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## Review

### Overview of Silicosis



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	<b>Abstract</b>
Published on: 05 Dec 2023	<p>Silicosis/<i>Pneumonoultramicroscopicsilicovolcanoconiosis</i> is a most common <i>pneumo-coniosis</i>. Silicosis is an irreversible fibrotic lung disease which is a form of occupational pulmonary disease caused by the inhalation and deposition of crystalline silicon dioxide (SiO<sub>2</sub>) in the bronchi, lymph node and/or lung Parenchyma as a result of exposure during mining, stone crushing and quarrying activities which occurs mostly in developing countries. Highly populated countries are exposed and the USA, Europe, China and India are listed in the top among the countries. However, the number is still too low because of parallel number of undiagnosed cases. Silicosis induces a series of Inflammatory reactions that further trigger a chronic or acute fibrotic change in the lungs. Silicosis is associated with other comorbidities among which TB, COPD and Cancer are the most prevalent. Till now, there is no curative treatment however some management strategies may help to recover quality of shelf life and slow deterioration.</p>
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<p><b>Keywords:</b> Occupational pulmonary disease, Crystalline silica, Silicosis, Silico-tuberculosis, Lung Cancer, Stone crushing, Quarrying</p>	

## INTRODUCTION

Silicosis/*Pneumonoultramicroscopicsilicovolcanoconiosis* (*Pneumo-coniosis*) is one of the oldest occupational disease. It is an irreversible fibrotic lung disease caused by the Inhalation and deposition of silica dust [1,2] in the bronchi, lymph node and/or pulmonary parenchyma [3]. Silicosis is also known as Miner's phthisis, Quarry grinder's asthma, Potter's rot [4], or by the invented name "Pneumono ultra microscopic silico volcanoconiosis" [5,6].

Ramazzini et al in 1713 found sand- like substances in the lungs of stone cutters. In 1860s, Peacock and Greenhow noted silica dust in the lungs of miners. In 1870s Achille Visconti, coined the term "silicosis" [7]. In India, silicosis was first reported by Subba Rao, Senior surgeon, Mysore Government in 1934 [8].

**SILICA (SiO<sub>2</sub>)**

- ❖ Silica exists in both crystalline and amorphous forms, the later having lesser toxicity[9]
- ❖ Quartz is the common form of crystalline silica the other types include cristobalite and tridymite. Respirable Crystalline Silica with a diameter less than 10 micron is easily trapped in airways[10]. Particle size of 1µm [ranged 0.5-3µm] are the most fibrogenic [11].

**SOURCE OF EXPOSURE TO RESPIRABLE CRYSTALLINE SILICA (RCS)**

The National Institute of Occupational Health (NIOH) reports the following industries where personal exposure to silica or prevalence of silicosis is very high:

- ✓ Construction work: sand blasting, crushing, drilling, masonry, tunneling and grinding.
- ✓ Mining, sand stone and granite drilling
- ✓ Foundry work
- ✓ Manufacturing of ceramic and clay pottery
- ✓ Agate cutting and polishing
- ✓ Glass manufacturing
- ✓ Slate pencil work [12].



**NORMAL LUNGS      SILICOSIS AFFECTED LUNGS**

**TYPES OF SILICOSIS**

**i) Chronic silicosis:** It is the most common form of silicosis, usually develops after the period of 15-20 years of exposure to relatively low dust concentrations.

**ii) Accelerated Silicosis:** It results from exposure to high concentrations of silica over a period of 5-10 years.

**iii) Acute silicosis:** It is rare, but highly fatal disease caused by brief but massive exposure to dust with high silica content, can occurs after a few months to as long as 2 years [7].

The development of silicosis depends on a number of factors including:

- Amount and variety of dust inhaled
- Percentage of free silica in the dust
- Form of silica
- Size of silica particles
- Duration of exposure
- Individuals natural body resistance
- Presence or absence of complicating factors (such as infection and other co- morbidities) [11].

**EPIDEMIOLOGY****GLOBAL**

→ According to NIOSH (National Institute of Occupational Safety and Health) report, death from silicosis declined from 1200 in 1968 to fewer than 100 in the early 2000s [13], the information was based on death certificate data and did not count morbidity cases due to silicosis. However, Rosenman et al. [2003] estimated about 3600-7300 cases occurred annually from 1987 to 1996 in the US [14]

→ In UK, death from silicosis declined from 28 in 1993 to 10 in 2008 [15,16]

→ In China, more than 74,000 deaths reported annually [10].

**INDIA**

→ C. Krishnaswamirao (1934) first reported the occurrence of silicosis in India from Kolar gold fields.

→ According to ICMR, 1999 report there are about 30 lakhs workers in India who are at a high risk of exposure to silica. Out of these, 17 lakhs are in mining/quarrying workers, 6.3 lakhs in glass and mica industry and 6.7 lakhs in metal industry [7].

→

## **PATHOPHYSIOLOGY**

Silica- induced lung damage occurs by five mechanisms:

**DIRECT CYTOTOXICITY:** The silica particles react with resident cells and cause lipid peroxidation of the membrane in bronchoalveolar cells[17].

