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A life threatening lung disease of Bundelkhand region- Silicosis

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ABSTRACT

Silica is the second most common element on the surface of the earth. It is found in sand, many rocks such as granite, sandstone, flint and slate, and in some coal and metallic ores. The cutting, breaking, crushing, drilling, grinding, or abrasive blasting of these materials may produce fine silica dust. Silicosis is a form of occupational lung disease caused by inhalation of crystalline silica dust, and is marked by inflammation and scarring in forms of nodular lesions in the upper lobes of the lungs. Silicosis is due to deposition of fine dust (less than 1 μ m in diameter) containing crystalline silicon dioxide in the form of alpha-quartz, cristobalite, or tridymite. Silicosis is characterized by shortness of breath, fever, and cyanosis. It may often be misdiagnosed as pulmonary edema (fluid in the lungs), pneumonia, or tuberculosis. Treatment options currently focus on alleviating the symptoms and preventing complication. So here an attempt has been made to attract the attention of doctors, researchers, NGO's to develop a proper medication and vaccine kit for the complete treatment of this disease.

Keywords: Silicosis, Lungs, Tuberculosis, NGO's

1. INTRODUCTION

Silicosis, known as Grinder's disease and Potter's rot, is a form of occupational lung disease caused by inhalation of crystalline silica dust, and is marked by inflammation and scarring in forms of nodular lesions in the upper lobes of the lungs^{1,2}.

Silica is the second most common element on the surface of the earth. It is found in sand, many rocks such as granite, sandstone, flint and slate, and in some coal and metallic ores. The cutting, breaking, crushing, drilling, grinding, or abrasive blasting of these materials may produce fine silica dust. It can also be in soil, mortar, plaster, and shingles. Silicosis is due to deposition of fine dust (less than 1 micrometer in diameter) containing crystalline silicon dioxide in the form of alpha-quartz, cristobalite, or tridymite. Silicosis is the most common occupational lung disease worldwide; it occurs everywhere but is especially common in developing countries^{3,4}.

2. EPIDEMIOLOGY

In India, a prevalence of 55% was found in one group of workers, many of them very young, engaged in the quarrying of shale sedimentary rock and subsequent work in small, poorly ventilated sheds. Studies on silicotic pencil workers in Central India demonstrated high mortality rates; the mean age at death was 35 years and the mean duration of the exposure was 12 years^{5,6}.

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During the period 1991 to 1995, China recorded more than 5, 00 000 cases of silicosis, with around 6,000 new cases and more than 24,000 deaths occurring each year mostly among older workers⁷.

3. TYPES OF SILICOSIS

Classification of silicosis is made according to the disease's severity, onset, and rapidity of progression^{8,9,10}. These include:

1. *Chronic silicosis* -occurs after 15–20 years of exposure to moderate to low levels of silica dust. Chronic silicosis itself is further subdivided into simple and complicated silicoses. This is the most common type of silicosis. Patients with this type of silicosis may not have obvious symptoms, so a chest X-ray is necessary to determine if there is lung damage.
2. *Asymptomatic silicosis* - Early cases of the disease do not present any symptoms.
3. *Accelerated silicosis* -Silicosis that develops 5–10 years after high exposure to silica dust. Symptoms include severe shortness of breath, weakness, and weight loss.
4. *Acute silicosis* - Silicosis that develops a few months to 2 years after exposure to very high concentrations of silica dust. Symptoms of acute silicosis include severe disabling shortness of breath, weakness, and weight loss, often leading to death.

Symptoms

Silicosis (especially the acute form) is characterized by shortness of breath, fever, and cyanosis (bluish skin). It may often be misdiagnosed as pulmonary edema (fluid in the lungs), pneumonia, or tuberculosis^{11,12,13}.

- Dyspnea exacerbated by exertion
- Dry or severe cough, often persistent and accompanied by hoarseness of the throat
- Fatigue
- Tachypnea (rapid breathing) which is often labored
- Loss of appetite
- Chest pain
- Fever
- Gradual dark shallow rifts in nails eventually leading to cracks as protein fibers within nail beds are destroyed.

