

Silicosis Diagnosed by Transbronchial Lung Biopsy – A Case Report and Review

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Abstract: *Silicosis is a fibrosing pneumoconiosis caused by inhalation, retention and pulmonary reaction to crystalline silica. Silica or silicon dioxide is the predominant component of the earth's crust. Despite knowledge of the cause of this disorder, this serious and potentially fatal occupational lung disease remains prevalent throughout the world. Occupational exposure to silica particles has been associated with mining, quarrying, drilling, tunnelling, and sandblasting. The diagnosis of silicosis is based on a thorough history and radiological evaluation, a biopsy being required only when the radiological diagnosis is uncertain. When required, an open or thoracoscopic lung biopsy is necessary to obtain adequate material for examination; however these procedures are associated with significant morbidity. Transbronchial lung biopsy is an alternative method of diagnosis associated with lesser morbidity. However in view of the smaller size of the sample the diagnostic yield is lower. We present a case of a 39 year old male with silicosis diagnosed by transbronchial lung biopsy.*

Keywords: Occupational Lung Disease, Silicosis, Transbronchial lung Biopsy

1. Introduction

Pneumoconiosis resulting from exposure to free silica may be the commonest and most extensively studied occupational disease of the lung and even today, it continues to be among the most serious occupational diseases.¹ The prevalence of silicosis in India ranges from 3.5% in the ordinance factory to 54.6% in the slate pencil industry. Stone quarry workers and sandblasting are associated with a prevalence of 22% and 27.8% respectively.² Exposure to large amounts of free silica can go unnoticed because silica is odourless, non-irritant and does not cause any immediate health effects. The diagnosis of silicosis is based on a thorough history of exposure to silica particles and is aided by radiological and in a few cases pathological findings. As silicosis is incurable, clinical management includes removing the worker from the industry and giving symptomatic treatment.³ We present a case of a 39 year old sandblaster who was diagnosed as silicosis by transbronchial lung biopsy.

2. Case Report

A 39 year old male presented to the Department of Pulmonary Medicine, Goa Medical College with a history of progressively increasing dyspnoea and non-productive cough of one year duration. His symptoms had further worsened over the last month. He was a chronic smoker with a smoking index of 100. He was working in a shipping company for the past 15 years and his job included welding and sandblasting. There was no clubbing, cyanosis or lymphadenopathy noted. The respiratory system examination was unremarkable. Spirometry revealed moderate obstructive defect with no significant

bronchodilator reversibility. Chest radiograph showed bilateral hilar prominence with reticulonodular shadows in mid and lower zones bilaterally (Fig 1). CT scan Thorax showed interstitial thickening with nodular appearance; multiple nodules in both perihilar regions and upper lobes and bilateral mediastinal lymphadenopathy with peripheral calcification (Fig 2-5). Based on his clinical and radiological findings a differential diagnosis of silicosis or sarcoidosis was considered. His ACE levels were mildly elevated (76 U/L). A flexible videobronchoscopy and a transbronchial lung biopsy were done to confirm the diagnosis. The histopathology of specimen showed small rounded fibroblast proliferation with dust laden macrophages; polarisation showed silica crystals, thus confirming the diagnosis of silicosis (Fig 6,7). There were no procedure related complications. As there is no specific cure for silicosis, he was treated symptomatically and advised to consider a change in his job.



Figure 1: Shows chest radiograph showing reticulonodular shadows in mid and lower zones bilaterally. Also note bilateral hilar prominence.