**GENERATION OF REACTIVE SPECIES:** Silica particles generates Reactive Oxygen Species (ROS) and Reactive Nitrogen Species (RNS). It activates cell signaling pathways, phophorylate and activate specific transcription factors (e.g.NF $\kappa$ B), which mediates various downstream cascade mechanisms leading to inflammation and tissue damage [18].

**PRODUCTION OF CYTOKINES AND CHEMOKINES:** ROS and RNS increase expression of cytokines. The release of cytokines, chemokines, lipid mediators and growth factors recruits polymorphonuclear and mononuclear cells to the alveolar spaces and around the silica particles which contribute for the formation of granulomas [17].

**FIBROSIS:** Once fibroblasts were recruited to the damage site, the transforming growth factor (TGF)- $\beta$  induces collagen deposition as well as increased elastin production [17].

**CELL DEATH BY APOPTOSIS:** The process of apoptosis is a result of mitochondrial dysfunction and increased expression of death receptors and their ligands, such as FasL and TNF. Macrophages undergoing apoptosis also release silica particles back to the lung parenchyma, where they are phagocytosed again by other macrophages, perpetuating the tissue damage cycle[17].

## **COMORBIDITIES AND COMPLICATIONS**

### **TUBERCULOSIS (SILICO-TUBERCULOSIS)**

There is an 8 to 20 fold increased risk of mycobacterial infections in patients with silicosis[19]. The potentiation of growth of tubercle bacilli by silica is probably due to impaired response of macrophages to intracellular mycobacterium tuberculosis altered in the presence of silica[20].

### **CANCER**

The International Agency on Cancer (IARC) declared crystalline silica as group 1 carcinogen to human[18]. According to the American College of Occupational and Environmental Medicine (ACOEM), the risk for lung cancer in silicotic patients tends to be greatest in workers with silicosis who smoke[19].

### **AUTOIMMUNE DISORDERS**

Silicosis is associated with autoimmune disorders such as Systemic Lupus Erythematosus (SLE), Systemic Sclerosis(SS) and Rheumatoid Arthritis (RA). SLE and RA are more common in men with silicosis than in the general population[19].

### **FUNGAL INFECTION (SILICO-MYCOSIS)**

Silicotic lungs are susceptible to various fungal infections. Chronic necrotizing pulmonary aspergillosis with aspergilloma has previously been identified as an atypical complication in a 52- year old man with a history of long lasting silica exposure[18].

## **SIGNS AND SYMPTOMS**

- ✓ Cough
- ✓ Dyspnea
- ✓ Tachypnea
- ✓ Fatigue
- ✓ Loss of appetite and weight loss
- ✓ Fever
- ✓ Chest pain
- ✓ Cyanosis (Blue skin)

**In advanced cases, the following may also occur :**

- ✓ Respiratory insufficiency
- ✓ Corpulmonale [5].

## **DIAGNOSIS**

### **CHEST RADIOGRAPHY**

The chest X-ray shows bilateral upper zone predominant reticulo nodular opacities, with hilar lymphadenopathy with or without eggshell calcification. Conglomerated nodules can appear as mass like lesion. Emphysematous bulla formation.

### **COMPUTED TOMOGRAPHY (CT) AND HIGH RESOLUTION COMPUTED TOMOGRAPHY (HRCT) SCANNING**

Useful in identifying large confluent lesions that a chest radiograph may miss.

### **HISTOPATHOLOGY**

The early lesions can be visualized by nodular to stellate aggregates of dust-filled macrophages assembled around a collagenous central region. With time, the central collagen becomes distinctly whorled with decreased number of inflammatory cells around the periphery [18].

### **PREVENTION**

**OSHA and NIOSH provide guidelines for safe practices in the workplace.**

- ✓ Using wet methods over dry methods at a site where silica dust is present.
- ✓ Using blasting cabinets and other methods to control dust
- ✓ Providing optimal ventilation methods.
- ✓ Only permitting potential exposure for very short periods.
- ✓ Giving workers additional respiratory protection, like special masks or respirators, (N95 Or better) if needed.
- ✓ Washing your hands & face before eating, drinking or smoking after exposure. [21]

### **TREATMENT**

Silicosis has no cure at present and is a permanent disease. Treatment options include alleviating the symptoms and preventing progress of the condition further.

- To treat bacterial lung infection, antibiotics should be used.
- Use cough suppressants.
- Chest physiotherapy in patients to help the bronchial drainage of mucus.
- In patients with hypoxemia, Oxygen administration
- To facilitate breathing , use of bronchodilators .
- The most effective treatment is lung transplantation, to replace the damaged lung tissue but is risky from consequences of long-term immunosuppressant (e.g., Opportunistic infections).
- To treat acute silicosis, broncho-alveolar lavage should be carried out to alleviate symptoms, but does not decrease overall mortality [22].

## **CONCLUSION**

Silicosis is not a curable disease; therefore, control of this disease lies mainly in its prevention. Silicosis continues to pose a major occupational health hazard amongst workers engaged particularly in construction, mining and foundry works. Reduction of exposure through various preventive measures and regular screening for early detection are thus crucial for reducing the global burden of silicosis.

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