Silicosis and Silicotuberculosis

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Introduction

- Silicosis, major occupational lung disease
- Problem in both industrialized and developing countries
- Due to inhalation of crystalline silica, quartz
- Tuberculosis contributes significantly morbidity and mortality

Etiology

- Caused by inhalation of tiny particles of silicon quartz, silicon dioxide
- Workers at greatest risk, who blast rock and sand (miners, quarry workers, stonecutters)
- who use silica-containing rock or sand abrasives (sand blasters; glass makers; foundry, gemstone, and ceramic workers; potters).
- Coal miners are at risk of mixed silicosis and coal workers' pneumoconiosis

Physical and Chemical Properties

- *Description* Transparent crystals
- *Molecular formula* SiO_2
- *Molecular weight* 60.09 g/mol
- *Density* 2.65 g/cm3
- *Melting point* 1610 °C
- *Boiling point* 2230 °C
- *Solubility* Practically insoluble in water or acids, except hydrofluoric acid; very slightly sol. in alkali.

Factors that influence the likelihood of progression to silicosis

- Duration and intensity of exposure,
- Form of silicon (exposure to crystalline form poses greater risk than bound form),
- Surface characteristics (exposure to uncoated form poses greater risk than coated form),
- Rapidity of inhalation after the dust is fractured and becomes airborne
- The current limit for free silica in the industrial atmosphere is 100 µg/m3

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%

Forms of silicosis

- Chronic (or Classic) Silicosis
- Accelerated Silicosis
- Acute Silicosis

Chronic (or Classic) Silicosis

- Most common form of the disease
- Usually follows one or more decades
- Respirable dust containing < 30% quartz
- Pathological hallmark- silicotic nodule
- Usually bilateral upper zones, visceral pleura, regional lymph nodes

Accelerated silicosis

- Results from heavier exposures
- Duration of 5 to 10 years
- More cellular than fibrotic in nature
- More diffuse interstitial pulmonary fibrosis
- Develop superimposed mycobacterial infection
- Scleroderma more frequent in this stage

Acute silicosis (Silicoproteinosis)

- Follows intense exposure to fine dust of high silica content
- Develops within a few months up to 5 years
- Shows all the features of PAP
- Rapid progression to severe HRF
- Radiographic finding –diffuse alveolar filling, lower lung zone

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

Simple silicosis

- **Widespread nodules measuring 2-5 mm in diameter, with a predominance in the middle and upper lung zones.**

Radiology

- **R/N opacities in the middle and upper lung zone.**
- **Large, round opacities on the right; conglomerate nodules.**
- **Eggshell calcification of the mediastinal lymph nodes**

Silicosis with progressive massive fibrosis

- **Large, conglomerate nodules in both the middle and upper lung zones**
- **Periphera hyperlucency lung tissue secondary to central migration of the large nodules**
- **Evidence of volume loss in both upper lobes**

Compilcated silicosis

- Conglomerate masses are 1 cm. to 10 cm. in diameter
- There is associated cicatrization atelectasis of the upper lobes, hilar retraction,
- Bibasilar hyperexpansion and emphysema. The masses may undergo ischemic necrosis and cavitation.

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

Diseases associated with exposure to Silica dust

- Chronic obstructive pulmonary disease
  - Emphysema
  - Chronic bronchitis
  - Mineral dust- induced small airway disease
- Lung cancer
- Mycobacterial infection
  - MTB
  - NTM
- Immune –Related Disease
  - PSS, RA, CRD, SLE

Complications

- Cor pulmonale
- Spontaneous pneumothorax
- Broncholithiasis
- Tracheobronchial obstruction
- Lung cancer
- Tuberculosis
- Hypoxemic ventilatory failure

Treatment

- Prevent further exposure to silica dust.
- Strongly advise patients to quit smoking
- Immunize against influenza, pneumococci
- No specific therapy for silicosis
- Experimental approaches are - whole-lung lavage, aluminum inhalation, and corticosteroids
- Latent tuberculosis infection should be treated with isoniazid
- Complications should be treated appropriately

Prevention

- Dust suppression,
- Process isolation,
- Ventilation,
- Use of non–silica–containing abrasives.
- Respiratory masks
- Surveillance of exposed workers with respiratory questionnaires, spirometry, and chest x-rays is recommended.

