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Progressive Massive Fibrosis Masquerading As Lung Malignancy

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Abstract

AIM: Silicosis is the most common occupational lung disease caused by inhalation of large amount of silica dust. High risk population includes the People who are employed in industries like mining, sandblasting, stone cutting, ceramic making, pottery and Brick making industries. There is no care for silicosis, and treatment options are often limited. Avoidance of further exposure is the first step in the treatment and other modalities like personal protective measures and periodic health check-up should be done in all industries with high chances of occupational dust exposure. We report a case of Progressive massive fibrosis masquerading as Lung mass.

Keywords: PMF, Silicosis, Progressive massive fibrosis, Pneumoconiosis

Introduction

Progressive Massive Fibrosis (PMF) is a chronic debilitating occupational health disease occurring in persons working in respirable dust industries[1].Commonly seen in association with occupational lung diseases like Silicosis and Coal workers pneumoconiosis. We report a case chronic complicated silicosis presenting as progressive massive fibrosis. The provisional diagnosis of PMF was made based on the occupational exposure history along with radiological features and later confirmed by histopathology.

Case Description:

A 58-year-old gentle man presented to our department on outpatient basis with history of progressive exertional dyspnoea and dry cough for past 2 years now increased for past 2 months. He was an Ex-Smoker but not an alcoholic. He had worked in stone quarry industry as a driller for thirty years and had no underlying comorbities. On examination his vitals were stable except he had grade 2 Clubbing. Respiratory system examination revealed bilateral scattered crepitations. His Chest x-ray (fig 1) showed large symmetrical mass like opacities in bilateral hilar regions with irregular margins and diffuse reticulonodular opacities in bilateral midzones. Contrast Enhanced Computed Tomography of Chest revealed Heterogenous soft tissue density lesion with foci of calcifications in bilateral upper lobes predominantly in the hilar and the peri hilar regions. Multiple calcified mediastinal lymph nodes seen along with multiple ill-defined lesions with with surrounding fibrosis seen in right middle lobe and superior segment of left lower lobe (Fig 2). The patient underwent CT guided trucut biopsy of lung lesion and Histopathology revealed features of Silicosis with fibrosis (Fig 3). The patient is currently managed symptomatically and advised to quit his job and is current on regular follow up, has not worsened till the last visit.

Figure 1 : Chest X-ray showing bilateral symmetrical mass like lesions with irregular margins in the hilar region in bilateral upper and midzones – commonly termed as "Angel wing Sign"

Figure 2: CECT Thorax: Mediastinal window axial cut shows Calcified mediastinal lymph nodes with irregular mass like lesions in bilateral upper lobes

