb lung.

Morphology

Gross:



Honeycomb change, Fibrosis in the subpleural region

Microscopic:

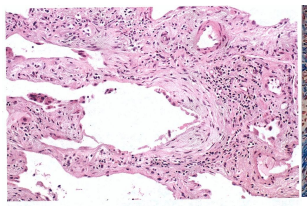
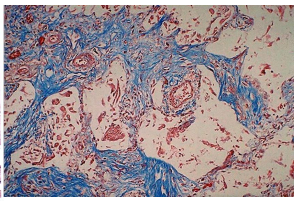


Figure 13–16
Usual interstitial pneumonia. Fibroblastic focus with fibers running parallel to surface and bluish myxoid extracellular matrix.



Interstitial fibrosis

Complications

- Hypoxia, peripheral edema
- Cyanosis, cor-pulmonale
- Clubbing
- Gradual deterioration in pulmonary status despite medical treatment

Treatment:
To delay progression of the disease we may use:
-Pirfenidone (TGF-b1 antagonists)
-Nintedanib (tyrosine kinase antagonist)

Note: The only definitive treatment is **lung transplantation.**

Pneumoconiosis

Definition

lung disorders caused by inhalation of mineral dusts leading to lung damage.

Etiology

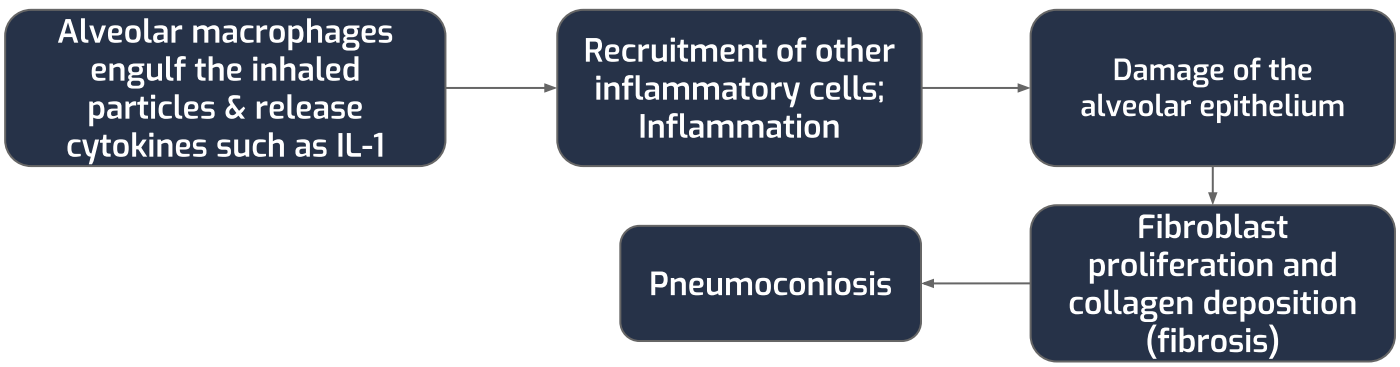
The most common mineral dusts are: coal, silica, asbestos, beryllium.

The development of pneumoconiosis is dependent on:

1. The **amount of dust** retained in the lung airways
   a. Concentration of the dust in the ambient air.
   b. Duration of the exposure. (more than 10 years **Long period**)
   c. Effectiveness of the clearance mechanisms.
2. The particles' **size (1-5) um** shape. (Particles > 1 um will be stuck in the air so it will enter & exit without causing problems)
3. Their **solubility** and **physiochemical activity.**
4. The possible additional effects of **other irritants**, **tobacco smoking**

Pathogenesis:

The pulmonary alveolar macrophage is a key cellular element in the initiation and perpetuation of inflammation, lung injury and fibrosis.



Mineral Dust–Induced Lung Disease

Agent	Disease	Exposure
Coal dust	Simple coal worker's pneumoconiosis: macules and nodules Complicated coal worker's pneumoconiosis: PMF	Coal mining
Silica	Silicosis	Sandblasting, quarrying, mining, stone cutting, foundry work, ceramics
Asbestos	Asbestosis, pleural effusions, pleural plaques, or diffuse fibrosis; mesothelioma; carcinoma of the lung and larynx	Mining, milling, and fabrication of ores and materials; installation and removal of insulation

PMF, Progressive massive fibrosis.

