

PNEUMOCONIOSES

سَحَارٌ ؛ تَغْبِرُ الرِّئَةُ

- ❑ reaction to inhaled inorganic dusts
0.5-5 mm in size
- ❑ no effective treatment, therefore key
is exposure prevention through the
use of protective equipment

Lung diseases caused by exposure to inorganic dusts

Cause	Occupation	Description	Characteristic pathological features
Coal dust	Coal mining	Coal worker's pneumoconiosis	Focal and interstitial fibrosis, centrilobular emphysema, progressive massive fibrosis
Silica	Mining, quarrying, stone dressing, metal grinding, pottery, boiler scaling	Silicosis	
Asbestos	Demolition, ship breaking, manufacture of fireproof insulating materials, pipe and boiler lagging	Asbestos-related disease	Pleural plaques Diffuse pleural thickening Acute benign pleurisy Carcinoma of lung Interstitial fibrosis Mesothelioma
Iron oxide	Arc welding	Siderosis	Mineral deposition only
Tin oxide	Tin mining	Stannosis	Tin-laden macrophages
Beryllium	Aircraft, atomic energy and electronics industries	Berylliosis	Granulomas, interstitial fibrosis

Lung diseases caused by inorganic gases and fumes

Cause	Occupation	Disease
Irritant gases (chlorine, ammonia, phosgene, nitrogen dioxide)	Various (industrial accidents)	Acute lung injury ARDS
Cadmium	Welding and electroplating	COPD
Isocyanates (e.g. epoxy resins, paints)	Plastic, paints; manufacture of epoxy resins and adhesives	Bronchial asthma Eosinophilic pneumonia

PNEUMOCONIOSES

- ⦿ prolonged exposure to inorganic dusts leads to diffuse pulmonary fibrosis
- ⦿ beryllium causes an interstitial granulomatous disease
- ⦿ silica is highly fibrogenic
- ⦿ iron and tin are almost inert.
- ⦿ a long period of dust exposure is required before radiological changes appear.

