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Study of Prevalence of Silicosis in Rural Area of Dausa

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Abstract

Silicosis is one of the most serious occupational health problems worldwide. It is thought to be more prevalent in low and medium-income countries. Individual studies among workers exposed to silica dust in various industries, reveals high prevalence of silicosis in India.

Keywords: Silicosis; Respirable crystalline silica.

Introduction

Silicosis is a fatal pneumoconiosis which affect millions of workers worldwide who are exposed to silica dust [1]. It is one of the most prevalent pneumoconiosis in India. The sources of silica dust generation are mining, blasting, stone cutting, rock drilling and quarry works. There are many other industries, where a high incidence of silicosis has been reported, like flour mills, agate industries and slate pencil industries which are very common in India. Despite the basic knowledge, silicosis is one of the neglected occupational diseases in India, with respect to policy-making as well as health research. As we saw various patients with clinical presentation of respiratory problems in our day to day practice and saw the prevalence of silicosis is very high.

Material and methods

We had examined hundred cases in outpatient department of Dausa a rural area of of Rajasthan, out of which 32 percent cases were diagnosed as of silicosis. Extensive history of patients who were at risk of developing silicosis based on exposure to various fields where they worked was taken. We have to differentiate between the major forms of silicosis by clinical presentation and radiographic findings. After taking detailed history of cough, fever, weight loss and haemoptysis we were able to suspect the diagnosis of silicosis. In initial phases, clinical manifestations may not be indicative therefore correlation with radiology is must.

Radiological alterations might be ovrerlapping from those due to tuberculosis. The appearance of opacities, and the finding of pleural effusion was seen radiologically. Overall, the disease was not easy to detect on clinical grounds in patients with silicosis. We in our study also correlate clinical, radiological as well as inflammatory marker as ESR and interleukin [6]. These were all together lead us to diagnose the Silicosis in thirty two patients out of hundred patients. Although the data is low but prevalence of silicosis in this belt was found to be thirty two percent as seen in one of the other study where the prevalence was, ranged from 37% among general mine workers to a higher 38-79% among stone mineworker [2,3].

Prompt detection and precise diagnosis of silicosis must be very important as differential with tuberculosis and silicotuberculosis changed the decision of treatment and this is dependent on radiographic techniques, including X-rays, high-resolution computed tomography and pulmonary function assessments [4,5].

Discussion

The burden of silicosis remains high in developing countries, and currently it is the world's most prevalent chronic occupational disease.

Long-term occupational exposure to Respirable crystalline silica is associated with the development of silicosis, an interstitial lung disease presented as diffuse pulmonary fibrosis [6].

Respirable crystalline silica particles are small enough to escape the pulmonary mucociliary defense system and reached to the terminal airways and alveoli, where they get coating of pulmonary surfactant [7]. The surfactant is believed to modify the particles in protective manner, and used to decrease their toxicity temporarily. In response to immune stimulation by the silica particles, alveolar macrophages influence alveolar type II cells and bronchiolar epithelial cells to produce large amount

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of surfactant. However, the protective effect of the surfactant against silica-induced lung damage is only temporary, lasting approximately two days, after which the surfactant undergoes enzymatic proteinaceous digestion. The copious production of surfactant and the accumulation of the denatured protein is an acute pathological feature of silica exposure. This build-up the proteinaceous debris in the alveoli, recognized as silicoproteinosis.

In the terminal airways, the Respirable crystalline silica particles are engulfed by alveolar macrophages to clear the lung of the inhaled debris. Particles become entrapped in lysosomes, where they are resistant to degradation.

Within the lysosome, silica produces reactive oxygen species, which damage the lysosomal membrane, leading to the spillage of the digestive enzymes into the cytoplasm of the macrophage and promoting apotosis. Any dysfunctional alveolar macrophages persisting in the alveoli cannot effectively clear debris from the alveoli, thus leads to collection of intra-alveolar material.

Macrophages collect at damaged pulmonary tissue, stimulating the activity of fibroblasts, leading to pulmonary fibrosis, formation of silicotic nodules, and reduction of lung area available for gas exchange [8]. The silica particles released by macrophage destruction get out by the mucociliary escalator, transported through lymphatic drainage, and engulfed by other alveolar macrophages, stimulating a vicious cycle of cellular destruction, inflammation, and pulmonary damage. The lymphatic spread of pulmonary macrophages may explain the origin of any extrapulmonary manifestations of silicosis, including lymphadenopathy. These pathologic changes are much more indolent than the abrupt silicoproteinosis experienced in voluminous acute exposures.

The 3 major forms of silicosis are acute, accelerated, and chronic. Chronic silicosis, the most commonly encountered form of silicosis, is also known as classic silicosis and may be simple or complicated. Complicated chronic silicosis is also known as pulmonary massive fibrosis.

Acute silicosis usually presents within a few weeks to less than five years after Respirable crystalline silica exposure. While anyone with high-intensity exposure to Respirable crystalline silica may develop silicoproteinosis, it is most commonly identified in individuals who have performed abrasive sandblasting or work in tunnels. The pathology of acute silicosis is characterized by significant alveolitis and alveolar proteinosis. Patients with acute silicosis are generally symptomatic, reporting pulmonary and systemic symptoms, including dyspnea, pleurisy, cough, fevers, fatigue, and weight loss. Physical examination frequently reveals hypoxia [9].

Accelerated silicosis is characterized by progression of disease which is more rapid than chronic silicosis. Specifically, the accelerated form of silicosis develops within approximately seven years of exposure of Respirable crystalline silica. Accelerated silicosis exhibits features that overlap acute and chronic silicosis. There may be evidence of silicoproteinosis where patients also demonstrate the silicotic nodules of chronic disease. However, the pulmonary nodules intensify more quickly.

Chronic silicosis is the most common presentation of this pneumoconiosis, usually presenting ten to thirty years after prolonged exposure to low concentrations of Respirable crystalline silica. Radiographic findings required to differentiate between simple and complicated chronic silicosis. Simple chronic silicosis is also called nodular silicosis, characterized by pulmonary nodules less than 10 mm in diameter.

The physical examination of patients with accelerated or chronic silicosis may show lymphadenopathy. Although, in lower levels of silica exposure, the development of lymphadenopathy may precede the development of pulmonary findings.

Patients with simple chronic silicosis are frequently asymptomatic they may present with a dry cough. Simple silicosis may be transition to complicated silicosis as the nodules progress. Pulmonary fibrosis develops when the pulmonary nodules fuse to conglomerate masses with a diameter of greater than 10 mm. Patients with Pulmonary fibrosis usually develop respiratory impairment due to the profound destruction of lung tissue, which results in decreased perfusion capacity, impairment in lung function, and elevated pulmonary resistance. Pulmonary hypertension and cor pulmonale may results from the lung fibrosis. Thus silicosis is a devastating and incurable disease that is often difficult to diagnose. Once diagnosed, proper patient education is very much needed which may lead to improved quality of life. A coordinated effort of primary care practitioners, pulmonologists, nursing staff, occupational health specialists, and transplant surgeons will lead to the best patient outcomes.

Limitation: As duration as well as the sample size was very low but in concordance with other studies on silicosis.

Conclusion

Patients must be counselled on preventing further exposure to RCS to avoid the development of silicosis as it significantly increased risk of pulmonary malignancy experienced in individuals exposed to crystalline silica. Thus patient education is must to eradicate silicosis.

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