Pneumoconiosis

1- Coal worker's pneumoconiosis (CWP)

Definition

Accumulation of coal dust in the lungs & the tissue's reaction to its presence.

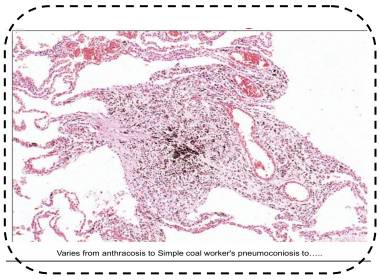
CWP categories

Anthracosis

- Asymptomatic.
- Commonly seen in urban dwellers and tobacco smokers.
- Caused by accumulation of carbon in the lungs.

Simple CWP

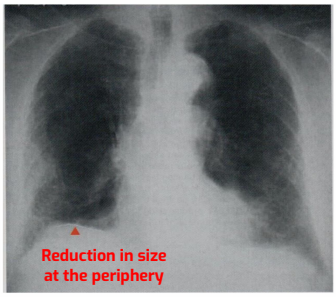
- Black macules 1-5 mm are scattered through the lung.



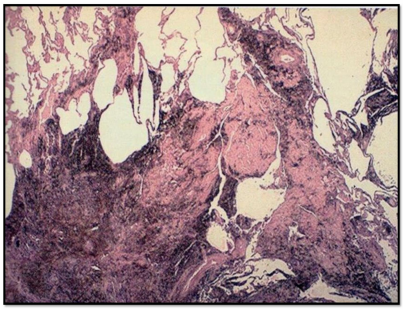
Varies from anthracosis to Simple coal worker's pneumoconiosis to.....

Complicated CWP

- Also called, progressive massive fibrosis (PMF).
- Extensive fibrosis & compromised lung function.
- Characterized by multiple, dark black scars exceed 2-10 cm.
- Produces cough, dyspnea, and lung function impairment.
- Complication: cor pulmonale.



Pneumoconiosis, radiograph



Coal worker's pneumoconiosis, microscopic

to progressive massive fibrosis (complicated coal worker's pneumoconiosis).
(a) Cut surface (b) thin section of whole lung.

Pneumoconiosis

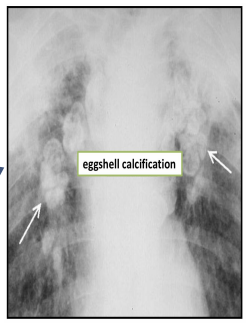
2- Silicosis

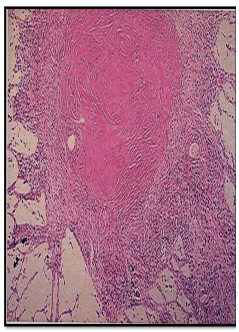
Definition

Fibro-nodular lung disease caused by long term exposure to inhalation of crystalline silica particles (**alpha-quartz or silicon dioxide**).

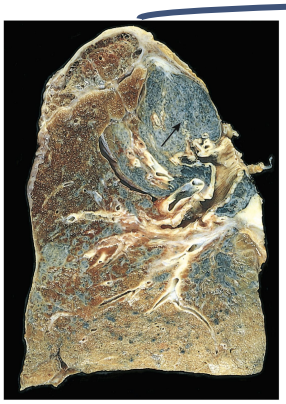
Characteristics:

1. Industrial exposure: mining of gold, tin, copper and coal, sandblasting, metal grinding, ceramic manufacturing.
2. Stony-hard large fibrous scars
3. Eggshell calcification
4. Fibrous pleural plaques may develop
5. Predispose to lung cancer & tuberculosis[1] for unknown reasons

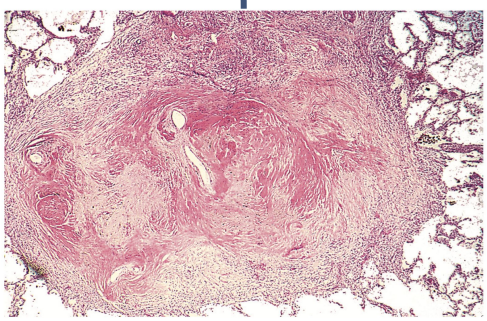




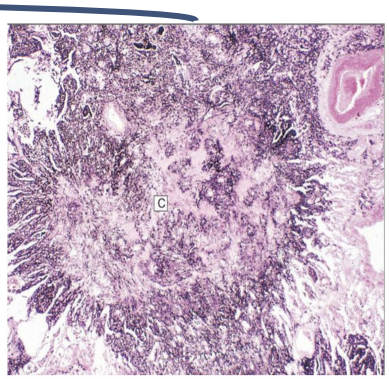
Morphology



Scarring has contracted the upper lobe into a small dark mass (arrow). Note the dense pleural thickening