Asbestosis

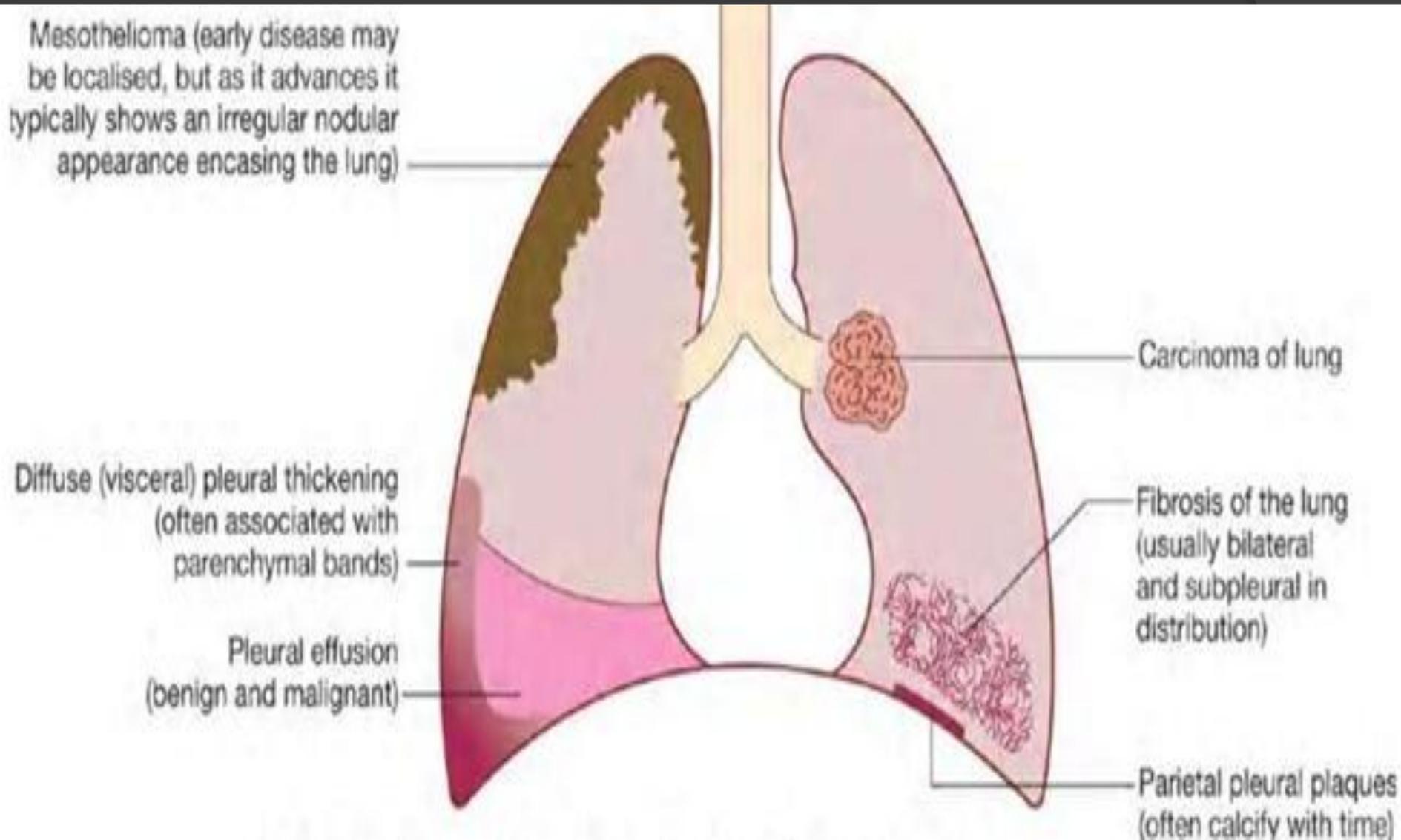
داءُ الأَسْبَسْت؛ دَاءُ الأَمِيَانْت

- ⦿ **workers at risk:**
insulation, shipyard, construction, brake linings
- ⦿ **usually need > 10-20 years of exposure; may develop with shorter but heavier exposure**
- ⦿ **asbestos exposure also increases risk of bronchogenic CA and malignant mesothelioma**
 - risk dramatically increased for smokers
- ⦿ **clubbing is much more likely in asbestosis than silicosis or coal worker's pneumoconiosis**

Clinical picture

- **exertional breathlessness**
- **fine late inspiratory crackles over the lower zones.**
- **Digital clubbing (40% of patients)**
- **40% of patients (who usually smoke) develop carcinoma of the lung**
- **10% may develop mesothelioma**

Asbestos exposure. The range of possible effects on the respiratory tract.