Chest X-ray Schedule

Duration	Age	X-ray schedule
<10 years	All age	Every 5 years
>10 years	<30 years	Every 5 years
>10 years	35-44 years	Every 2 years
>10 years	>45 years	Every year

(Donaldson k et al. Ann Occ Hyg 1998;42)

SILICOTUBERCULOSIS

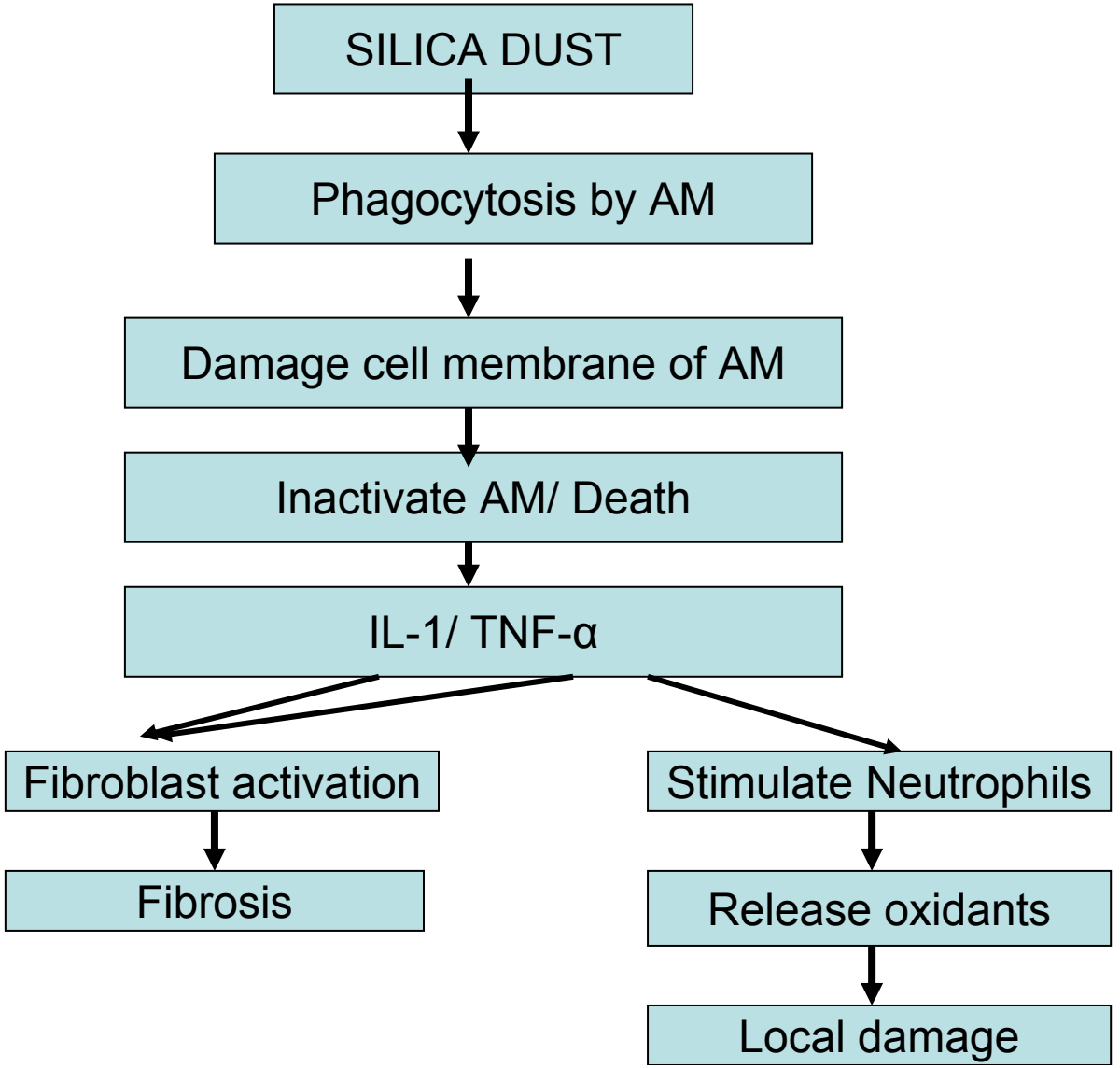
Introduction

- The association of Silicosis and TB has been suspected several hundred years
- In 1902 JS Holdene committee reported that “Stone dust predisposes enormously to TB in the lung”
- Exposure to silica causes a renewed multiplication of bacilli in the healing TB lesions

