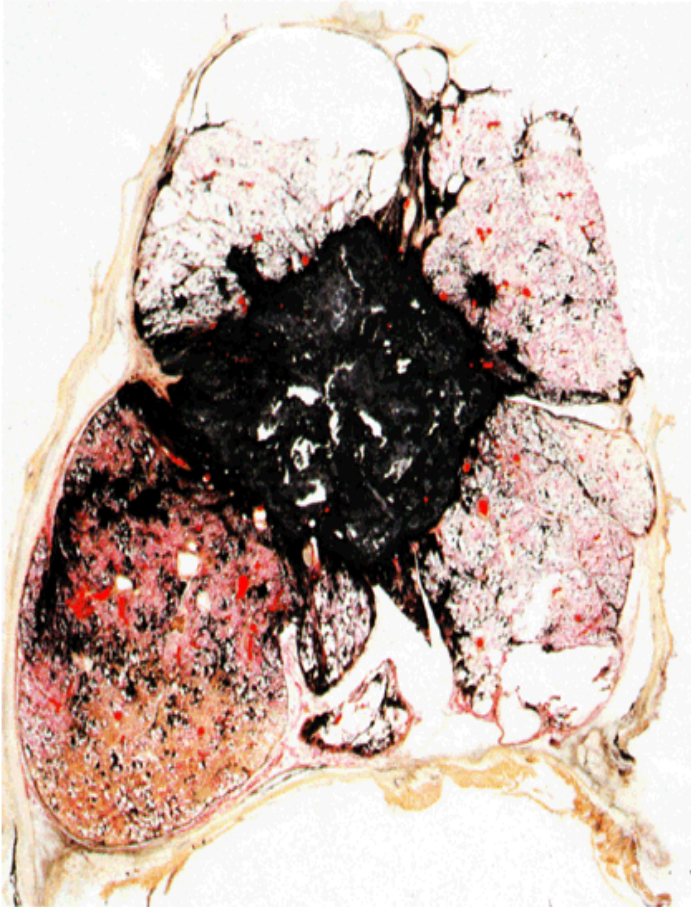


Occupational Lung diseases



Dr Deepak Aggarwal
Dept. Of Pulmonary Medicine

To be discussed...

- Pneumoconiosis
- Hypersensitivity Pneumonitis

PNEUMOCONIOSIS

Pneumoconioses are pulmonary diseases caused by mineral dust inhalation in workplace

The specific types of pneumoconioses are named by the substance inhaled (e.g., silicosis, asbestosis, anthracosis)

TABLE 15-6 Lung Diseases Caused by Air Pollutants

Agent	Disease	Exposure
Mineral Dusts		
Coal dust	Anthracosis Macules Progressive massive fibrosis Caplan syndrome	Coal mining (particularly hard coal)
Silica	Silicosis Caplan syndrome	Foundry work, sandblasting, hardrock mining, stone cutting, others
Asbestos	Asbestosis Pleural plaques Caplan syndrome Mesothelioma Carcinoma of the lung, larynx, stomach, colon	Mining, milling, and fabrication; installation and removal of insulation
Beryllium	Acute berylliosis Beryllium granulomatosis Bronchogenic carcinoma (?)	Mining, fabrication
Iron oxide	Siderosis	Welding
Barium sulfate	Baritosis	Mining
Tin oxide	Stannosis	Mining
Organic Dusts That Induce Hypersensitivity Pneumonitis		
Moldy hay	Farmer's lung	Farming
Bagasse	Bagassosis	Manufacturing wallboard, paper
Bird droppings	Bird-breeder's lung	Bird handling
Organic Dusts That Induce Asthma		
Cotton, flax, hemp	Byssinosis	Textile manufacturing
Red cedar dust	Asthma	Lumbering, carpentry
Chemical Fumes and Vapors		
Nitrous oxide, sulfur dioxide, ammonia, benzene, insecticides	Bronchitis, asthma Pulmonary edema ARDS* Mucosal injury Fulminant poisoning	Occupational and accidental exposure

*Acute respiratory distress syndrome.

PNEUMOCONIOSIS

Mineral Dust-Induced Lung Disease

Coal dust	Simple coal workers' pneumoconiosis: macules and nodules Complicated coal workers' 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

Fibrogenic pneumoconioses

"true pneumoconioses"

- asbestosis
- silicosis
- hard metal disease
- aluminum fibrosis,
Shaver's disease

- berylliosis
- talcosis
- kaolin pneumoconiosis.
- coal workers'
pneumoconiosis

Non-fibrogenic pneumoconioses

benign pneumoconioses

Causes:

- antimony
- barium
- boric acid
- manganese
- iron
- tin
- titanium
- bismuth

PNEUMOCONIOSIS

Pathogenesis

The development of a pneumoconiosis depends on

- (1) the amount of dust retained in the lung and airways
- (2) the size, shape, and buoyancy of the particles
- (3) solubility and physiochemical reactivity
- (4) the possible additional effects of other irritants (e.g., concomitant tobacco smoking)

PNEUMOCONIOSIS

Pathogenesis

(1) The amount of dust retained in the lungs is determined by

- dust concentration in surrounding air
- duration of exposure
- effectiveness of clearance mechanisms

PNEUMOCONIOSIS

Pathogenesis

(2) the size, shape, and buoyancy of the particles

The most dangerous particles range from 1 to 5 μm in diameter because they may reach the terminal small airways and air sacs and settle in their linings

PNEUMOCONIOSIS

(3)The solubility and cytotoxicity of particles modify the nature of the pulmonary response

Smaller particles tend to cause acute lung injury

Larger particles resist dissolution and so may persist within the lung parenchyma for years - tend to evoke fibrosing collagenous pneumoconioses

PNEUMOCONIOSIS

Pathogenesis

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

The more **reactive particles** trigger the macrophages to release a number of products that mediate **an inflammatory response** and initiate **fibroblast proliferation** and **collagen deposition**

PNEUMOCONIOSIS

Pathogenesis

(4) the possible additional effects of other irritants (e.g., concomitant tobacco smoking)

➤ tobacco smoking worsens the effects of all inhaled mineral dusts

Coal worker pneumoconiosis

- Coal Workers' Pneumoconiosis Is Due to Inhalation of Carbon Particles
- Associated with coal mining industry
- Carbon + silica (anthracosilicosis)