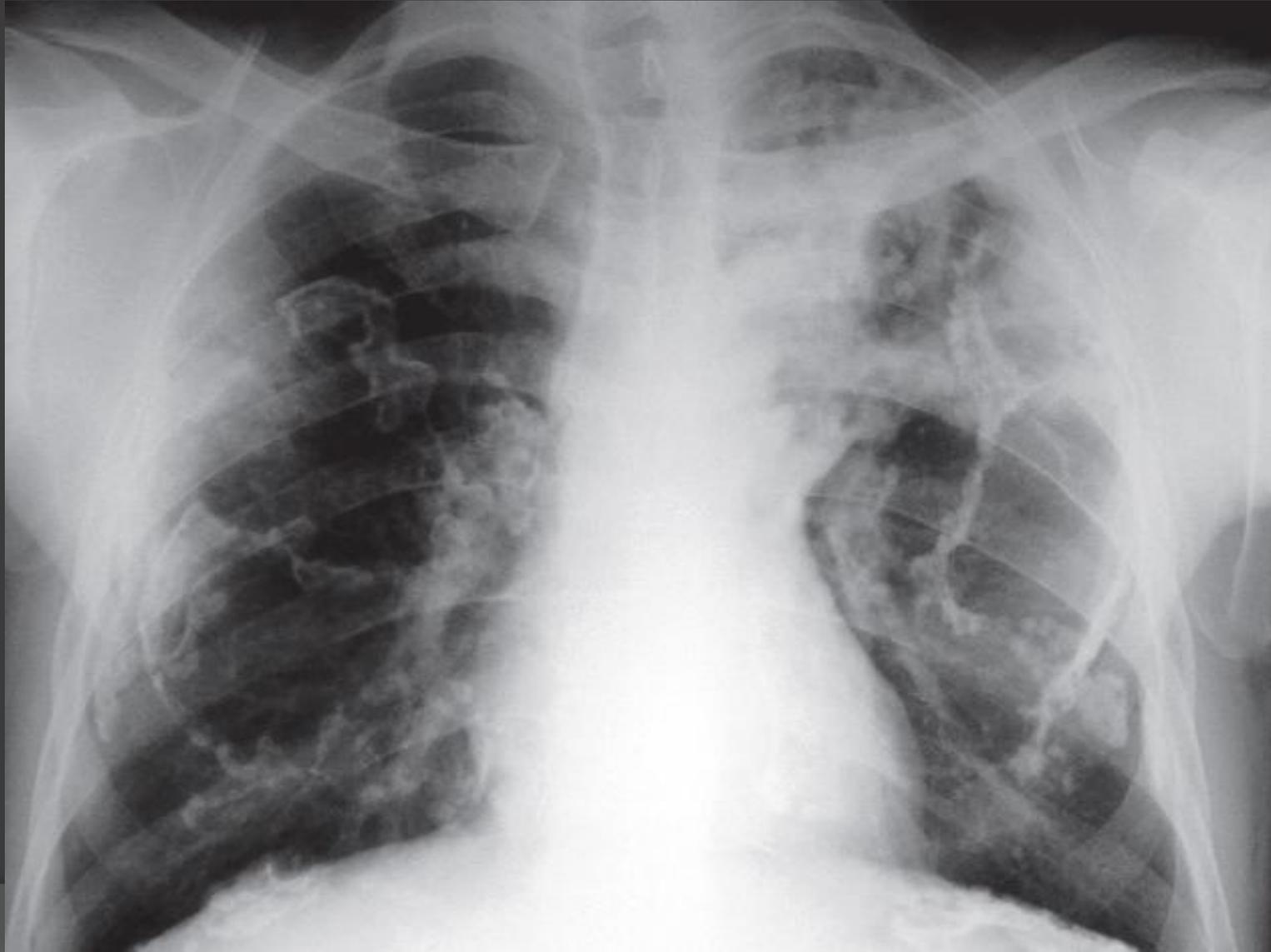
CXR

- **lower > upper lobe**
- **bi-basal reticulonodular shadowing**
- **early: fibrosis with linear streaking**
- **later: cysts and honeycombing**
- **asbestos exposure can also cause pleural thickening (+/- calcification) or pleural effusion**

