**Occupational Lung Disease**

**Silicosis** – inhalation of crystalline silica in quartz, granite, sandstone – the most widespread pneumoconiosis in the US – occupations include mining, quarrying, drilling, sandblasting

- **Simple Silicosis** – upper lobe, small rounded opacities, centrilobular and perilymphatic nodules
- **Complicated Silicosis** – confluence of silicotic nodules into large opacities – this evolves into progressive massive fibrosis – “angel wing” appearance
  - 10% of silicosis will have UIP fibrosis
  - Hilar and mediastinal adenopathy is common - Calcium salt deposits lead to eggshell calcification
- **Tuberculosis** is found in 25% due to toxic effect of silica on alveolar macrophages
- **Acute silicosis** – usually in sandblasters – looks like alveolar proteinosis on imaging with ground glass and crazy paving
- **Erasmus syndrome** – association of occupational silica exposure with development of scleroderma

**Coal worker’s pneumoconiosis** – silica free coal dust particles that reach the alveoli – looks similar to silicosis but the pathology is distinct.

- Upper lobe small, rounded, opacities, that can develop into progressive massive fibrosis
- 10-40% with UIP fibrosis and honeycombing – associated with bronchogenic carcinoma
- **Caplan syndrome** – coal worker pneumoconiosis with rheumatoid arthritis

**Talcosis** – exposure to ceramic, paper, plastics, rubber, paint, cosmetics

- Similar appearance to silicosis – upper lobe opacities, bases are spared
- Often contaminated with amphibole asbestos fibers – will get pleural plaques
- Don’t confuse with **talc granulomatosis** which is from crushing and injecting pills

**Hard Metal Pneumoconiosis** – produced by compacting tungsten carbide with cobalt called sintering.

- Manifested by interstitial fibrosis and asthma, sometimes obliterative bronchiolitis
- Pathology is alveolar macrophages and multinucleated giant cells – **giant cell pneumonia**

**Asbestos-related diseases** – primary exposure in mining and milling, secondary exposure with insulation, brakes, shipbuilding, construction, textiles

- **Pleural plaques** – most common manifestation, usually on parietal and diaphragmatic pleura; rarely associated with restrictive physiology
- **Benign asbestos pleural effusion** – 10-12 years after exposure, exudative and unilateral
- **Diffuse pleural thickening** – starts unilateral in the base and progresses to bilateral; can lead to pleurogenic fibrosis or retractile pleuritic with restrictive physiology
- **Malignant mesothelioma** – latent period of 30-45 years; pleural thickening > 1cm
- **Asbestosis** – lower lobe fibrosis, think septal lines; seen in up to 1/3 of exposed patients
- **Bronchogenic carcinoma** – usually lower lobe adenocarcinoma, also consider rounded atelectasis

**Berylliosis** – granulomatous lung disease; beryllium is a lightweight metal used in dental, computer, nuclear weapons, nuclear reactor, and aerospace industries

- **Imaging:** early is ground glass, later is perilymphatic small nodules, mediastinal adenopathy in 25%
- **Pathology** looks like sarcoidosis with noncaseating granuloma
- **Acute berylliosis** looks like noncardiogenic pulmonary edema

**Hypersensitivity pneumonitis** – inhalation and sensitization to agricultural dusts, bioaerosols, microorganisms, or reactive chemicals

- 34% birds; 21% hot tub (mycobacteria); 11% farmer lung (actinomycetes); 25% no cause identified
- Imaging hallmark is **centrilobular** ground glass nodules; mosaic attenuation, bases usually spared
- Chronic hypersensitivity – fibrosis tends to start lower and progress to upper regions, endstage is severe upper lobe scarring and distortion.

**Noxious Fumes** – nitric acid, sulfur dioxide, chlorine gas, ammonia, phosgene

- Acute lung injury, noncardiogenic pulmonary edema, asthma, delayed-onset obliterative bronchiolitis