Findings

The spectrum of lung findings in coal workers is wide, varying from

- (1) Asymptomatic anthracosis
- (2) Simple CWP with little to no pulmonary dysfunction
- (3) Complicated CWP (progressive massive fibrosis)

Anthracosis (urban dwellers) morphology

- Carbon particles (anthrocoitic pigment) in alveolar and interstitial macrophages, in connective tissue and lymphatics and lung hilus.
- Generally asymptomatic

Simple CWP

Coal macules (1 to 2 mm in diameter, consists of peribronchiolar carbon-laden macrophages and dilated terminal bronchiole)

larger coal nodules (contains small amounts of a delicate network of collagen)

located primarily adjacent to respiratory bronchioles

Microscopy:

- Carbon laden macrophages & delicate collagen fibres adjacent to respiratory bronchioles initially (where dust settles), later interstitium & alveoli
- Dilatation of respiratory bronchioles –focal dust emphysema

Radiographic finding of bilateral small parenchymal nodules

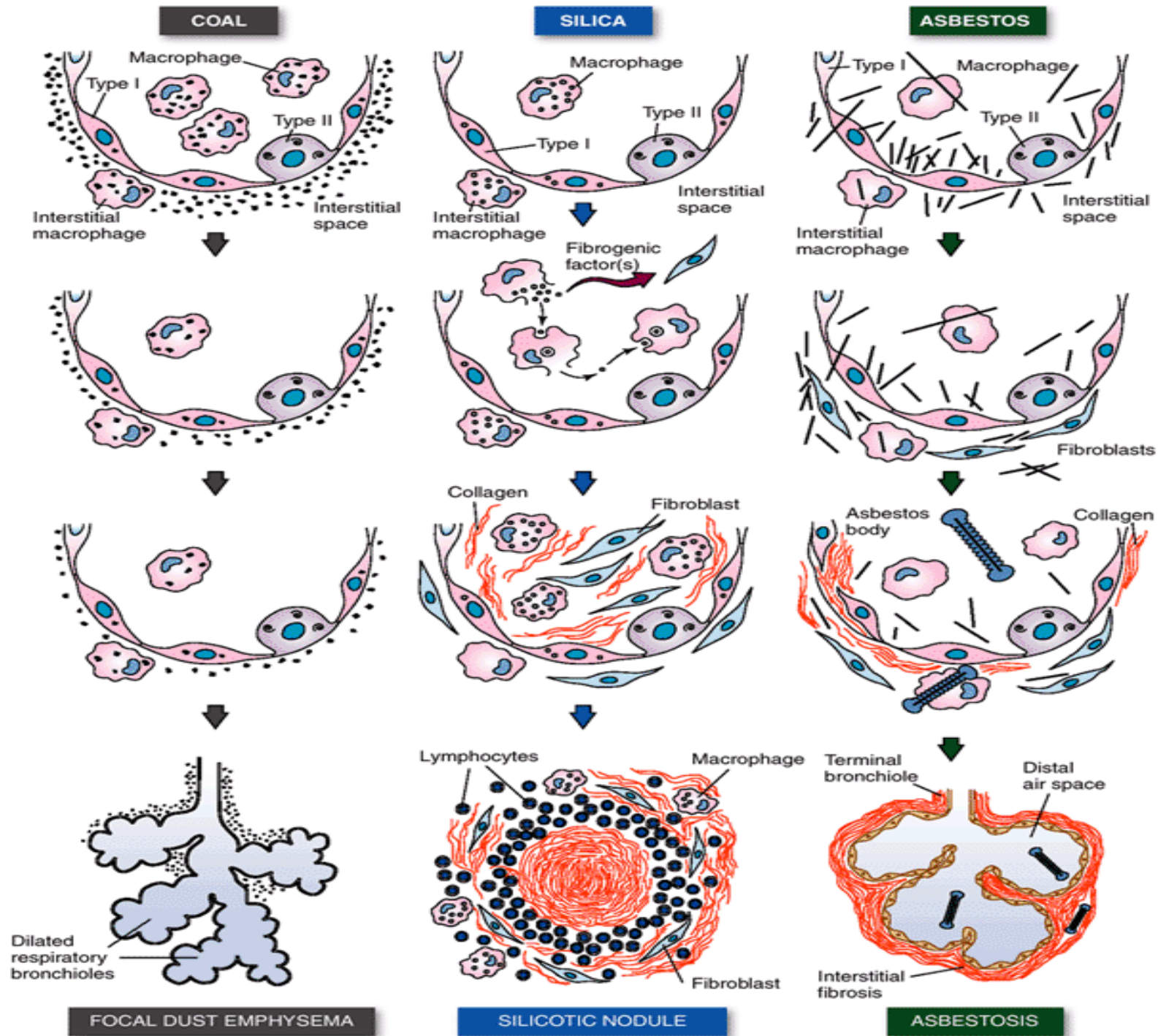
- CXR typically shows **upper lung field** nodules

Complicated CWP

- **Gross**
- Multiple., >2 cm ,v dark scars
- **Microscopy:**
- Dense collagen and carbon pigment.
- Central necrosis (+/-)
- Progression does NOT correlate with amount of coal dust deposition in lungs.
- Cigarette smoking increases rate of deterioration of pulmonary function.