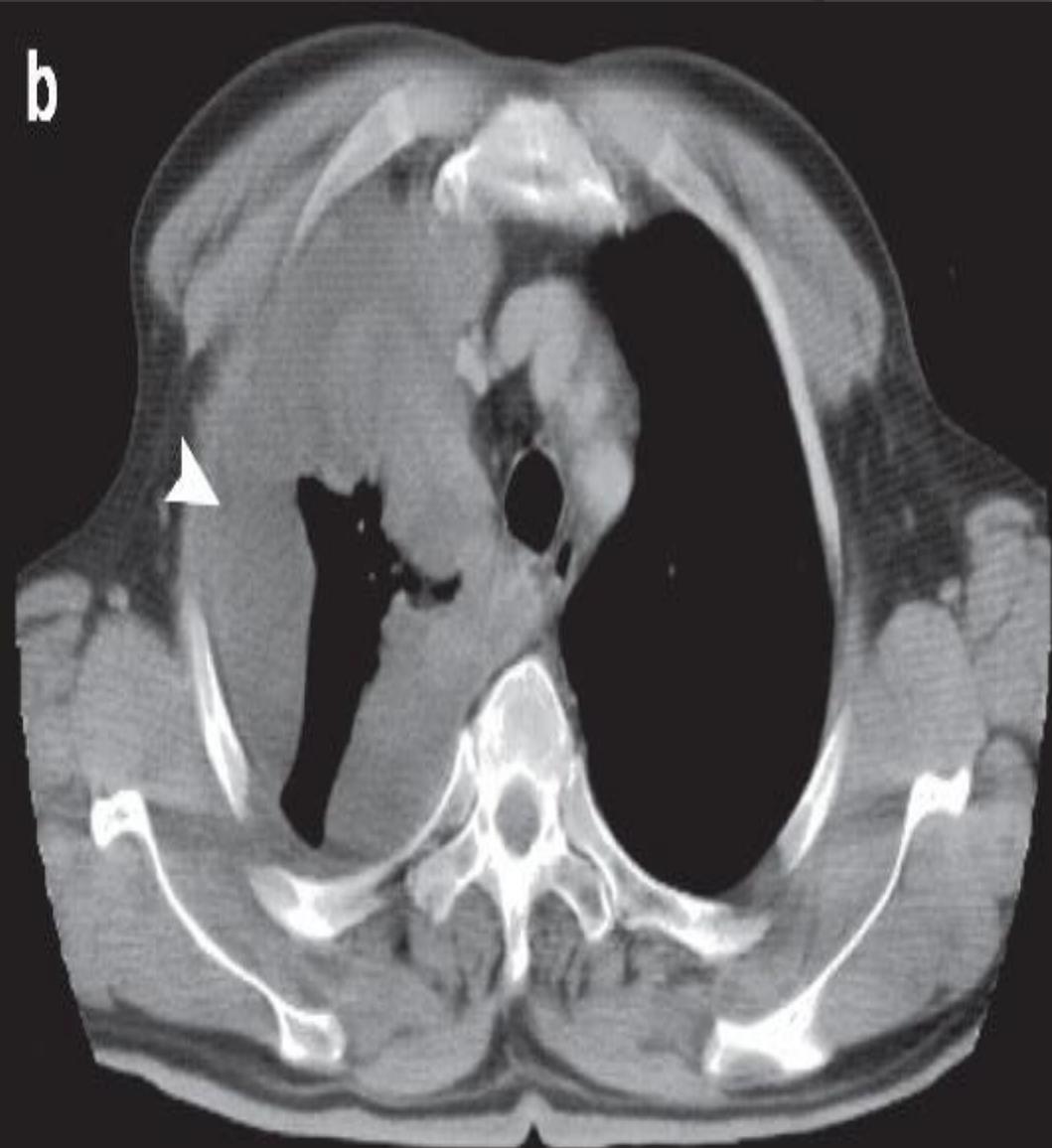
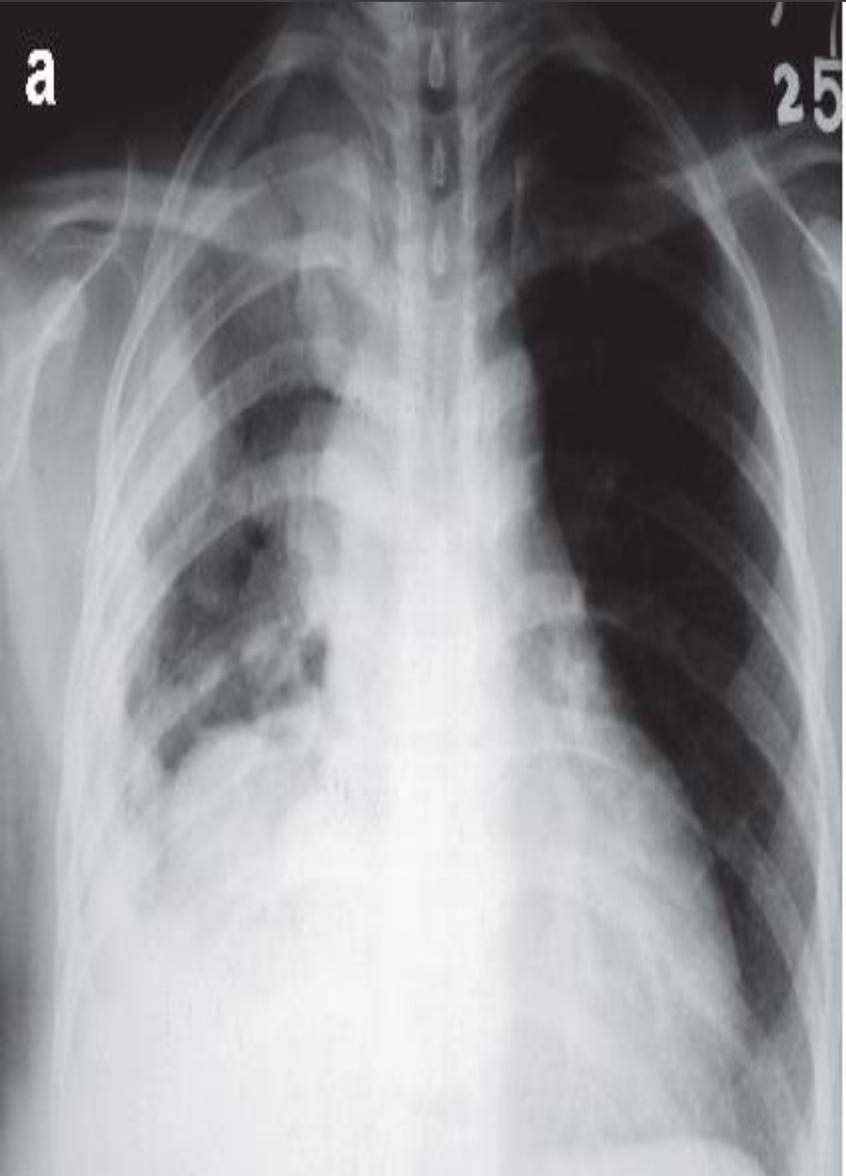
Asbestos-related benign pleural plaques. Chest X-ray showing extensive calcified pleural plaques ('candle wax' appearance), particularly marked on the diaphragm and lateral pleural surfaces.