concentrically arranged hyalinized collagen fibers surrounding an amorphous center. The "whorled" appearance of the collagen fibers is quite distinctive for silicosis (HOW? silica in sand contains quartz, which is fibrogenic)



Silicosis of lung. In silicosis, nodules of collagen (C) contain silica particles.

1: silicosis depresses cell-mediated immunity, and crystalline silica may inhibit the ability of pulmonary macrophages to kill phagocytosed mycobacteria.

Pneumoconiosis

3- Asbestosis

Definition

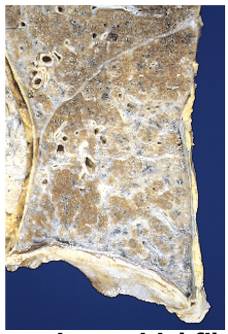
occupational exposure to asbestos is linked to parenchymal interstitial fibrosis.

Etiology

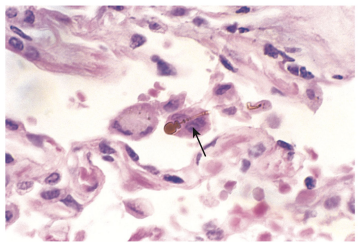
Characterized by the presence of asbestos bodies (Ex:Ship-building industry), which are seen as golden brown, fusiform or beaded rods with a translucent center. Apparently they are formed when macrophages attempt to phagocytose asbestos fibers; the iron "crust" is derived from phagocyte ferritin.

Complications

- localized fibrous plaques[1], or, rarely, diffuse fibrosis in the pleura
- Pleural effusion
- Pleural adhesions
- Lung carcinoma (Bronchogenic carcinoma)
- malignant pleural and peritoneal mesothelioma



severe interstitial fibrosis diffusely affecting the lower lobe of the lung.



Asbestos bodies

Asbestos fibers causes bleeding > forms hemosiderin (prussian blue stain shows ferruginous bodies)

The risk for developing lung carcinoma is increased about 5-fold for asbestos workers; the relative risk for mesothelioma[2], is more than 1000 times greater. Concomitant cigarette smoking greatly increases the risk for lung carcinoma but not for mesothelioma.

1: Are well-circumscribed plaques of dense collagen often containing calcium.
2: A malignant tumor affecting the mesothelial cells in the pleura.

Granulomatous diseases

1- Sarcoidosis

Definition

Immunological multisystem disease of unknown etiology (thought to be autoimmune) characterized by noncaseating granulomatous inflammation in many tissues and organs.

Characteristics

Epidemiology

- Affecting all races & both sexes equally

Sites

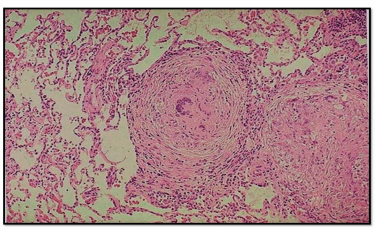
- Lungs
- Predominantly, Intrathoracic hilar and paratracheal lymph nodes
- Skin
- Eyes

Symptoms include:
1- Uveitis
2- arthritis
3- dryness of mouth
4- lack of lacrimation

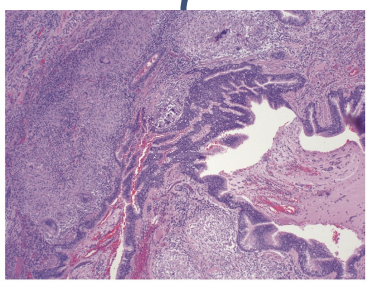
Prognosis

- Unpredictable, It can be progressive & chronic.
- It may present as episodes of activity.
- Majority of the patients respond well to treatment.