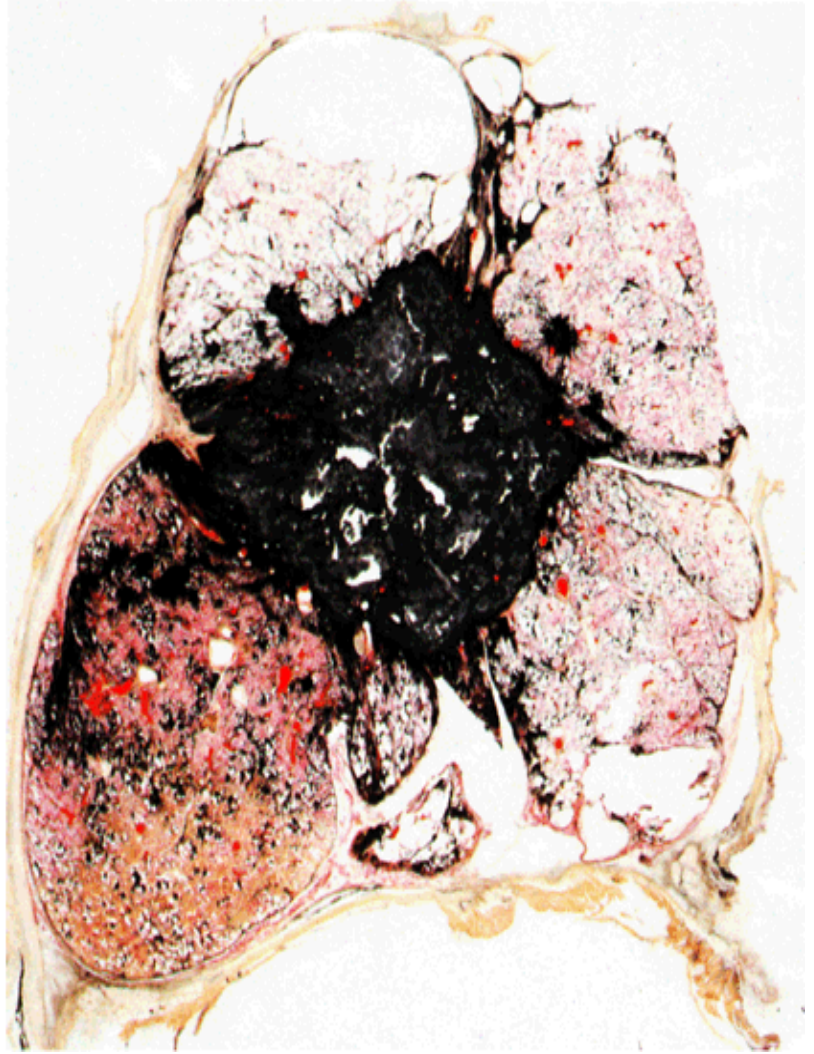
Caplans syndrome

- 1st described in coal workers, may be seen in other pneumoconiosis
- ?? Immunopathologic mechanism
- Rheumatoid arthritis (RA) + Rheumatoid nodules (Caplan nodules) in the lung
- **Rheumatoid arthritis + pneumoconioses**
- Caplans nodule = necrosis surrounded by fibroblasts, monocytes and collagen
- s/s RA > lung symptoms



Clinical course

- Usually asymptomatic with little decrease of lung function
- Progressive Dyspnoea, cough, expectoration
- PMF pulmonary dysfunction (restrictive)
- Pulmonary hypertension, cor pulmonale
- Progressive even if further exposure to dust is prevented
- ↑ chronic bronchitis and emphysema
- No association with TB or carcinoma



Management

- Diagnosis by presence of clinical features along with history of exposure to coal dust of the magnitude that is sufficient to cause the disease
- Imaging, PFT, sputum examination
- Treatment: no specific treatment, removal from further exposure is important.
- Treat underlying airway disease

Silicosis

- Silica is silicon dioxide, the oxide of silicon, chemical formula SiO_2 .
- SiO_2 is the most abundant mineral on earth
- **Silicosis** (also known as **Grinder's disease** and **Potter's rot**) is a form of occupational lung disease caused by inhalation of crystalline silica dust, and is marked by inflammation and scarring in forms of nodular lesions in the upper lobes of the lungs

- It is found in sand, many rocks such as granite, sandstone, flint and slate, and in some coal and metallic ores.
- The cutting, breaking, crushing, drilling, grinding, or abrasive blasting of these materials may produce fine silica dust.

Silicosis – Foundry work



Silicosis - Stone cutting

-



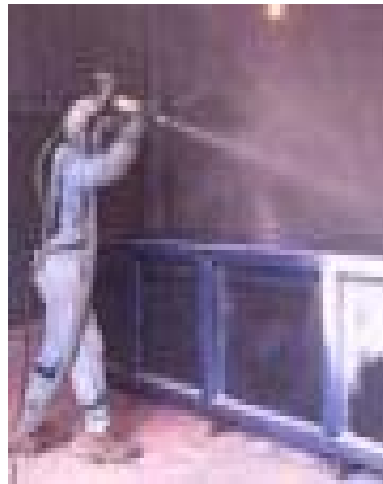
Silicosis - Tunnel construction

Worst single incidence of silicosis in U.S. –
Hawk's Nest Tunnel, Gauley Bridge, W. Va., 1930-1931



Silicosis - Sandblasting

- Compressed air at high pressure is used to blow *fine sand or other abrasive material* through a hardened spray nozzle. The abrasive particles quickly eat away whatever they are directed at, leaving a clean, matte surface.



Diseases Associated with Exposure to Silica Dust

- **Silicosis**
 - Chronic silicosis
 - Accelerated silicosis
 - Acute silicosis (silicoproteinosis)(fine dust, intense exposure , high silica)
 - Progressive massive fibrosis
- **Chronic Obstructive Pulmonary Disease**
 - Emphysema
 - Chronic bronchitis
 - Mineral dust-induced small airway disease

Diseases Associated with Exposure to Silica Dust

- **Lung Cancer**
- **Mycobacterial Infection**
- **Immune-Related Diseases**
 - Progressive systemic sclerosis
 - Rheumatoid arthritis
 - Chronic renal disease
 - Systemic lupus erythematosus

Silicosis

- **The most prevalent occupational disease in the world.**
- The induction period between initial silica exposure and development of radiographically detectable nodular silicosis is usually **>10 years**. Shorter induction periods are associated with heavy exposures, and acute silicosis may develop within **6 months to 2 years following massive silica exposure**

Three 'types' of **Silicosis**

- Simple chronic silicosis
- Most common form
- After long-term exposure (10-20 years) to low amounts of silica dust.
- **Nodules** of chronic inflammation and **scarring** form in the lungs and chest lymph nodes.
- Patients often asymptomatic, seen for other reasons.
- subdivided into:
 - simple
 - complicated silicosis(PMF)

Accelerated silicosis (= PMF, progressive massive fibrosis)

- Occurs after exposure to larger amounts of silica over a shorter period of time (5-10 years).
- Inflammation, scarring, and symptoms progress faster in accelerated silicosis
- Patients have symptoms, especially shortness of breath.

Acute silicosis

- From short-term exposure to very large amounts of fine silica dust.
- The lungs become very inflamed, causing severe shortness of breath and low blood oxygen level.
- Killed hundreds of workers during Hawk's Nest Tunnel construction in early 1930s.

