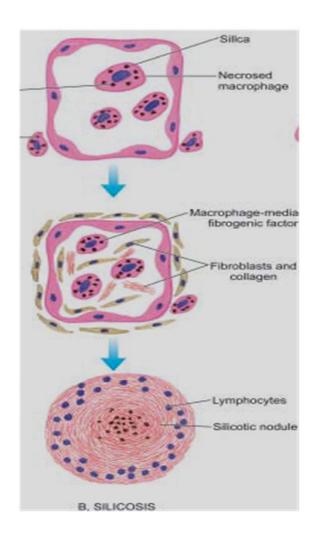
Silicosis

- Caused by prolonged inhalation of silicon dioxide, commonly called silica. Persons at increased risk:
- Miners (e.g. of granite, sandstone, slate, coal, gold, tin and copper), quarry workers, tunnellers, sandblasters, grinders, ceramic workers, foundry workers and those involved in the manufacture of abrasives containing silica.
- Peculiar to India are the occupational exposure to pencil, slate and agate-grinding industry carrying high risk of silicosis (agate = very hard stone containing silica)

Pathogenesis

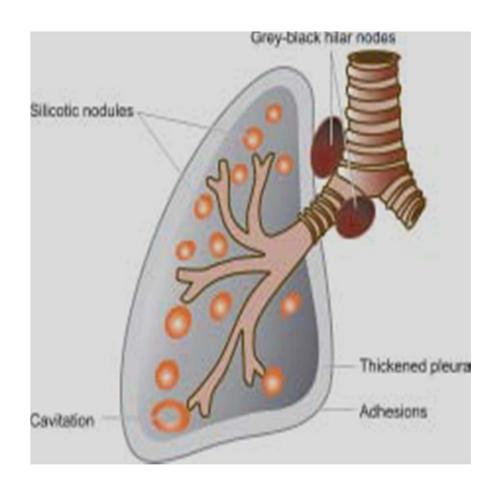
- Occurs after exposure for decades. Other factors, total dose, duration of exposure, the type of silica inhaled and individual host factors.
- Sequence of events:
- 1. Silica particles between 0.5 to 5 µm size on reaching alveoli taken by the macrophages which undergo necrosis.
- 2. Some silica-laden macrophages carried to respiratory bronchioles, alveoli and in interstitial tissue. Some silica dust transported to subpleural and interlobar lymphatics and into regional lymph nodes

- 3. Silica dust is *fibrogenic. Crystalline form, ie.* quartz, more fibrogenic than non-crystalline silica.
- 4. Activation of T and B lymphocytes, results in increased serum levels of immunoglobulins (IgG and IgM), antinuclear antibodies, rheumatoid factor and circulating immune complexes as well as proliferation of T cells.
- 5. Silica is *cytotoxic* and *kills* the macrophages which engulf it. Released silica dust activates viable macrophages leading to secretion of macrophage derived growth factors as interleukin-1 that favour fibroblast proliferation and collagen synthesis.



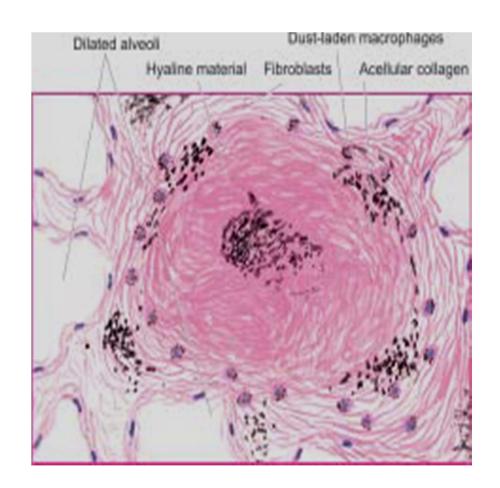
Morphology

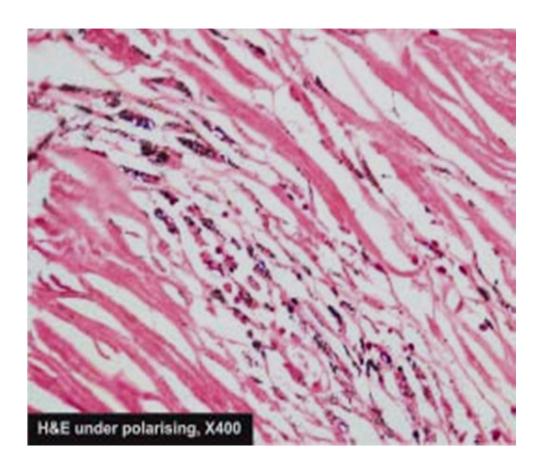
- G/A:
- lung studded with well-circumscribed, hard, fibrotic nodules, 1 to 5 mm in diameter
- Nodular lesions frequently have simultaneous deposition
- of coal-dust and may develop calcification.
- Pleura grossly thickened, adherent to chest wall.
- Nodular lesions detectable as egg-shell shadows in chest X-rays.
- Lesions may undergo ischaemic necrosis, develop cavitation, or complicated by tuberculosis and rheumatoid pneumoconiosis