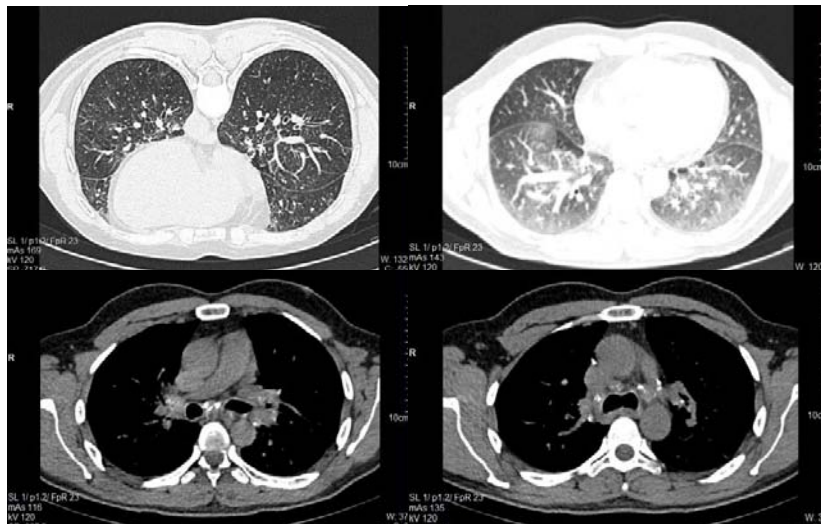


Figure 2,3,4,5: Show Computed tomography images showing thickening of axial interstitium with few interfaces showing nodular appearance. There is bilateral symmetrical distribution of small nodules in perihilar and peribronchovascular interstitium, as well as bilateral hilar and mediastinal lymphadenopathy with peripheral calcification.

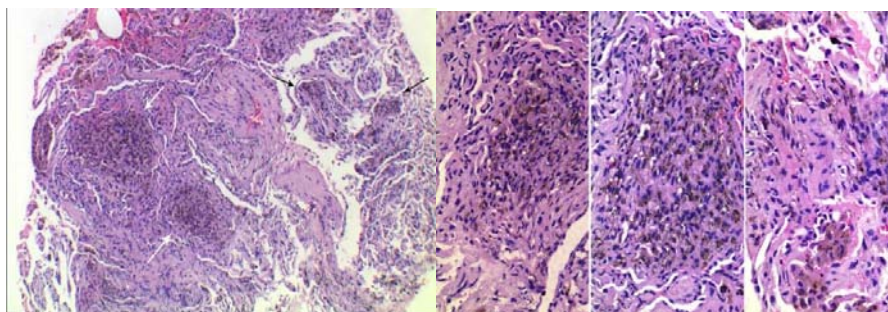


Figure 6: Shows transbronchial lung biopsy images showing small rounded fibroblastic proliferation with dust laden macrophages. Polarisation showed silica crystals. Fig 7 shows the images with higher magnification

3. Discussion

Silicosis is an occupational lung disease attributable to the inhalation of silicon dioxide; commonly known as silica.⁴ The problem of silicosis is confined not only to the developing nations, but is also not uncommon in industrialized nations. Since the earth's crust contains about 12% free silica mostly in the form of quartz, mining and tunnelling are the occupations most closely related to the hazard of silica exposure. The sand stone industry, stone quarrying and dressing, granite industry, grinding of metals, sand blasting, iron and steel foundries, silica milling, flint crushing and manufacture of abrasive soaps are some of the occupations related to silica exposure. Some of the occupations such as slate pencil industry and agate grinding industry which carry high risk of silicosis are peculiar to India. There are about 3 million workers at high potential risk of silica exposure. They are employed in various occupations such as mining and quarries (17 lakhs); manufacture of non-metallic products *ie.*, refractory products, structural clay, glass, mica, *etc.* (6.3 lakhs) and manufacture of basic metals and alloys, *ie.* iron and steel, copper, ferroalloys, aluminium, *etc.* (6.7 lakhs) In addition many of the 54 lakhs construction workers are also at risk of silica exposure.⁵

The primary pathology in silicosis is the formation of silicotic nodules. The precise mechanism of silicosis is not known but is thought to be mediated by the generation of

reactive oxygen species both by the surface of the silica particles themselves as well by the activation of alveolar macrophages.⁷ The inhaled silica particle is engulfed by the alveolar macrophages. However they are unable to digest the material. The silica particle in the macrophages damages the lysosomal membranes of the alveoli which triggers the release of proteolytic enzymes into the cytoplasm leading to death of the macrophage. Continued exposure results in an alteration of the macrophage function. Release of inflammatory cytokines like interleukin1, free radicals and growth factor follows which stimulates collagen synthesis and production of antibodies against collagen. These anticollagen antibodies stimulate fibroblasts to then produce more collagen which eventually leads to silicotic nodule formation.^{12,13}

Three forms of silicosis have been described – Acute, Chronic and Accelerated Silicosis. Chronic or classic silicosis results from 15 or more years of exposure to respirable silica, may be asymptomatic or may be associated with progressive dyspnoea and cough. Accelerated Silicosis results from heavier exposures with a latency of 5- 10 years. It generally progresses even after exposure is interrupted. Acute silicosis is rapidly progressive following high levels of exposure to silica over few months up to five years.⁶

The primary symptom is dyspnoea which progressively increases over time. However, at times the presentation may be an asymptomatic worker with an abnormal radiograph.