CLINICAL FEATURES

- The main symptom is **breathlessness**, first noted during exertion and later at rest as the large working reserve of the lung is diminished.
- a patient with chronic silicosis may present **without symptoms** for assessment of an abnormal chest radiograph
- **Cough and sputum production** are common symptoms and usually relate to chronic bronchitis
- **Clubbing** is also not a feature of silicosis

- Patients with silicosis are particularly susceptible to tuberculosis (TB) infection - known as **silicotuberculosis**. The reason for the increased risk - **10-30 fold increased incidence** - is not well understood. It is thought that **silica damages pulmonary macrophages, inhibiting their ability to kill mycobacteria**

Diagnosis of Silicosis

- In general, three key elements play a role in the diagnosis of silicosis:
- **A history of silica exposure** sufficient to cause the degree of illness and the appropriate latency from the time of first exposure
- Chest imaging (usually a conventional chest radiograph) that shows **opacities consistent with silicosis**
- **Absence of another diagnosis** more likely to be responsible for the observed abnormalities

- *Pulmonary function tests are helpful to gauge severity of impairment, but NOT for diagnosis.*
- *Lung biopsy rarely indicated* (since no effective treatment, biopsy is done only when other diagnoses are being considered)

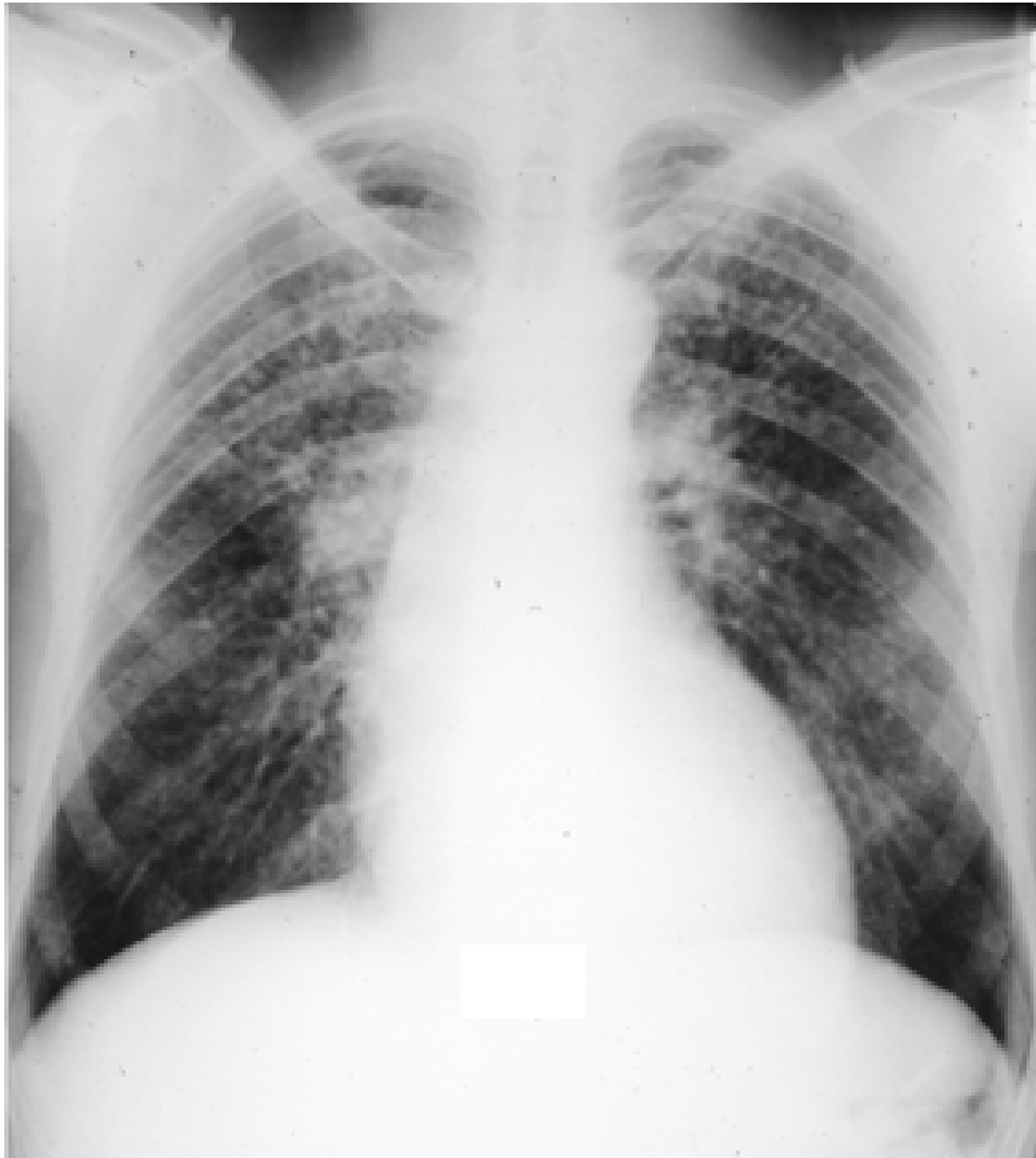
Silicosis can be misdiagnosed

- Silicosis can mimic:
 - Sarcoidosis (benign inflammation of unknown cause)
 - Idiopathic pulmonary fibrosis (lung scarring of unknown cause)
 - Lung cancer
 - Several other lung conditions (chronic infection, collagen-vascular disease, etc.)

Can usually make right diagnosis with detailed history (occupational & medical) or, rarely, a lung biopsy.

- The **three main radiographic presentations** of silicosis are:
 - simple silicosis
 - progressive massive fibrosis
 - silicoproteinosis

- Simple silicosis refers to a profusion of small (less than 10 mm in diameter) nodular opacities (nodules). The nodules are generally rounded but can be irregular, and are distributed predominantly in the upper lung zones

