Pneumoconioses

- Lung diseases caused by inhalation of dust, mostly at work (*pneumo* = lung; *conis* = dust in Greek).
- Diseases are also called ‘dust diseases’ or ‘occupational lung diseases’.
- Type of lung disease varies according to the nature of inhaled dust.
- Some dusts are inert, cause no reaction, no damage, others cause immunologic damage and predispose to tuberculosis or to neoplasia.
- Factors which determine the extent of damage caused by inhaled dusts are:
• 1. Size and shape of the particles;
• 2. Solubility and physicochemical composition;
• 3. Amount of dust retained in lungs;
• 4. Additional effect of other irritants such as tobacco smoke; and
• 5. Host factors such as efficiency of clearance mechanism and immune status of the host.
• Inhaled dust particles larger than 5 μm reach the terminal airways where they are ingested by alveolar macrophages.
• Most of these too are eliminated by expectoration but the remaining accumulate in alveolar tissue.
• Tissue response to inhaled dust may be one of the following three types:

• *Fibrous nodules e.g. in coal-workers’ pneumoconiosis and silicosis.*

• *Interstitial fibrosis e.g. in asbestosis.*

• *Hypersensitivity reaction e.g. in berylliosis*
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Coal-Workers’ Pneumoconiosis

- Lung disease resulting from inhalation of coal dust particles, in coal-miners engaged in handling soft bituminous coal for a number of years, often 20 to 30 years.

- **2 types:**
  - Milder form of the disease called *simple coal workers’ pneumoconiosis*
  - *Advanced form termed progressive massive fibrosis (complicated coal-miners’ pneumoconiosis)*

- *Anthracosis, is not a lung* disease in true sense, is common, benign and asymptomatic accumulation of carbon dust in lungs of most urban dwellers due to atmospheric pollution and cigarette smoke
Pathogenesis

- Predisposing factors:
  - 1. Older age of the miners.
  - 2. Severity of coal dust burden engulfed by macrophages.
  - 3. Prolonged exposure (20 to 30 years) to coal dust.
  - 4. Concomitant tuberculosis.
  - 5. Additional role of silica dust.

- Activation of alveolar macrophage plays the most significant role in the pathogenesis of progressive massive fibrosis by release of various mediators
• i) Free radicals which are reactive oxygen species which damage the lung parenchyma.

• ii) Chemotactic factors for various leucocytes (leukotrienes, TNF, IL-8 and IL-6) resulting in infiltration into pulmonary tissues by these inflammatory cells which on activation cause further damage.

• iii) Fibrogenic cytokines such as IL-1, TNF and platelet derived growth factor (PDGF) which stimulate healing by fibrosis due to proliferation of fibroblasts at the damaged tissue site.
Morphology

- Pathologic findings at autopsy of lungs in the major forms of coal-workers’ pneumoconiosis are of 3 types
- Simple coalworkers’ pneumoconiosis
- Progressive massive fibrosis
- Rheumatoid pneumoconiosis (Caplan’s syndrome)

**Simple coalworkers’ pneumoconiosis:**
- **G/A:**
  - Lung parenchyma shows small, black focal lesions, measuring less than 5 mm in diameter and evenly distributed throughout the lung called *coal macules*, *and if palpable are called nodules.*
• **M/E:**
  1. Coal macules are composed of aggregates of dust laden macrophages, present in the alveoli and in the bronchiolar and alveolar walls.
  2. There is some increase in the network of reticulin and collagen in the coal macules.
  3. Respiratory bronchioles and alveoli surrounding the macules are distended without significant destruction of the alveolar walls.
Progressive massive Fibrosis:

G/A:

• Besides coal macules and nodules of simple pneumoconiosis, there are larger, hard, black scattered areas measuring more than 2 cm in diameter and sometimes massive.

• Sometimes, these masses break down centrally due to ischaemic necrosis or due to tuberculosis forming cavities filled with black semifluid resembling India ink.

• Pleura and regional lymph nodes are also blackened and fibrotic.
- **M/E:**
  - 1. The fibrous lesions composed almost entirely of dense collagen and carbon pigment.
  - 2. Wall of respiratory bronchioles and pulmonary vessels included in the massive scars are thickened and their lumina obliterated.
  - 3. Scanty inflammatory infiltrate of lymphocytes and plasma cells around the areas of massive scars.
  - 4. Alveoli surrounding scars are markedly dilated.
• **Rheumatoid Pneumoconiosis (CAPLAN’S Syndrome):**
  • Development of rheumatoid arthritis in a few cases of coal-workers’ pneumoconiosis, silicosis or asbestosis is called rheumatoid pneumoconiosis or Caplan’s syndrome.
  • **G/A:** Lungs have rounded, firm nodules with central necrosis, cavitation or calcification.
  • **M/E:**
    • Lung lesions are modified rheumatoid nodules with central zone of dust-laden fibrinoid necrosis enclosed by palisading fibroblasts and mononuclear cells.
    • Lung lesions have immunological basis for their origin as there is + rheumatoid factor and antinuclear antibodies.