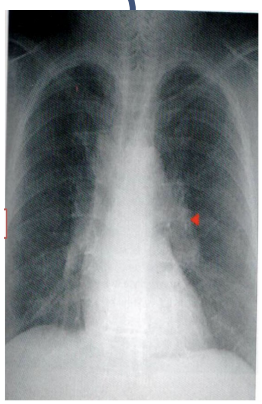
Morphology



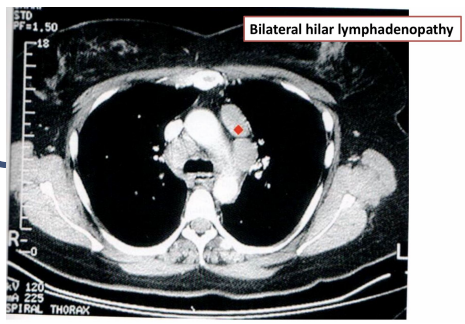
Sarcoidosis, microscopic
noncaseating interstitial granulomas



Non-Necrotizing interstitial granuloma



Bilateral hilar lymphadenopathy



-Sarcoidosis is often confused with TB (the distinction is that there is no caseation.)

Granulomatous diseases

2- Hypersensitivity Pneumonitis

Definition

Immunologically mediated disorder affecting airways (Alveoli) and interstitium

Associated with

- Heightened sensitivity to inhaled antigens

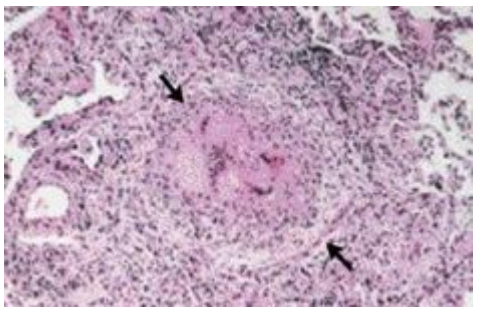
Disease	Antigen / Source of antigen
Farmer's Lungs	• Micropolyspora faeni in hay • Thermophilic actinomycetes • microsporum→ extrinsic allergic alveolitis
Pigeon breeder's (*psittacosis*)	• Birds • pigeons
Air-cooler lung	• Thermophilic bacteria • Source of antigen: Desert cooler
Bagassosis	• Sugarcane bagasse

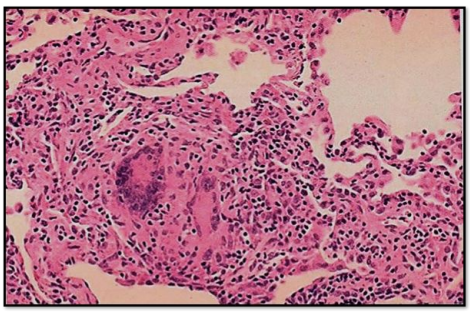
Granulomatous diseases

2- Hypersensitivity Pneumonitis (cont.)

Morphology

- Non-caseating Granuloma
  (Poorly defined granulomas)
- Chronic inflammation
- Cells:
  CD4+
  CD8+
  Plasma cells
  Macrophages





Clinical Features

- Fever
- Cough
- Dyspnea
- Decrease in diffusion capacity
- Decrease in lung compliance
- Decrease in total lung volume

Summary

Pneumoconiosis

	Exposure	Pathological features
Coal worker's pneumoconiosis	Coal Mining	Anthracosis: **accumulation of carbon**. Simple CWP: **black macules** Complicated CWP: **multiple, dark black scars**
Silicosis	Sandblasting quarrying, mining, stone cutting, foundry work, ceramics	• Eggshell calcification • Predispose to lung cancer & tuberculosis
Asbestosis	Mining, milling, and fabrication of ores and materials; installation and removal of insulation	• Asbestosis bodies • Localized fibrous plaques • Predispose to bronchogenic carcinoma & malignant mesothelioma