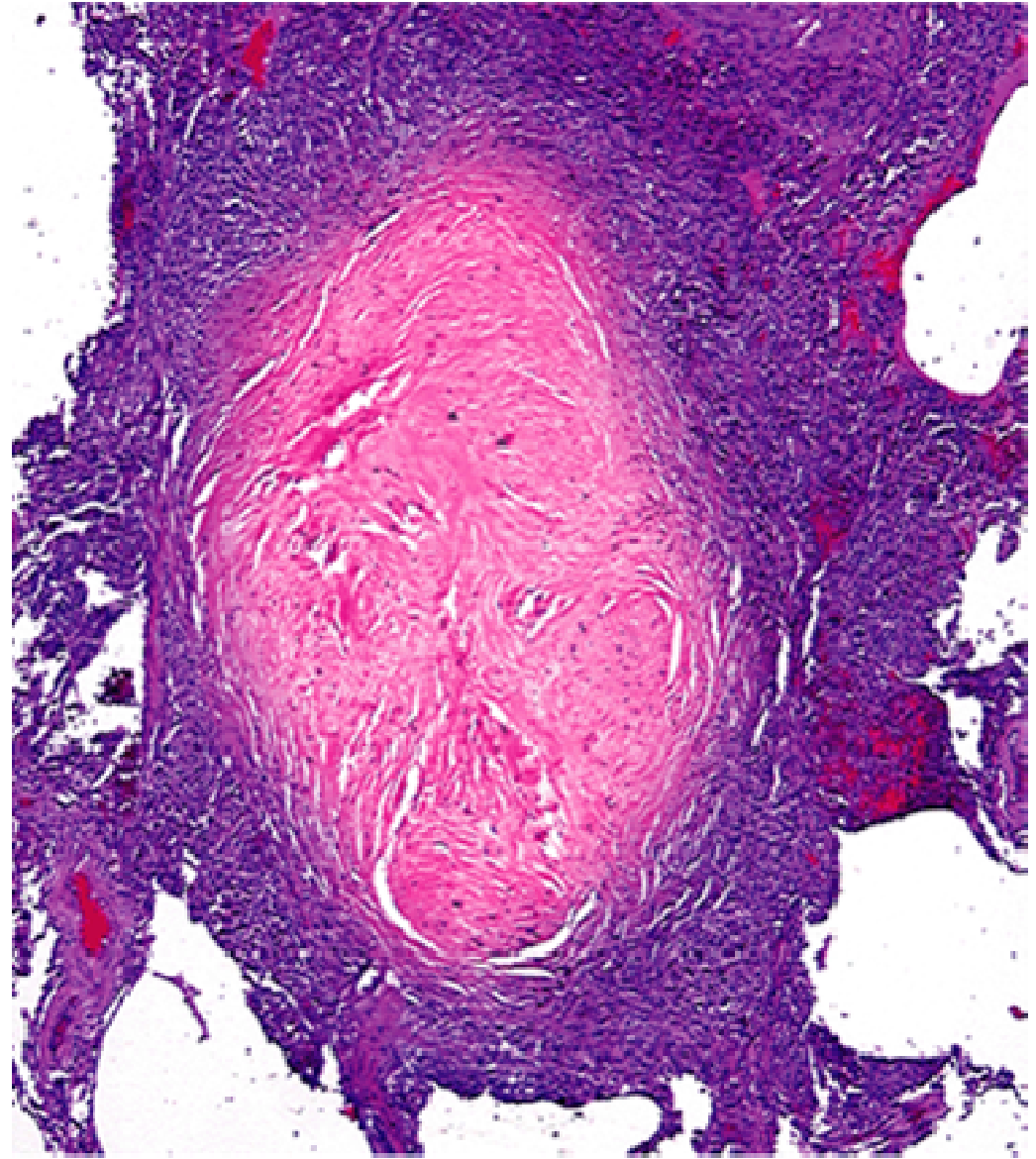


Eggshell calcification – almost exclusively silicosis



- Progressive massive fibrosis (PMF, or conglomerate silicosis) occurs when these small opacities gradually enlarge and coalesce to form larger, upper- or mid-zone opacities more than 10 mm in diameter
- The hila are retracted upward in association with upper lobe fibrosis and lower lobe hyperinflation





Silicoproteinosis

- Silicoproteinosis occurs following overwhelming exposure to respirable crystalline silica over a short time, and is the radiographic hallmark of acute silicosis *The chest radiograph demonstrates a characteristic basilar alveolar filling pattern, without rounded opacities or lymph node calcifications.*

Treatment

- Silicosis is an irreversible condition with no cure. Treatment options currently focus on alleviating the symptoms and preventing complications
- The disease will generally progress even without further exposure, but the rate of deterioration is probably reduced
- Treatment of all forms of silicosis should be directed toward control of mycobacterial disease.
- Lung lavage, transplantation
- Prevention is the key

Asbestosis

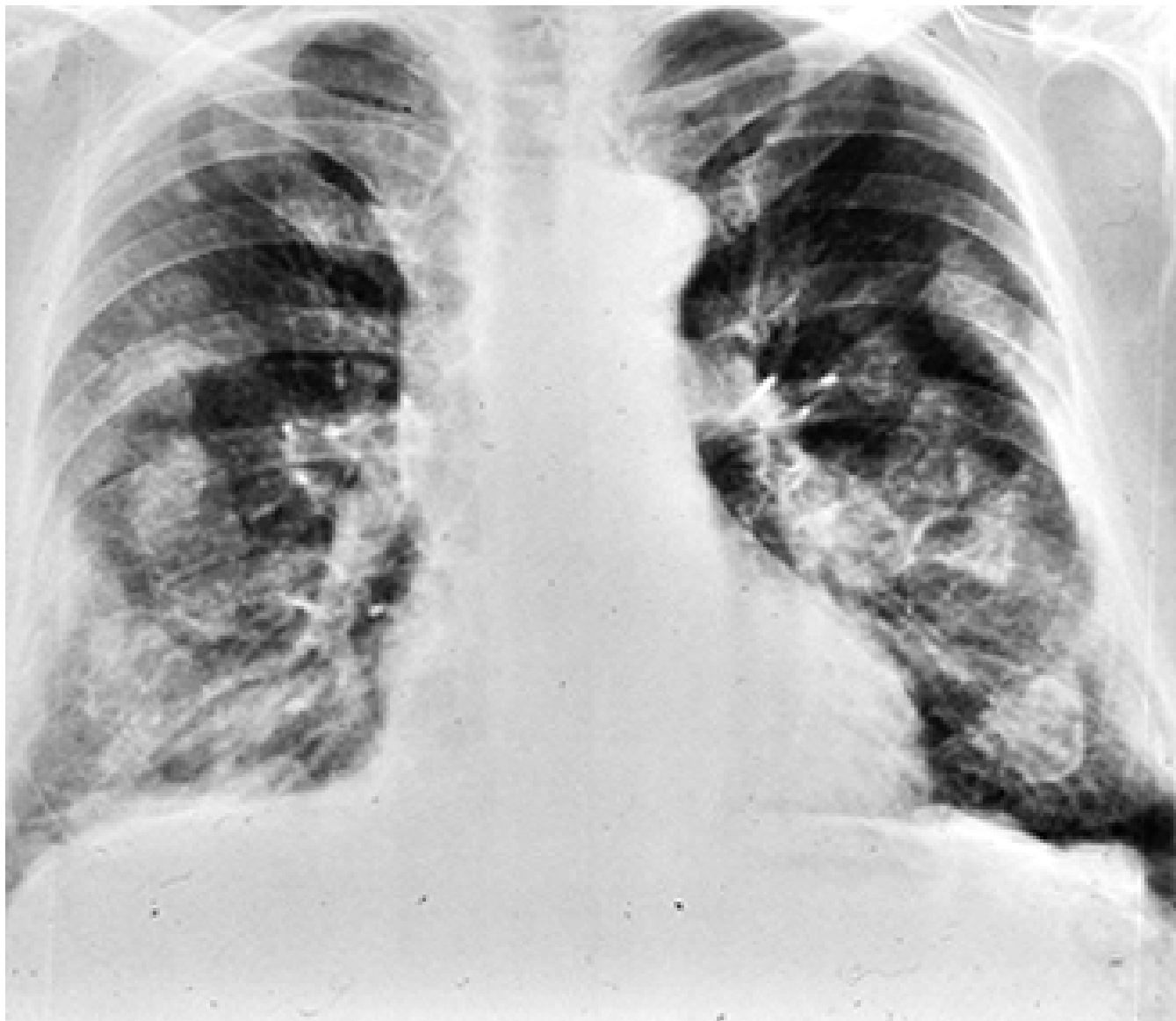
- The pulmonary parenchymal fibrosis develops **mostly in the bases**.
- Generally occurs **with >10 years exposure**, but the latency period can be >30 years.
- Smoking has a synergistic effect with asbestosis in the development of lung cancer.
- Clinically is indistinguishable from IPF
- Associated lung CA: Squamous and adenocarcinoma, NOT small or large cell.