asbestosis shows bilateral calcified pleural plaques



Posteroanterior chest radiograph (a) and axial chest CT (b) of a patient with malignant pleural mesothelioma show nodular thickening of the pleura in the right lung field with extension toward the apex in (a) and (b). Notice the nodular mass that follows the pleural distribution in (b)



HRCT

- ⦿ **more sensitive than plain radiography**
- ⦿ **typically :**
bi-basal, subpleural, dot-like opacities, curvilinear subpleural lines, ground glass opacification and interlobular septal thickening.
- ⦿ **In more advanced disease :**
honeycombing may be present.



microscopic examination

- ① **Asbestos bodies may be identified in sputum or BAL or Lung biopsy**
- ① **yellow-brown rod-shaped structures which represent asbestos fibres coated in macrophages**

treatment

- ⦿ prevention of disease progression and development of complications
- ⦿ reduce exposure

Silicosis

- ❑ **workers at risk:**
 - sandblasters, rock miners, quarry workers, stone cutters
 - inhalation of crystalline or free silica (quartz)

- ❑ **generally need > 20 years of exposure; may develop with much shorter but heavier exposure**

- **Accelerated silicosis :**
 - shorter duration of dust exposure (typically 5-10 years), may present as early as 1 year of exposure

- **symptoms : cough, with sputum and breathlessness.**

- ❑ **risk factor for mycobacterial infection (i.e. TB)**
 - increased risk of lung cancer and COPD

CXR

- ⦿ upper > lower lobe
 - ⦿ multiple well-circumscribed 3-5 mm nodular opacities
 - ⦿ early: nodular disease (simple pneumoconiosis)
 - ⦿ late: nodules coalesce and enlarge (progressive massive fibrosis)
 - ⦿ egg shell calcification is classical & non-specific
- ☐ *when nodules become larger and coalescent, disease has changed from simple silicosis to complicated silicosis (progressive massive fibrosis)*
- ☐ *possible hilar lymph node enlargement (frequent calcification)*

Silicosis.

