Occupational Lung Disease

SS Visser
Internal Medicine
UP
Classification

- Anorganic (mineral) dust/Pneumoconiosis
  Fibrogenic - silica, asbestos, talc, silicates
  Non-fibrogenic - Fe, barium, tin
- Immunologic/Pharmacologic
  Allergic alveolitis (hypersensitivity pneumonitis)
  Asthma - feathers, enzymes, cotton, platinum
- Irritant gases, vapours, smoke: high levels → pulmonary oedema, low levels → industrial bronchitis
- Respiratory carcinogens: asbestos, bis-chloro-methylether
Silicosis

Professions associated with exposure:

• Mining - ore of Au, Fe, Pb, Zn, Cu, rock
• Production - rock & concrete works, abrasives, pottery, isolation material
• Construction - tunnels, roads and train tracks, boiler makers, restoration of old buildings.
Prevalence and risks

- Prevalence: 22/1000 miners (1917-20) to <8/1000 miners currently
- Risk: 24 years vs 36 years exposure to:
  low dust levels: 5% 10%
  high dust levels: 15% 40%
Pathology

Macroscopic:

- hard gray-black nodules upper lobes and perihilar
- Massive fibrosis - large firm masses, shrunken upper lobes, emphysematous lower lobes and subpleural blebs
- PMF (progressive massive fibrosis): upper mid and lower lobes (accelerated silicosis)
- Cavitation (ischaemic necrosis) → secondary Tb → silicotuberculosis
Pathology

Microscopic: silicotic nodule

- Central zone: hialine connective tissue in concentric layers - acellular, no capillaries, varying silica content, occasional ischaemia
- Middle zone: cellular connective tissue
- Peripheral zone: halo of macrophages projecting into parenchyma, high silica content
- Located around respiratory bronchioli, blood vessels, pleural surfaces, interlobular septae
Clinical manifestations

- Classic / Uncomplicated silicosis
- Chronic silicosis
- Accelerated silicosis
- Acute silicosis
- Silicoproteinosis
- Silico-tuberculosis
- PMF
- Complicated silicosis
Diagnosis

• History of exposure
• Radiology
• Eliminate potentially treatable diseases (Tb, sarcoidosis, Idiopathic pulmonary fibrosis)
• Lung biopsy
Diagnosis: Radiology

- XR Chest - small nodules, 1-10 mm upper lung zones, calcification
- Reticular veiling
- Hilar and mediastinal lymphadenopathy (egg shell calcification)
- Cavitation
- Pneumothorax
- Alveolar veiling
- Caplan syndrome
Diagnosis: Physiology

- Lung function: varies from normal to obstructive or restrictive or combination
- Diffusion decreased
- Hypoxaemia on exertion
Diagnosis: Serology

- Hypergammaglobulinemia
- RF
- ANF
- S-ACE
- Increased incidence of systemic sclerosis described in SA gold miners
Treatment

- Terminate exposure to prevent PMF
- Corticosteroids, pulmonary lavage, lung transplantation
- Treat complications: Tb, pneumothorax, COPD, Cor pulmonale, collagen vascular diseases.
Asbestos

- Serpentine asbestos: Chrysotile (white asbestos) – RSA, Russia, Canada
- Amphibole asbestos: Crocidolite (blue asbestos) – RSA Limpopo, Mpumulanga, Northern Cape
  Amosite (brown asbestos) - as above
  Anthophyllite – Finland, worldwide
  Actinolite – RSA, Taiwan
Asbestos fibers

- Fibers 10 –20 µm length are fibro- & oncogenic
- Asbestos body – rod shaped, 1-6 µm wide and 10 – 30 µm long, yellow-brown with pale center, may be clubbed at one or both ends, usually contains amphibole asbestos, is formed intracellularly in macrophages, Fe is deposited, 40% of fibres are transformed into asbestos bodies which are not fibrogenic or carcinogenic – marker of asbestos exposure, not of disease, Asbestos bodies are found in lung parechyma, pleura, sputum, pleural effusion, other organs
Asbestos related lung disease

• Non-malignant pleural disease: pleural thickening, pleural plaques, benign exsudative pleuritis, round atelectasis (pseudo tumor)
• Pleural disease is a marker of exposure, usually no lungfx impairment
• Pleural disease puts patient at risk for other asbestos related diseases – 10% get interstitial fibrosis within 10 years and 2x higher risk for further pleural disease
Asbestos related lung disease

- Asbestosis = parenchymal disease interstitial fibrosis
- Associated more with crocidolite
- Smokers more prone to disease and XRC interstitial infiltrates
- Smokers 2.6x greater risk to die of asbestosis
Asbestos related lung disease

- Clinical presentation: exertional dyspnea, late inspiratory creps, clubbing (60%)
- XRC: reticular veiling lower lobes, ground glass veiling, pleural changes, PMF in mixed exposure, rarely Caplan syndrome
- Lungfx: restrictive, diffusion↓, art hypoxemia, elastic recoil ↑
- Non-specific immunologic findings: ANF, RF, S-ACE elevation, ? HLAB27 association
Asbestos related lung disease

- Bronchogenic Ca: 5x higher incidence in non-smoking asbestos workers, 90x higher in smoking asbestos workers
- Prevalence for adeno ca
- Chrysotile highest risk bronchus Ca
- Crocidolite highest risk for mesothelioma
- Other neoplasms: larynx ca, GIT ca, breast ca, ovarian ca renal ca.
Asbestos related lung disease

- Diffuse malignant mesothelioma: 35-45 y after exposure, more with crocidolite, pleural plaques not a precursor
- 4 histologic patterns: epithelial, mesothelial, mixed, undifferentiated
- Presentation: Chest pain prominent, dyspnea, clubbing, pleural effusion
- XRC: effusion
- CT: pleural based lobular mass with chest wall and rib involvement
Coal workers pneumoconiosis
( = Anthracosis)

- Simple anthracosis – asymptomatic or slight productive cough, radiologic diagnosis after 15 y exposure,

- Complicated anthracosis – chronic bronchitis, small % get PMF, functional impairment, PHT and cor pulmonale, may develop PTb, associated auto-immune reactions and ↑ incidence of RA

- When exposure is terminated the simple type will not progress; PMF type will progress
Coal workers pneumoconiosis

- Radiology: nodular veiling upper lung zones, nodules > 1 cm indicative of PMF
- Lungfx: normal – simple type restrictive – complicated type in smokers – obstructive
- Prognosis: simple type – good complicated type – cardio-respiratory failure
Metals

Metal ore – mixed dust exposure in open spaces – progressive pneumoconiosis

Welding – fumes and respirable particles (< 1μm cs) in closed spaces – acute pulmonary disease = metal fume fever, influenza –like manifestations lasting 24 hours.

Acute Cadmium toxicity – pulmonary oedema, chemical pneumonitis, destructive emphysema with severe exposure
• Beryllium – inhaled, binds to protein and is distributed to liver, spleen and bone, may cause granulomatous ulcers of skin, perforation of nasal septum, upper airway irritation, bronchiolitis, pneumonia, pulmonary oedema, chronic disease after 5-10 y – granulomatous pneumonitis (similar to hypersensitivity pneumonitis)