• M/E:

- 1. Silicotic nodules have central hyalinised material with scanty cellularity and some amount of dust. Hyalinised centre surrounded by concentric laminations of collagen.
- 2. On polarisation demonstrate numerous birefringent particles of silica.
- 3. Severe and progressive form of disease result in coalescence of adjacent nodules and cause complicated silicosis.
- 4. Intervening lung parenchyma show hyperinflation or emphysema.
- 5. Cavitation, complicated by tuberculosis and rheumatoid pneumoconiosis





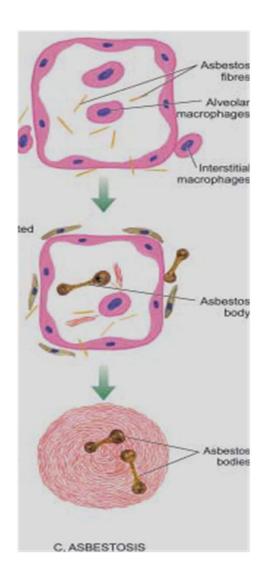
Asbestos disease

- Coal is lot of dust and little fibrosis, asbestos is little dust and a lot of fibrosis.
- Three types of severe diseases:
- Asbestosis of lungs,
- Pleural disease
- Tumours.
- In nature, asbestos exists as long thin fibrils which are fire-resistant and can be spun into yarns and fabrics suitable for thermal and electrical insulation and has many applications in industries

- Persons at risk are workers engaged in mining, fabrication and manufacture of a number of products from asbestos eg. Asbestos pipes, tiles, roofs, textiles, insulating boards, sewer and water conduits, brake lining, clutch castings.
- Two major geometric forms of asbestos:
- **Serpentine** consisting of curly and flexible fibres is most common chemical form *chrysotile* (white asbestos) comprising more than 90% of commercially used asbestos.
- Amphibole consists of straight, stiff and rigid fibres and include less common chemical forms crocidolite (blue asbestos), amosite (brown asbestos), tremolite, anthophyllite and actinolyte. associated with induction of malignant pleural tumours, with crocidolite.

Pathogenesis

- 1. Inhaled asbestos fibres phagocytosed by alveolar macrophages from where they reach the interstitium.
- 2. Asbestos-laden macrophages release chemo-attractants for neutrophils and for more macrophages, inciting cellular reaction around them.
- 3. Asbestos fibres coated with glycoprotein and endogenous haemosiderin; beaded or dumbbell-shaped asbestos bodies.
- 4. All types of asbestos, fibrogenic result in interstitial fibrosis.
- 5. Few *immunological abnormalities as ANA* and rheumatoid factor have been found, their role in the genesis of disease is not clear.
- 6. Asbestos fibres are carcinogenic cause mesothelioma



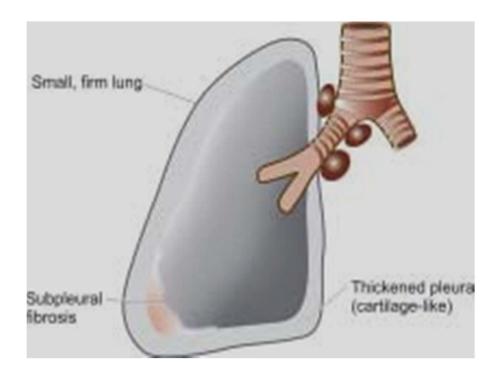
Morphology

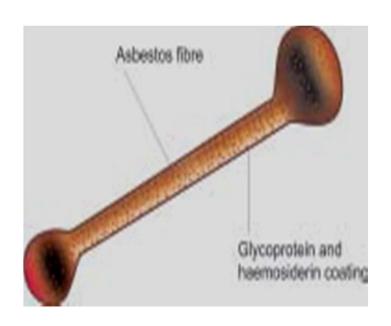
• G/A:

 Lungs small and firm with cartilage-like thickening of the pleura. C/S: Variable degree of pulmonary fibrosis, in subpleural areas and in bases of lungs.

• M/E:

- 1. Non-specific interstitial fibrosis.
- 2. Presence of characteristic asbestos bodies in involved areas, coating stains positively for Prussian blue reaction.
- 3. May be changes of emphysema in pulmonary parenchyma between the areas of interstitial fibrosis.





Pleural Disease:

 3 types of lesions: Pleural effusion, Visceral pleural fibrosis, Pleural plaques

• G/A:

 Circumscribed, flat, small (upto 1 cm in diameter), firm or hard, bilateral nodules

• M/E:

 Hyalinised collagenous tissue may calcify so that they are visible on chest X-ray. Asbestos bodies generally not found within the plaques.

Tumors:

- Number of cancers, most importantly bronchogenic carcinoma and malignant mesothelioma, others are: carcinomas of oesophagus, stomach, colon, kidneys and larynx and various lymphoid malignancies.
- Bronchogenic carcinoma: Incidence is 5 times higher in non-smoker asbestos workers, 10 times higher in smoker asbestos workers.
- Malignant mesothelioma: Association with asbestos exposure is present in 30 to 80% of cases with mesothelioma