Data available on Silicotuberculosis

- In autopsy material – over 25 %
  (Gooding CG at al Lancet, 2:891,1946)
- Bacteriological evidence- 12.9%
  (Theodas PA Am Re Tuber:65,24 1952)
- Hong kong chest service -27%
  (Am Rev Respir Dis -1992;145:36-41)
- In India silicotuberculosis incidence - 28.6%
  (Sikand BK,Pamra SP)
- Tuberculosis is 3 to 7 times higher in person with silicosis
  (Gupta SP et al. India J Med Res 1972; 60:1909-15)

Pathogenesis



Iron hypothesis

- Mycobacteria are dependent on iron for growth and produce the iron chelators mucobactin
- Silica particles absorbed body iron and act as a reservoir of iron
- Silicoto-iron complexes may activate dormant tubercle bacilli

Influence of TB

- Exposure of silica has an unfavourable influence on the course of induced TB
- There is more fibrosis is produced by combination
- Synergistic effect of silicosis and TB – proliferative fibrous reaction
- TB may complicate simple silicosis as well as advanced disease
- It may develop massive fibrosis

Diagnostic problem

- Symptoms of silicosis and silicoTB are misleading
- Interpretation of the Chest X ray flim of the silicotic is difficult

Diagnosis

- High degree of suspicion
- Radiographic abnormalities in the apical area of either lungs
- Poorly demarcated infiltrates of variable size that do not cross the lung fissures
- Opacities may surround pre-exiting silicotic nodules
- Presence of a cavity in a nodule

Diagnosis

- Frequent sputum examination for AFB
- Mycobacterial culture where high prevalence of atypical mycobacteria
- For early and accurate diagnosis

  FOB, BAL, TBLB
- Therapeutic trial of ATT

Immunodiagnosis

- ↑ levels of total IgE and IgG
- ↑ Fibronectin
- ↑ CD4+ and CD20+ markers
- ↓Concentration of the mucinic antigen 3EG5

The use of a complex of immunological studies promoted the better early diagnosis of silicotuberculosis.

Diagnosis

- Sputum culture for Mycobacterium tuberculosis L forms is a convenient and rapid way to detection of Mycobacterium tuberculosis

- Detection rate of Mycobacterium tuberculosis L forms significantly increases with deterioration of silicosis.

  (Zhonghua Jie He He Hu Xi Za Zhi. 2001 Apr;24(4):236-8.)

Radiology

- Rapidly developing soft nodulation
- Conglomeate massive shadowing
- Evidence of cavitations

