

CASE REPORT

SILICOSIS- From Grain To Granuloma

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ABSTRACT

BACKGROUND: Silicosis is a fibrosing disease of lungs caused by inhalation, retention, and pulmonary reaction to crystalline silica due to an occupational exposure to silica particles of respirable aerodynamic size¹. In India it is estimated that about 3 million people working in various types of mines, ceramics, potteries, foundries, metal grinding, stone crushing, agate grinding, slate pencil industry etc., are occupationally exposed to free silica dust and are at potential risk of developing silicosis². The disease is often unrecognized and underreported, and thus its true prevalence is substantially underestimated.³ We hereby present two cases portraying the diverse clinic-radiological spectrum of silicosis.

Key Words : Silicosis, occupational lung disorder, pulmonary nodules, dyspnoea

CASE REPORT

Case 1

A 50 year old male non smoker working as a supervisor in a ceramic tile factory was referred to the department of pulmonary medicine suspecting lung malignancy with non progressive breathlessness since 9 years. Accompanying chest radiographs, nine and four years before, revealed incremental lung masses.

Figure1: Chest Xrays



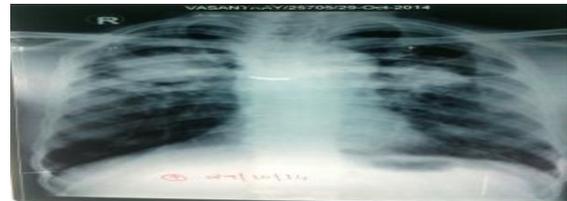
9years back



4years back

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Current

Patient was physically active without chest pain, hemoptysis or weight loss. On examination, patient was afebrile, with normal vital parameters, clubbing and lymphadenopathy were absent. Respiratory system examination was unremarkable except for bilateral auscultatory rhonchi on deep breathing. Hematological and biochemical investigations were normal. Sputum smear and culture was negative for acid fast bacilli. Spirometry showed mild fixed obstruction with mixed restrictive component. A chest CT scan was obtained which revealed well defined minimally enhancing mass lesion with internal amorphous calcifications in both upper lobes. Ill defined centrilobular and subpleural nodules were seen.

Figure2: Contrast enhanced CT chest



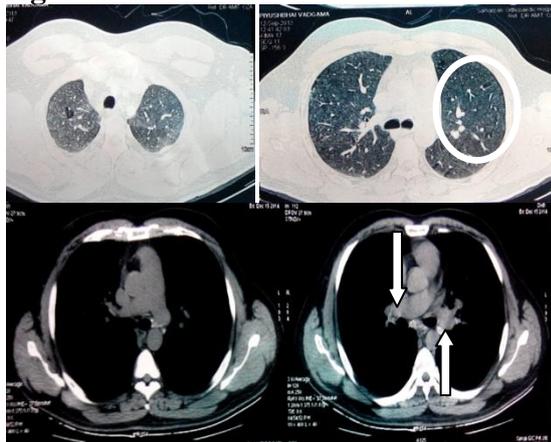


Hilar lymphadenopathy was also noted. (thin arrow- bilateral parenchymal mass lesions; thick arrow- internal foci of calcification within mass lesion) Fine needle aspiration cytology and biopsy was negative for malignancy. Biopsy revealed conglomerated hyalinized nodules with peripheral cellularity.

Case 2

A 45 year male non smoker factory worker engaged in amry unit for 5 years, presented with one year history of progressively worsening dyspnoea, without fever, cough or hemoptysis. He was diagnosed to have hypersensitivity pneumonitis based on the HRCT chest scan available

Figure4: HRCT chest



(Circle- Area on HRCT scan showing dense centrilobular nodules; Arrows- calcified mediastinal nodes)

There was no exposure to birds or any organic dust either at home or at workplace, neither was he treated by any herbal medications. Patient was afebrile with normal vital parameters. Respiratory system examination was unremarkable except for exertional tachypnoea. Biochemical investigations were normal. Spirometry showed a mixed obstructive as well as restrictive pattern. 6MWD was reduced and significant oxygen desaturation was observed. A chest

radiograph revealed bilateral diffuse micronodular opacities and prominent hila

Figure3: Chest Xray



HRCT chest showed diffuse bilateral multilobar ill defined centrilobular, subpleural nodules with focal calcified mediastinal lymph nodes.