- Asbestos is a fibrous hydrated magnesium silicate with more than 3000 commercial uses due to its indestructible nature, fire resistance, and spinnability
- fireproof textiles, as insulation for boilers and pipes, used in paper, paints, cloth, tape, filters, and wire insulation. More recently, asbestos has been used in cement pipes and in friction materials, including brake linings, and roofing and floor products.

Manifestations

- ***Pleural Plaque: most common***
- They are focal, irregular, raised white lesions found on the parietal and, rarely, the visceral pleura
- commonly they occur in the **lateral and posterior midlung zones**, where they may follow rib contours and the diaphragm.
- **Histologically**: paucity of cells, extensive collagen fibrils arranged in a basket-weave pattern, and a thin covering of mesothelial cells
- **Usually bilateral** and remain stable over months
- No treatment; observation with periodic CXR

Asbestos exposure





Pleural plaque. The dome of the diaphragm is covered by a smooth, pearly white, nodular plaque



Asbestos-related pleural plaques
Large, discrete fibrocalcific plaques are seen on the pleural surface of the diaphragm

- **Diffuse Pleural thickening**
- thick white peel that can encase significant pulmonary structures.
- diffuse pleural thickening or fibrosis is a disease of the visceral pleura
- *Develops either due to confluence of pleural plaques, due to extension of subpleural fibrosis or due to fibrotic resolution of benign pleural effusion.*
- Assymptomatic or may cause s/s
- No specific therapy

- **Rounded atelectasis**
- Rare complication
- It is caused by scarring of the visceral and parietal pleura and the adjacent lung, with the pleural reaction folding over on itself.