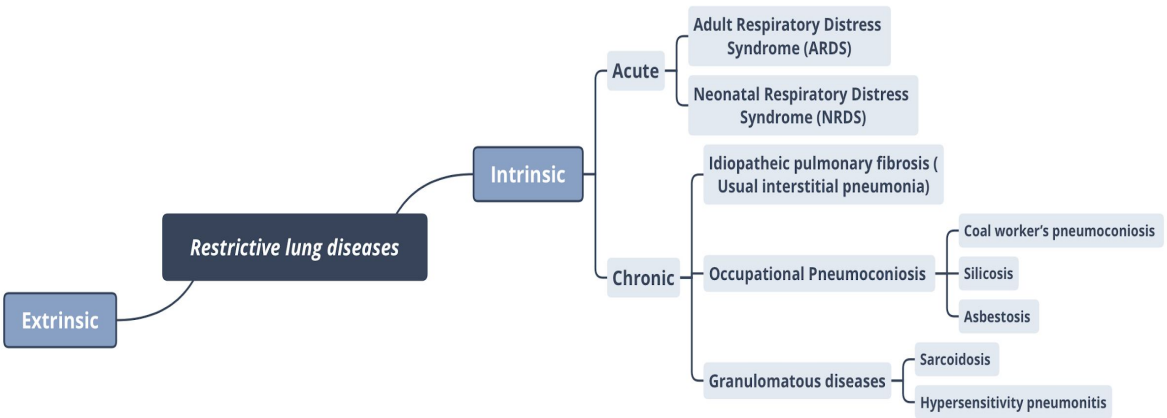
Granulomatous diseases

Sarcoidosis

- immunological multisystem disease, unknown etiology, > female
- noncaseating granulomas in various tissues:-
  1- lymph node enlargement (almost all cases),
  2- eye involvement [dry eyes],
  3- iritis,
  4- skin lesions (erythema nodosum, painless sub-cutaneous nodules), and viscera (liver, skin, marrow).
  5- Lung involvement (90% of cases), with formation of granulomas and interstitial fibrosis

Hypersensitivity pneumonitis

- Immunologically mediated disorder affecting airways (Alveoli) and interstitium.
- Associated with hypersensitivity to various antigens



Dr. AlRikabi's notes

Definition: Group of diseases (of various etiologies) characterized by decreased lung volume and compliance.
Spirometry: FEV1 and FVC decreased (ratio is normal)

Symptoms:
-Chronic dry cough
-Dyspnea (varying in severity)

Complication:
Cor pulmonale (Pulmonary hypertension→25+mmHg in the pulmonary artery)

Diagnostics:

- Radiology (x-ray, CT)
- Spirometry
- Cytology (sputum, bronchial brushing/washing, bronchoalveolar lavage)
- Biopsy: Endobronchial, transbronchial, open lung biopsy, VAT (video assisted thoracoscopic)

Etiologies:

1- Thoracic cage deformity:
- decreased lung expansion
-Severe kyphoscoliosis
-Guillain-Barré syndrome ; weakens intercostal muscles

2- Idiopathic pulmonary fibrosis:
-Familial
- Affecting the interstitium of the lung/alveolar wall
- Do not affect the air spaces themselves, but the tissues around them
-Honeycombed lung due to entrapped air (anthracosis→low po2 high pco2)
-Affects lower part of the lung
-Bilateral peripheral reticulation—>fibrosis shrinks the lung (trapped air→dilated alveoli)
-Temporal heterogeneity fibrotic distribution
- Histological: Blue stain indicating prevalent connective tissue (Masson's trichrome stain)
-Associated with usual interstitial pneumonia

Pathogenesis:
- Injury affecting macrophages leading to cytokine release
- MUCB4 gene mutation (chromosome 9) mutation→higher tendency to develop fibrosis
-shorter telomeres (Reduced genes that encode for telomerase)→shorter cell life (type I pneumocytes)→senescence + apoptosis→
when they die they secrete:
- TGF-b1→fibrogenic→stimulating fibroblasts and myofibroblasts > collagen
- Low Caveolin (inhibits TGF-b1> there will be nothing to counteract it)

Treatment:
-Perfinidone (TGF-b1 antagonists) -
-Nentedanib (tyrosine kinase antagonist)

Dr. AlRikabi's notes (Cont.)