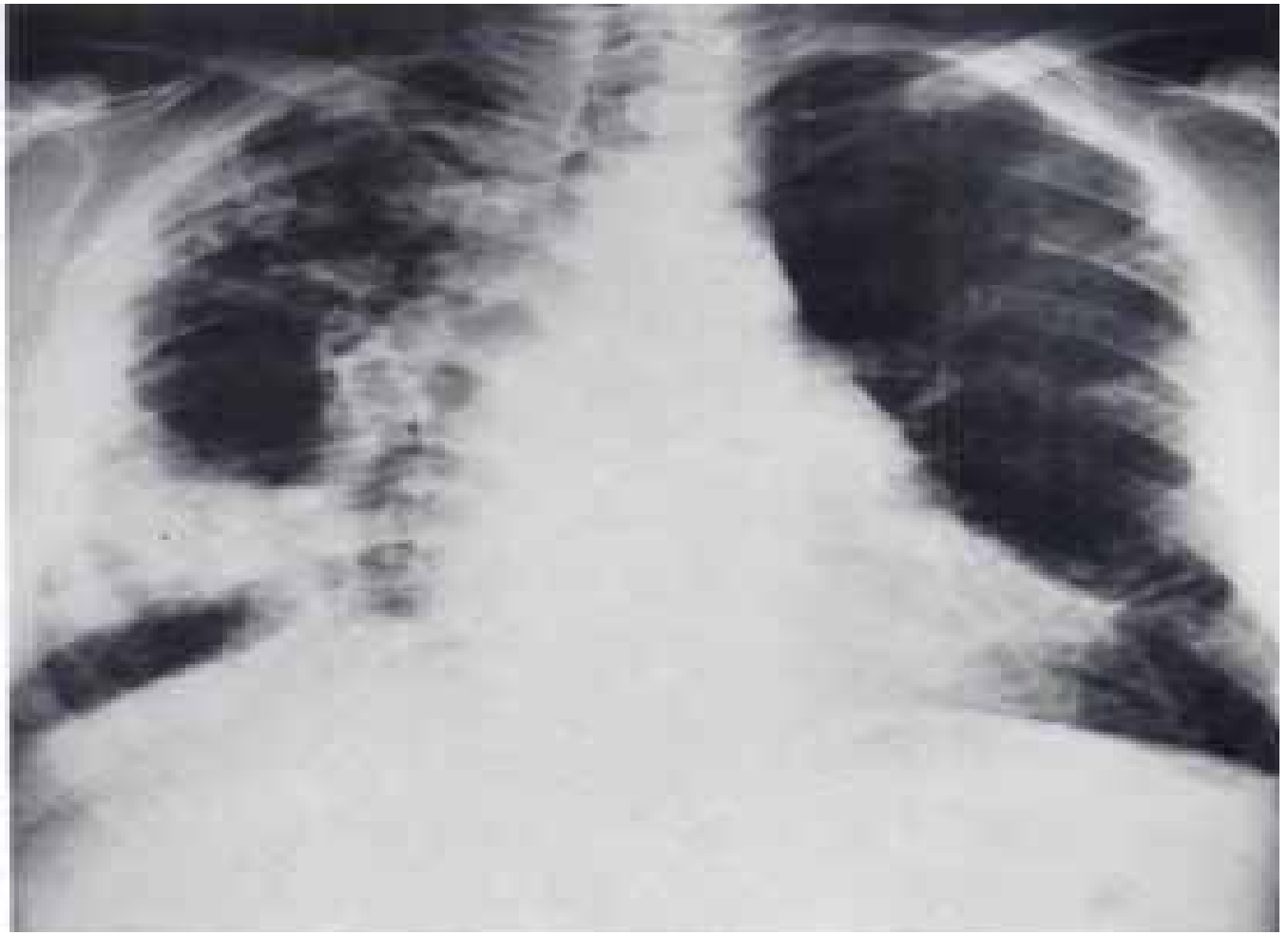
(Pancoast HK Am J Roen 14, 381;1925)

Additional finding

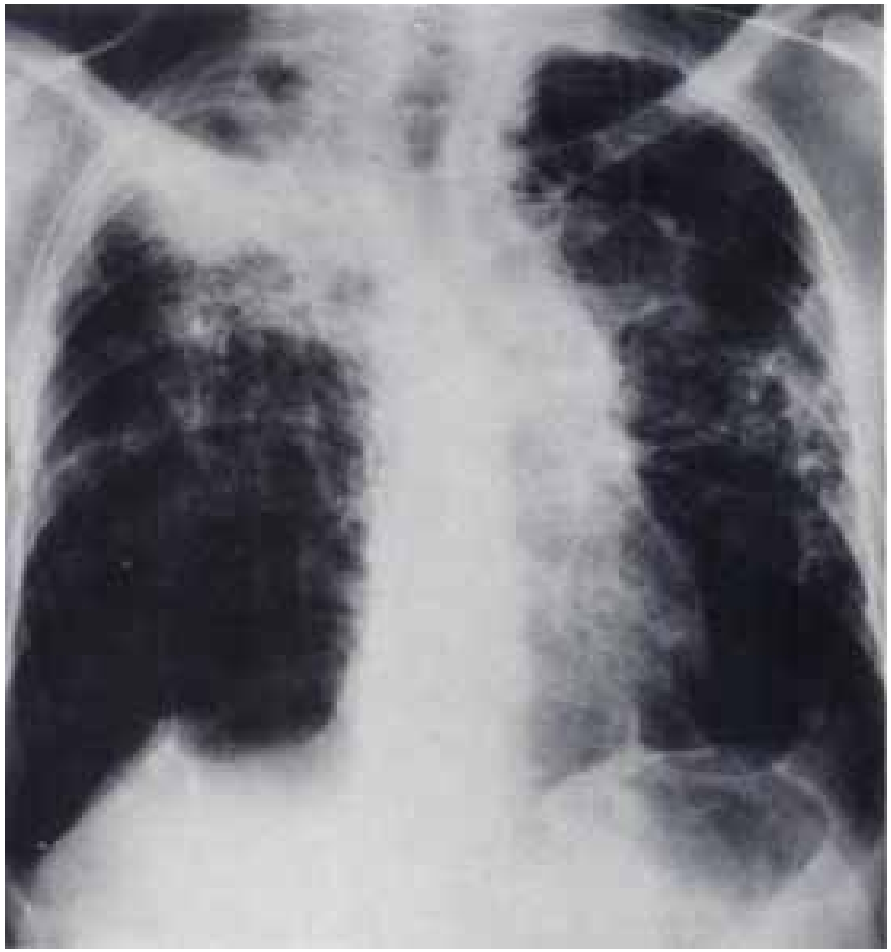
- Rapid changes in the radiographic picture
- Development of pericardial or pleural effusion
- Bronchial stenosis especially right middle lobe