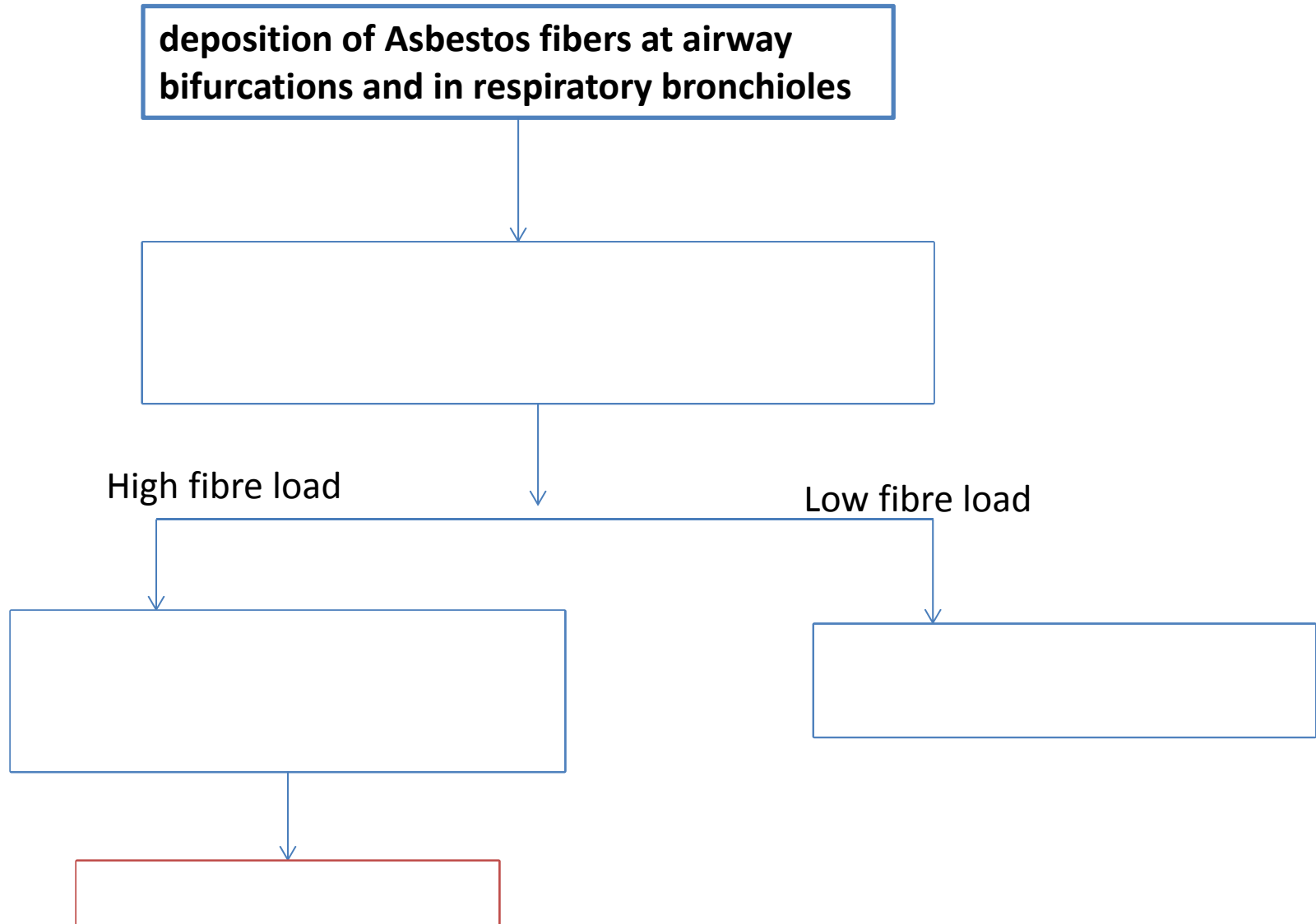
Malignancies

- **Mesothelioma:** Malignant mesotheliomas are associated (80%) with asbestos exposure, and latency period can be as long as 40 years. Unlike in asbestosis, it is NOT associated with smoking and tends to be rapidly fatal
- **Lung cancer:** besides smoking tobacco, asbestos exposure has been linked to increased incidence of lung cancer.
- Adenocarcinoma: most common histological type

Asbestosis

- **Interstitial pneumonitis and fibrosis caused by exposure to asbestos fibers.**
- Macrophage accumulation is a prominent feature of this cellularity.
- The prevalence of parenchymal asbestosis among asbestos workers increases as the length of employment increases.

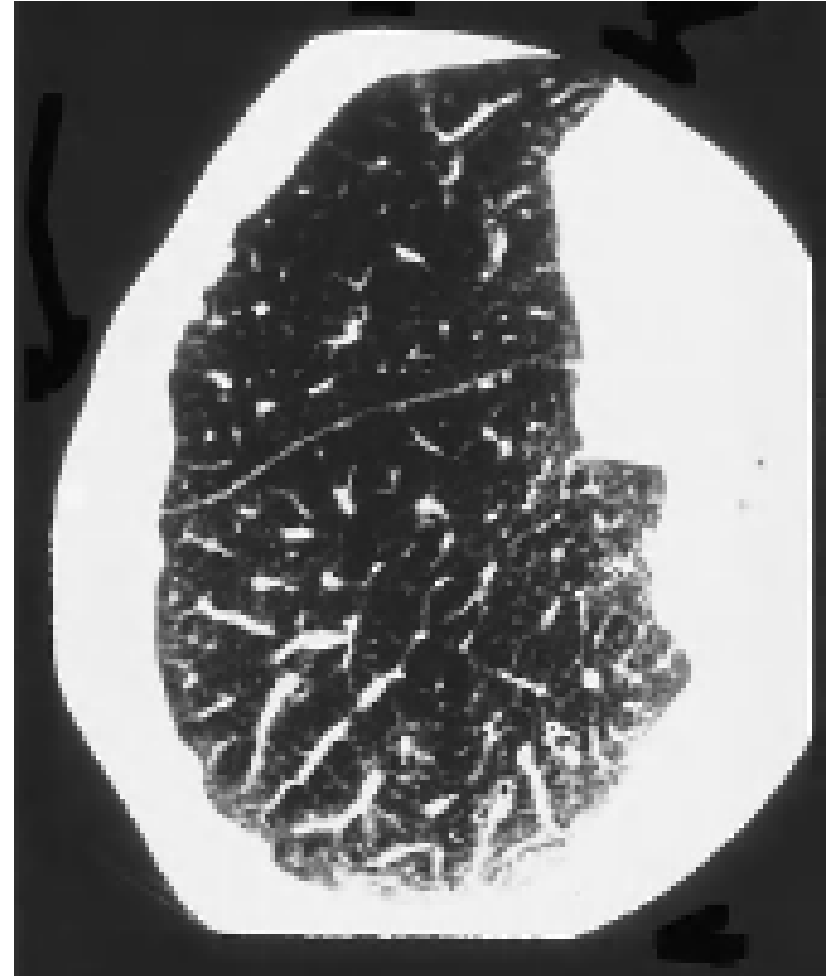
Course of Asbestosis



- Depending on the duration and intensity of exposure, the latent period for the development of symptoms can vary from 1 decade to 2- 3 decades
- Dyspnoea, cough, rales (bilateral, late to paninspiratory) heard best at posterior lung bases
- Bilateral diffuse reticulo-nodular pattern on CXR

CT scan thorax

1. curvilinear subpleural Lines
2. increased intralobular septa
3. dependent opacities,
4. parenchymal bands and interlobular core structures
5. honeycombing.



Diagnosis

- Presence of symptoms along with history of exposure to asbestos
- Duration, onset, type, intensity of exposure
- CXR /CT
- PFT

Bronchoscopy

- Bronchoscopy: biopsy/Bal may show the presence of coated asbestos fibres which are called as **asbestos bodies**
- The presence of more than one coated fiber has been cited as a necessary criterion for the pathological diagnosis of asbestosis



Treatment

- No established treatment available for the disease
- Medical surveillance is recommended due to risk of lung cancer and mesothelioma

Organic Dust (Byssinosis/Brown lung disease)

- Caused by inhalation of cotton, flax, or hemp dust.
- Not immune-related, no sensitization is needed.
- Early stage: occasional chest tightness
- Late stage: regular chest tightness toward the end of the 1st day of the workweek “**Monday chest tightness**” and may slowly increase to include more days.
- Tt: Early on may focus on reversing obstructive disease with antihistamines and bronchodilators. Removal of causative agent.

Hypersensitivity Pneumonitis

- An immune-mediated granulomatous inflammatory reaction to **organic antigens** in the alveoli and in the respiratory bronchioles
- Also called: extrinsic allergic alveolitis
- Dx and etiology is often in the history



HRCT in Acute HP

Examples of EAA

- **Farmer's lung**
- **Saw mill worker's lung**
- **Bird fancier's lung**
- **Mushroom worker's lung**
- **Malt worker's lung**
- **Humidifier lung**

- **Cheese washer's lung**
- **Suberosis**

- **Diisocyanate lung**
- **Hard metal worker's lung**

Etiology

mouldy hay
mouldy wood dust
proteins in bird droppings
spores, moulds
mouldy malt
contaminated humidifier water
Penicillium casei
cork dust mould

polyurethane hardeners
hard metal dust, cobalt

Symptoms

- flu-like illness
- cough
- high fever, chills
- dyspnea, chest tightness
- malaise, myalgia

4-8 hours after exposure

- Chronic disease: dyspnea in strain, sputum production, fatigue, anorexia, weight loss

Acute: Hypersensitivity Pneumonitis

- may have febrile illness, tachypnea, cough and chest tightness 3-8 hours after exposure.
- Transient hypoxemia and leukocytosis may occur. Hypoxemia may be severe if persons inhale large quantities of antigen.
- CXR may show small nodular opacities or patchy infiltrates.
- Symptoms typically peak –24 hours after onset and resolve in 1-3 days.