In advanced cases, the following may also occur:

- Cyanosis
- Cor pulmonale
- Respiratory insufficiency
- Silicotuberculosis- Patients with silicosis are particularly susceptible to tuberculosis infection known as silicotuberculosis. The reason for the increased risk 10–30 fold increased incidence is not well understood. It is thought that silica damages pulmonary macrophages, inhibiting their ability to kill mycobacteria.

4. PATHOPHYSIOLOGY

When small silica dust particles are inhaled, they can embed themselves deeply into the tiny alveolar sacs and ducts in the lungs, where oxygen and carbon dioxide gases are exchanged. There, the lungs cannot clear out the dust by mucous or coughing¹³. When fine particles of silica dust are deposited in the lungs, macrophages that ingest the dust particles will set off an inflammation response by releasing tumor necrosis factors, interleukin-1, leukotriene B4 and other cytokines. In turn, these stimulate fibroblasts to proliferate and produce collagen around the silica particle, thus resulting in fibrosis and the formation of the nodular lesions. The inflammatory effects of crystalline silica are apparently mediated by the Nalp3 inflammasome.¹⁴

Furthermore, the surface of silicon dust can generate silicon-based radicals that lead to the production of hydroxyl and oxygen radicals, as well as hydrogen peroxide, which can inflict damage to the surrounding cells¹⁵.

5. DIAGNOSIS

Characteristic lung tissue pathology in nodular silicosis consists of fibrotic nodules with concentric "onion-skinned" arrangement of collagen fibers, central hyalinization, and a cellular peripheral zone, with lightly birefringent particles seen under polarized light.¹⁶ In acute silicosis, microscopic pathology shows a periodic acid-Schiff positive alveolar exudate (alveolar lipoproteinosis) and a cellular infiltrate of the alveolar walls. Physical check up will reveal decreased chest expansion and abnormal breath sounds. Pulmonary function test will reveal reduced lung capacity.^{17,18}

A computed tomography or CT scan can also provide a more detailed analyses of the nodules, and can reveal cavitation due to concomitant mycobacterial infection.

Silicosis is an irreversible condition with no cure. Chest x-ray will confirm the presence of nodules in the lungs, especially in the upper lobes. Typically, it will also reveal eggshell calcification of the hilar lymph nodes. In rare cases, pulmonary nodules may also be calcified. In advanced cases of silicosis, coalescence of nodules may show up as large masses.^{19,20}



Photo1-X-Ray report of silicotic patient

6. TREATMENT

Treatment options currently focus on alleviating the symptoms and preventing complications.^{21,22} These include:

1. Stopping further exposure to silica and other lung irritants, including tobacco smoking.
2. Cough suppressants.
3. Antibiotics and antitubercular agents to prevent tuberculosis. These include isoniazid, rifampin, and pyrazinamide.
4. Chest physiotherapy to help the bronchial drainage of mucus.
5. Oxygen administration to avoid hypoxemia.
6. Bronchodilators to facilitate breathing.
7. Lung transplantation to replace the damaged lung tissue is the most effective treatment, but is associated with severe risks of its own.
8. Whole-lung lavage
9. Inhalation of powdered aluminium, d-penicillamine and polyvinyl pyridine-N-oxide.
10. Corticosteroid therapy.
11. The herbal extract tetrandine may slow progression of silicosis.^{23,24}

Prevention

The following precaution should be taken by the workers to prevent from silicosis^{25,26}

- Wear dust masks
- Do not smoke
- Respirator should be provided from the organisations
- Water spray is often used where dust emanates
- Dust can also be controlled through dry air filtering
- Identify work-place activities that produce crystalline silica dust and then to eliminate or control the dust.

7. CONCLUSION

From the above study it can be concluded that the silicosis is an incurable disease for both coal and stone mine workers, which is caused by inhalation of silica dust particles. By this study an attempt has been made to attract the attention of doctors, researchers, NGO's to develop a proper medication and vaccine kit for the complete treatment of this disease. It is also suggested that the precautions should be taken to get rid from this disease because precaution is better than cure.

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