A chest X-ray from a patient with silicosis showing the presence of small rounded nodules predominantly seen in the upper zones.

HRCT from the same patient demonstrating conglomeration of nodules with posterior bias.



treatment

- ① prevention, removal from exposure

Berylliosis

- ⦿ **a worker exposed to dusts, fumes or vapours containing beryllium**
- ⦿ **cough, progressive breathlessness, night sweats and arthralgia**
- ⦿ **radiographic appearances are similar in type and distribution to sarcoid**
- ⦿ **biopsy shows sarcoid-like granulomas.**

Coal Worker's Pneumoconiosis (CWP)

- ❑ coal is less fibrogenic than silica**

- ❑ pathologic hallmark is coal macule:**
 - coal dust surrounded by little tissue reaction and focal emphysema**

 - found around respiratory bronchioles near the centre of the secondary pulmonary lobule and a fibrotic reaction ensues**

simple CWP

- ① **no signs or symptoms**
- ① **CXR:**
multiple nodular opacities, mostly upper lobe
- ① **respiratory function well preserved**

complicated CWP

(progressive massive fibrosis)

- usually associated with cough, sputum that may be black (melanoptysis), and breathlessness.
 - Caplan's syndrome :
coexistence of rheumatoid arthritis and pneumoconiosis, with rounded fibrotic nodules 0.5-5 cm in diameter.
 - extreme cases :
respiratory failure and right ventricular failure.
 - CXR:
opacities larger and coalesce
(large dense masses mainly in the upper lobes; cavitation may occur)
- only small minority progress to complicated

Drug-Induced INTERSTITIAL LUNG DISEASE

- ❑ chemotherapeutics: bleomycin, mitomycin, busulfan, cyclophosphamide, MTX
- ❑ amiodarone
- ❑ gold
- ❑ nitrofurantoin

Drug-induced respiratory disease

Non-cardiogenic pulmonary oedema (ARDS)

- Hydrochlorothiazide
- Thrombolytics (streptokinase)
- I.v. β -adrenoceptor agonists (e.g. for premature labour)
- Aspirin and opiates (in overdose)

Non-eosinophilic alveolitis

- Amiodarone, flecainide, gold, nitrofurantoin, cytotoxic agents-especially bleomycin, busulfan, mitomycin C, methotrexate, sulfasalazine

Pulmonary eosinophilia

- Antimicrobials (nitrofurantoin, penicillin, tetracyclines, sulphonamides, nalidixic acid)
- Antirheumatic agents (gold, aspirin, penicillamine, naproxen)
- Cytotoxic drugs (bleomycin, methotrexate, procarbazine)
- Psychotropic drugs (chlorpromazine, dosulepin, imipramine)
- Anticonvulsants (carbamazepine, phenytoin)
- Others (sulfasalazine, nadolol)

Pleural disease

- Bromocriptine, amiodarone, methotrexate, methysergide
- Induction of SLE-phenytoin, hydralazine, isoniazid

Asthma

- Pharmacological mechanisms (β -blockers, cholinergic agonists, aspirin and NSAIDs)
- Idiosyncratic reactions (tamoxifen, dipyridamole)

parenchymal reactions of drugs

- ⦿ **ARDS**
- ⦿ **eosinophilic reactions**
- ⦿ **diffuse interstitial inflammation/scarring (most frequently with bleomycin, methotrexate, amiodarone and nitrofurantoin).**
- ⦿ **Asthma**
- ⦿ **pleural effusions and pleura**

Radiation-Induced INTERSTITIAL LUNG DISEASE

- the effects are cumulative.
- ❑ early pneumonitis: 1-3 months post-exposure
- Acute radiation pneumonitis :
 - cough and dyspnoea.
 - may resolve spontaneously but responds to corticosteroid treatment.
- ❑ late fibrosis: 6-12 months post-exposure (exertional dyspnoea and cough).
- ❑ infiltration conforms to the shape and field of the irradiation
- post-irradiation fibrosis does not usually respond to corticosteroid treatment.
- exacerbated by treatment with cytotoxic drugs