(Barras G Schweiz Med. Wochenschr 1970;100:1802-8)

chest x-ray film showing conglomerate silicosis and tuberculous cavity in right middle pulmonary field.



Chest x-ray film showing conglomerate silicosis, tuberculosis, and bullae at both bases



Selection of patients for treatment

- History of exposure to silica.
- X-ray film suggestive of actual silicotuberculosis.
- Serial x-ray film evidence of progression of disease.
- Positive tuberculin test.
- Other evidence of activity, such as hemoptysis, silicosis, pleural effusion, or fever, elevated sedimentation rate,

Silicotuberculosis the results are not as good

- Silicotuberculosis affects not only the parenchyma but also the arteries and the veins.
- There is thickenings of the intima, hyaline and lipoid degenerations, scars in the vessels, impeding the blood circulation.
- Moreover, tuberculous cavities often occur inside silicotic nodes, which can hardly be reached by chemotherapeutic drugs.
- Fibrotic scars can prevent the collapse and scarification of a cavities

Treatment

- SCCT has been established in patients with silicotuberculosis
- Prolongation of the continuation phase from 4 to 6 months decreased the rate of relapse from 22 to 7%

  (Blumberg et al. Am J Resp & crit care Med Febb 15- 2003)
- Presently, a closely supervised eight to nine months treatment is recommended

Silicotuberculosis: long-term outcome after short-course chemotherapy

		Silicosis (186)	No silicosis (403)
Age (SD) years	*	40.7 (9.34)	35.5 (8.80)
Environmental mycobacteria	NS	15 (8%)	17 (4%)
Failed initial Rx	NS	2	3
Relapses	*	29 (17%)	43 (11%)
Time to relapse (SD) years	NS	2.6 (1.89)	3.1 (2.23)
Non-relapses still in mine		93 (66%)	222 (65%)
Service at 5 years	NS		

NS: no significant difference.
*$P < 0.05$.

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Treatment and sputum conversion

Treatment group	*Number*	*Sputum remaining positive*	*Sputum conversion*
1. 24 months or longer...	15	1	14
2. 12-24 months.....	6	0	6
3. Less than 12 months, or interrupted treatment.	8	3	5
4. No treatment.....	1	0	1
Total.....	30	4	26 (86.7%)

Tubercle and Lung Disease (I 995) 76, 3942

Relationship of x-ray changes to treatment

Treatment group	*Improvement*	*Appearance stationary*	*Deterioration*
1. 24 months or longer . . .	7	34	2
2. 12-24 months	2	20	1
3. Less than 12 months, or interrupted treatment.	0	35	5
4. No treatment	0	27	4
Total	9	116	12

Tubercle and Lung Disease (I 995) 76, 3942

Prevention

- Active surveillance of the workers in both pre-employment and post-employment periods

  Periodic CXR

  Tuberculin test

- Engineering measures to reduce or eliminate the exposure to silica dust