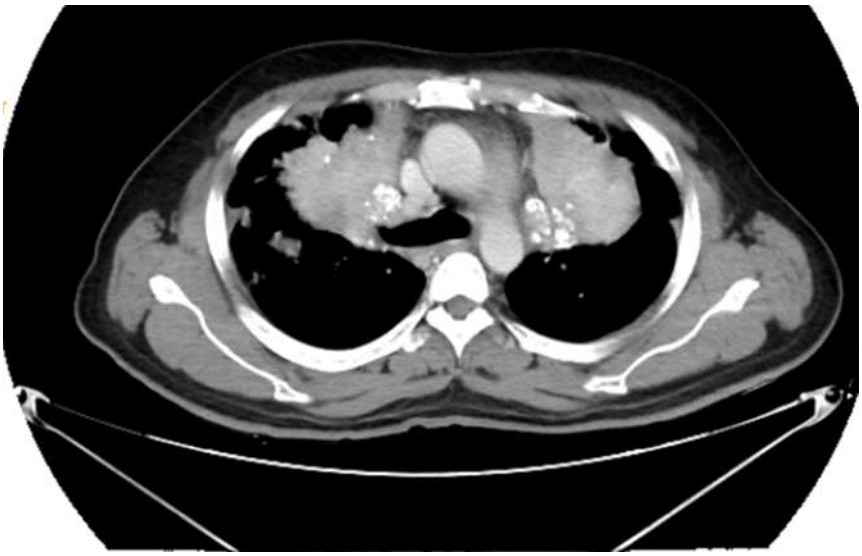

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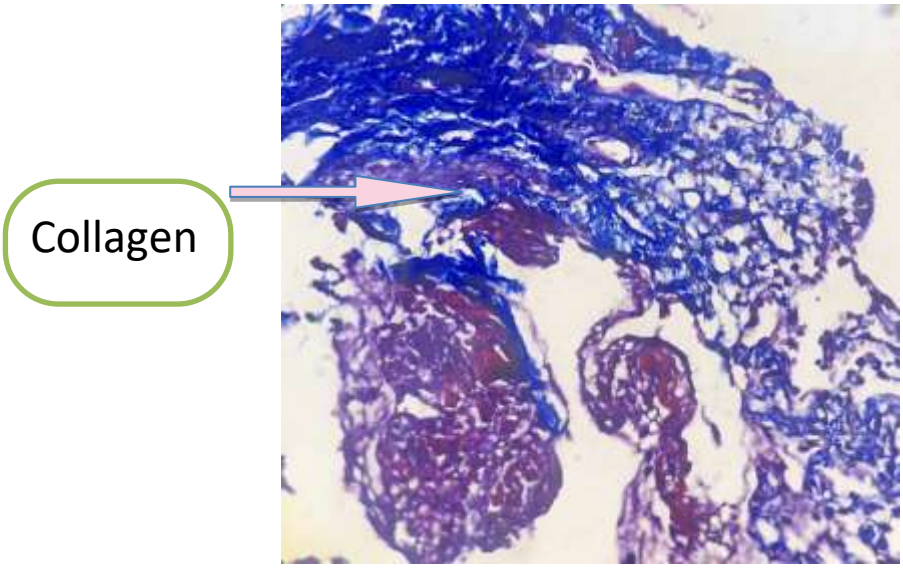
Figure: 3 –Trucut biopsy of lung mass – Histopathology with Mason trichrome staining shows collagen deposits suggestive more in favour of silicotic nodules with occasional fibroblast deposition suggestive of fibrosis.



Discussion:

Silicosis is a fibrotic occupational lung disease caused by inhalation, retention and pulmonary reaction to large amount of crystalline silica (silica dust). Silicon is abundant in nature as quartz or as silicates. Majority of silica exists in quartz form which constitutes around 28 % of total earth crust[2]. People employed in occupations like mining, sandblasting, stone cutting, ceramic making, pottery and Brick making industries are predisposed for developing silicosis. In India, people working in Slate pencil industries and agate grinding industries carry a high risk of developing silicosis[2–4].

The pathognomical feature of silicosis is the presence of silicotic nodules. The inhaled silica dust is phagocytosed by the Alveolar macrophage. The phagocytosis process is however incomplete due to Alveolar cell membrane damage leading to release of proteolytic enzymes in to the cytoplasm leading to the death of the macrophage. Prolonged exposure results in altered macrophage function which in releases inflammatory cytokines like Interleukin-1 and Free radicals leading to fibroblast activation and more collagen production which in turn leading to silicotic nodule formation[3–5].

Based on the onset of symptoms and duration of exposure, this silicosis can be classified in to acute silicosis, chronic silicosis and accelerated silicosis. Commonest presentation is the "Chronic simple silicosis" which develops following low to moderate level of exposure to silica dust for more than 20 years. "Accelerated silicosis" develops following large exposure to silica for a period of 5 to 10 years and had faster progression than chronic silicosis and sometimes is associated with Connective tissue disorders. Acute silicosis develops when there is inhalation of very high concentration of silica over weeks to months and leads to intense inflammatory reaction due to freshly fractured silica particles causing respiratory failure and death[6].

Majority of the patients usually seek medical help only in the chronic silicotic stage. Commonest symptom being dyspnoea on exertion and is usually progressive in nature. Cough is usually nonproductive and in later stages can present with expectoration suggestive of secondary infection. Chest pain and haemoptysis usually indicates complications like tuberculosis, lung cancer and autoimmune disorders. Silicosis patients are more vulnerable for developing pulmonary tuberculosis," Silicotuberculosis" [7]. These patients are also susceptible for recurrent respiratory tract infections and tracheobronchial compression by Enlarged mediastinal nodes.

Radiological patterns vary in different types of silicosis. In Acute silicosis, High Resolution

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Computed Tomography (HRCT) thorax shows multifocal patchy ground glass opacities with occasional crazy paving pattern mimicking pulmonary alveolar proteinosis. HRCT in chronic simple silicosis shows silicotic nodules which are peri lymphatic distribution in nature, Hilar and mediastinal lymphadenopathy with Egg shell calcification. Complicated silicosis, also known as progressive massive fibrosis occurs when all the silicotic nodules enlarge and coalesce (>2cm) together appearing like lung masses. On CT scan PMF appears as focal mass like lesions with irregular margins with mediational lymphadenopathy and egg shell calcifications involving upper lobes surrounded by areas of emphysematous changes. In Silicotuberculosis the radiological picture could be asymmetric randomly distributed nodules or consolidation, cavitation with a rapid disease progression[8–9].

Diagnosis of silicosis is mainly obtained by correlating the occupational exposure history along with radiological features of silicosis. Lung biopsy is rarely required in the diagnosis of PMF; however, biopsy is indicated to rule out lung malignancy and confirm the diagnosis of silicosis which shows silicotic nodules with collagen deposits[5]. Recently newer biomarkers like Serum CC16 (Club cell protein) helps detection of silicosis in their earlier stages, which inturn helps in preventing premature deaths in PMF[12].

Avoidance of further exposure is the first step in the treatment, as there is no permanent cure this dreadful disease. Most of these patients are symptomatically managed with bronchodilators and corticosteroids[10–11].

Conclusion

Education of workers and use of PPE along with periodic medical check-up should be made mandatory in all industries with silica dust exposure. If PMF is diagnosed the patient should be immediately advised to quit his job and consider an alternative occupation to prevent further worsening. In countries like india with high burden tuberculosis, possibility of Silicotuberculosis should always be considered in patients with high clinical suspicion.

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