The dyspnoea is usually accompanied by a productive cough. Other respiratory symptoms are rare. Haemoptysis if present should raise suspicion of complications such as tuberculosis or malignancy. Wheeze and chest tightness may occur; chest pain and clubbing are not typical features of silicosis. In late stages, corpulmonale results. Silicosis may be complicated with other lung diseases including lung cancer and autoimmune diseases. Patients with silicosis are more susceptible to developing pulmonary tuberculosis, "silicotuberculosis".¹⁴ Patients with silicosis are also prone to develop repeated infections, chronic obstructive pulmonary disease and tracheobronchial compression by enlarged mediastinal lymph nodes. Pleural involvement in silicosis is rare. Few instances of bilateral pneumothorax have been reported.

Radiological lesions vary in different types of silicosis. On HRCT thorax, multiple bilateral centrilobular opacities, multifocal patchy ground-glass opacities, and consolidation with occasional crazy paving characterize acute silicosis. Chest radiograph of patients with chronic simple silicosis shows the presence of multiple small nodules, 2-5 mm in diameter with associated calcifications. These nodules have upper lobe predominance. HRCT in chronic simple silicosis shows perilymphatic distribution of nodules in centrilobular, paraseptal, and subpleural regions. Hilar and mediastinal lymphadenopathy may precede the parenchymal lesions. Eggshell pattern calcification of lymph nodes is common.

Complicated silicosis, also known as progressive massive fibrosis, develops with confluence of individual silicotic nodules. On CT scan, PMF appears as focal soft-tissue masses, usually measuring more than 1 cm in diameter, with irregular margins, calcification, and involving apical and posterior segments of the upper lobes, surrounded by areas of emphysematous changes. With advancing fibrosis, these opacities migrate towards hila with resultant paracicatricial emphysema. In silicosis associated with tuberculosis (silicotuberculosis) the radiological picture includes asymmetric nodules or consolidation, cavitation and a rapid disease progression.^{15,16,17}

With a characteristic history and Chest radiograph the diagnosis is usually easily established. Challenges arise when the radiologic features are unusual or history of exposure doubtful. Lung biopsy is required in these cases. Open or thoracoscopic lung biopsies are generally necessary to obtain an adequate sample. Some evidence suggests BAL and TBLB may be of diagnostic aid.⁹ TBLB achieves a high diagnostic yield in diffuse parenchymal lung diseases (DPLDs) with centrilobular accentuation, such as granulomatous and metastatic diseases, infection, alveolar proteinosis and eosinophilic pneumonias. Problems with TBLB include sampling errors and small specimen size, making it difficult to distinguish different patterns of DPLD with overlapping histological features. Crushing of the specimen and failure to penetrate beyond the peribronchial sheath may also preclude histological assessment. Overall, the diagnostic rate is ~38–79%.¹⁸ However there is not much literature on the diagnostic efficacy of TBLB in silicosis. The pathologic hallmark in the lungs of patients with chronic silicosis is the silicotic nodule. The lesion is characterised by a central acellular area of concentrically

arranged, whorled hyalinised collagen fibers surrounded by connective tissue with reticulin fibers.¹⁰

If silicosis is diagnosed in a worker, termination of further exposure is advisable. However, often the disease progresses even after elimination of silica exposure. Patients diagnosed with silicosis should be screened for tuberculosis. Patients are usually treated symptomatically for their complaints. Bronchodilators and inhaled steroids usually offer little or no benefit. It should be emphasized that although silicosis is not a curable disease it is preventable. Education to workers, use of personal protective equipment and periodic medical screening should be done compulsorily. The finding of a case of silicosis should be considered as a sentinel health event and prompt the examination of other workers and the workplace exposure and control measures.

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