Chronic Hypersensitivity Pneumonitis

- Can consist of constitutional symptoms such as **wt loss, fever and fatigue.**
- Radiographic findings more c/w with typical interstitial fibrosis – dyspnea, bilateral crackles, cxr with reticulonodular opacities and honeycombing, poor response to steroids.
- *Of note: eosinophilia is NOT characteristic of hypersensitivity pneumonitis.*

EAA, clinical findings

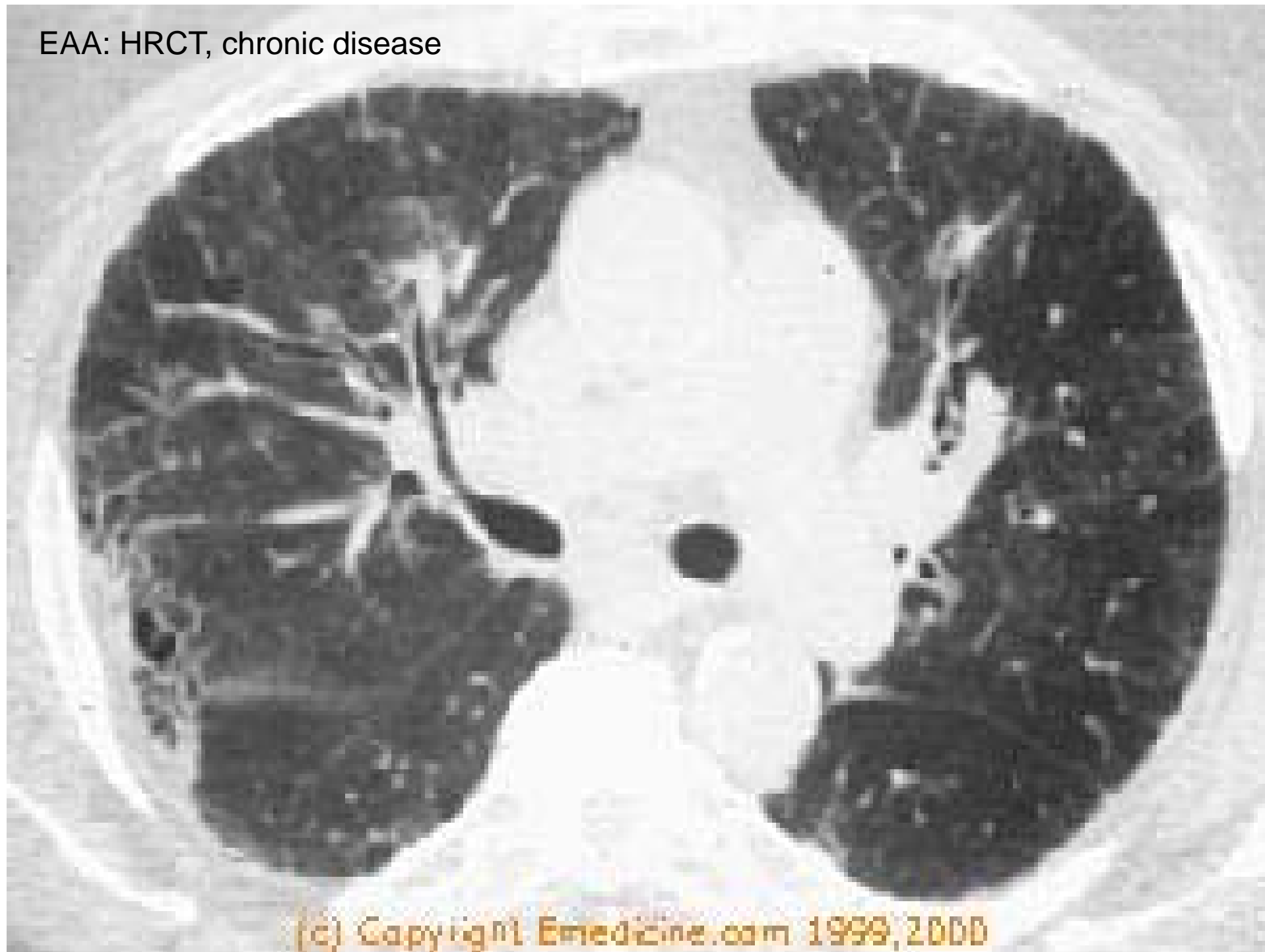
- **Status** **dyspnea, cyanosis,
crepitant rales
digital glubbing (chronic form)**
- **Chest X-ray** **normal or small nodules/diffuse infiltrates/
ground glass appearance
chronic form: pulmonary fibrosis**
- **HRCT** **normal or ground glass appearance
centrilobular micronodules**
- **lung function** **restriction, diffusing capacity decreases,
hypoxemia, obstruction, hyperreactivity**
- **lab. tests** **rise of sedimentation rate,
leukocytosis, neutrophilia**
- **BAL** **marked lymphocytosis,
T helper / T supressor cells decreased**

EAA: HRCT, acute disease



(c) Copyright Emedicine.com 1999, 2000

EAA: HRCT, chronic disease



[c] Copyright Emedicine.com 1999, 2000

Diagnosis

Main criteria

1. Exposure to arganic dust (history, spesific IgG antibodies, work place measurements).
2. Typical symptoms
3. Chest X-ray findings

Additional criteria

1. Decreased diffusion capacity
2. Hypoxia during rest or decreasing during excercise
3. Restriction in spirometric values
4. Lung biopsy with findings of allergic alveolitis
5. Provocation test (at work place) positive

All main criteria and two of the additional ones are needed for diagnosis.

Treatment

- Treatment: Remove the patient from the offending antigen.
- Short course Corticosteroids may be of help in acute disease.