DISCUSSION

Pathogenesis and resultant clinical manifestations of silicosis depend on intensity and duration of exposure to crystalline silica, three forms of the disease are well characterized- chronic (classic), accelerated, and acute. The clinical presentation varies from acute rapidly progressive, often fatal, to the more indolent stable course leaving the patient chronically breathless.^{4,5,6} Chronic form develops after one or more decades of low intensity exposure with clinical scenario ranging from being asymptomatic to breathlessness of variable grade and progression. Chronic silicosis can be either simple or complicated. Simple silicosis is radiologically defined by diffuse small (<10mm) rounded pulmonary opacities which usually predominate the upper lobes. Coalescence of these nodules results in larger conglomerated opacities which is indicative of complicated silicosis or progressive massive fibrosis.^{7,8} Accelerated form develops from heavier exposure of 5-10 years duration presenting with more rapidly developing dyspnoea than the chronic form. On chest CT scan, diffuse bilateral small rounded opacities, similar to those in the chronic form, are present. Acute silicosis develops within few months of intense and massive silica dust exposure leading to rapidly progressive dyspnoea and respiratory failure. On chest CT, an alveolar proteinosis like appearance with septal thickening and diffuse bilateral ground glass opacities are seen, also termed as the crazy-paving appearance. Dyspnoea is the common symptom of all forms of silicosis. Cough is often accompanying symptom

due to occupational or smoke related chronic bronchitis or irritation of airways secondary to enlarged lymph nodes. Chest pain, hemoptysis and fever are not the usual symptoms, but when present should arouse the suspicion of secondary infection, including tuberculosis, or onset of neoplasm. Radiologically, classic silicosis or the simple nodular form comprises of either ILO type p opacities (<1.5mm diameter) comprising of ill defined centrilobular and peribronchiolar opacities, or ILO type q (1.5-3 mm diameter) ILO type r (>3mm diameter) which are characterized by sharply demarcated, rounded or irregular, contracted nodules. Nodules are perilymphatic in distribution, involving centrilobular, paraseptal and subpleural regions. Subpleural opacities may have a triangular configuration, and if they are confluent may resemble pleural plaques. Focal emphysema surrounding the nodule may be common. Nodules can be calcified. Enlargement of hilar and mediastinal lymph nodes is the usual accompanying feature and can precede the parenchymal lesions. Calcification in the periphery and the eggshell calcification pattern can be highly suggestive of silicosis.⁹ Progressive massive fibrosis on CT chest appears as large focal masses with irregular dull margins and punctate calcification, with apical scarring and distortion of lung architecture and development of adjacent bulla (paracicatrizing emphysema). Cavitation secondary to ischemic necrosis may occur in a mass larger than 5 cm.⁹ Differential diagnoses include miliary tuberculosis, sarcoidosis and lymphangitic spread of carcinoma. Patient in case one was diagnosed to have chronic complicated silicosis with progressive massive fibrosis. Patient in case two was diagnosed to have accelerated silicosis with nodular CT pattern Effective exposure controls are available for most processes, and include process enclosure, wet abrasive techniques, and local exhaust ventilation, combined with a comprehensive approach to personal protection. Wherever possible, less hazardous agents should be substituted for

silica. Patients ought to be counselled regarding occupational rehabilitation and if necessary a change in job.

Preventing infection: In the medical management of silicosis, vigilance for complicating infection, especially tuberculosis, is critical, the use of preventive isoniazid (INH) therapy in the tuberculin-positive silicotic patient is advised.

For acute presentation: Ventilatory support might be necessary when patient presents with respiratory failure. An alveolar proteinosis pattern on HRCT with severe hypoxia might need aggressive therapy with whole lung lavage under general anaesthesia. Corticosteroids and bronchodilators are of unproven benefit.¹⁰ Selected younger patients with end-stage disease may be considered candidates for lung or heart– lung transplantation by centers experienced with this expensive and high-risk procedure.¹⁰

Learning Points

- Silicosis has a wide range of clinico-radiographic abnormalities.
- Dyspnoea is a dominant symptom across all forms of the disease.
- Detailed occupational history with quantitative and qualitative assessment of workplace exposure is of paramount importance.
- Combination of silicosis specific radiographic(CT) abnormalities and documented workplace exposure of silica dust clinches the diagnosis.
- Timely avoidance of silica dust exposure, prevention of complications and control of dyspnoea are remedial measures

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