3- RDS(Adult/Neonatal), DAD(diffuse alveolar damage), HMPD(hyaline membrane pulmonary disease)

Causes:

-Very severe dyspnea and hypoxia
- Very severe road traffic accident, major surgery, aspiration of gastric content, C section, severe acute pancreatitis, hypovolemic shock, septicemia
-70% die

Effect:

-Edema
in the lung→atelectasis→collapse
The 30% who survive end up with chronic pulmonary fibrosis
Morphology:
CT scan→White lung syndrome Fibrin and debris→form a hyaline membrane around the alveoli

Risk factors of NRDS
(surfactant deficiency):
-Premature neonates (<36 weeks)
- Multiple pregnancies
-Maternal diabetes
- C-section
-Amniotic fluid aspiration

4- Atypical pneumonia

- Could lead to interstitial pneumonitis
- Usually caused by Influenza virus
- Edema in the interstitium and chronic inflammatory infiltration
-Inflammatory cells are lymphocytes (viral infection), and not neutrophils

5- Pneumoconiosis

- Caused by inhaling mineral dust>1-5mm in diameter
Coal→coal worker's pneumoconiosis
Silica→Silicosis (most common) (silica in sand contains quartz, which is fibrogenic)
- Building industry
-Concentric fibrosis
-Higher risk of TB for unknown reasons
Asbestos→Asbestosis
-Carcinogenic (mesothelioma)
- Ship-building industry
- Asbestos fibers causes bleeding > forms hemosiderin (prussian blue stain shows ferruginous bodies)

6- Drug addiction

-Heroin
- Amiodarone (antiarrythmic)

7- Sarcoidosis

-Idiopathic but now thought to be autoimmune
-Symptoms include Uveitis, arthritis, dryness of mouth, lack of lacrimation, etc
- Often confused with TB
(the distinction is that there is no caseation)

8 -Hypersensitivity pneumonitis (extrinsic allergic alveolitis)

-Sensitivity to inhaled organic material
-Ill defined granulomas (poor granulomas)
- Especially in upper lobes
Causes:
- Pigeons - Desert cooler - Incense - Birds
-Farmer's lung→microsporum→ extrinsic allergic alveolitis

Quiz

1) A 40-year-old woman with leukemia is treated with chemotherapy. During treatment she develops increasing cough and shortness of breath. A chest X-ray shows diffuse lung infiltrates. Sputum cultures are negative, and the patient does not respond to routine antibiotic therapy. An open lung biopsy is diagnosed by the pathologist as viral pneumonia. Which of the following histopathologic findings would be expected in the lungs of this patient?

(A) Clusters of epithelioid macrophages
(B) Confluent areas of caseous necrosis
(C) Fibrous scarring of lung parenchyma
(D) Hyaline membranes and interstitial inflammation
(E) Sheets of bacilli-filled macrophages

2) A 50-year-old woman presents with a 4-week history of fever, shortness of breath, and dry cough. She reports that her chest feels "tight." The patient is a pigeon fancier. Blood tests show leukocytosis and neutrophilia, an elevated erythrocyte sedimentation rate, and increased levels of immunoglobulins and C-reactive protein. A lung biopsy reveals poorly formed granulomas composed of epithelioid macrophages and multinucleated giant cells. Which of the following is the appropriate diagnosis?

(A) Actinomycosis
(B) Goodpasture syndrome
(C) Hypersensitivity pneumonitis
(D) Nocardiosis
(E) Wegener granulomatosis

3) A 25-year-old black woman presents with a 3-month history of cough and shortness of breath on exertion. A chest X-ray reveals enlargement of hilar and mediastinal lymph nodes. Laboratory studies show elevated serum levels of angiotensin-converting enzyme and an increase in 24-hour urine calcium excretion.
Stains for microorganisms in the tissue are negative. Which of the following is the most likely diagnosis?

(A) Goodpasture syndrome
(B) Sarcoidosis
(C) Silicosis
(D) Tuberculosis
(E) Wegener granulomatosis

4) A 62-year-old woman is rushed to the emergency room following an automobile accident. She has suffered internal injuries and massive bleeding and appears to be in a state of profound shock. Her temperature is 37°C (98.6° F), respirations are 42 per minute, and blood pressure is 80/40mmHg. Physical examination shows cyanosis and the use of accessory respiratory muscles. A CT scan of the chest is normal on arrival. Her condition is complicated by fever, leukocytosis, and a positive blood culture for staphylococci (sepsis). Two days later, the patient develops rapidly progressive respiratory distress, and a pattern of "interstitial pneumonia" can be seen on a chest X-ray. Which of the following is the most likely diagnosis?
(A) Acutebronchiolitis
(B) Alveolarproteinosis
(C) Atelectasis
(D) Desquamative interstitial pneumonitis
(E) Diffuse alveolar damage

Answer Explanation File

1-D | 2- C | 3- B | 4- E

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Thank you

Pathology
Teamwork438