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### RESEARCH ARTICLE

#### A CASE OF HEMOPTYSIS DUE TO SILICOTUBERCULOSIS: CASE REPORT.

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#### Manuscript Info

##### Manuscript History

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#### Abstract

A 60year old gentleman, mason by occupation, presented with shortness of breath since 9months, cough since 5months, streaky hemoptysis since 5days. He is a smoker with 20 pack-years. On examination patient is tachypneic and sings of bilateral lung consolidation was present. Chest X-ray showed Non-homogenous opacity in bilateral upper zones and left mid zone and calcified lesions in left para-tracheal location and in right cardio phrenic angle. Sputum for AFB was negative. Spiral CT-chest revealed calcified mediastinal lymphnodes and bilateral hilar soft tissue density lesions extending peripherally into posterior segment of upper lobe and superior segment to lower lobe- suggestive of progressive massive fibrosis. In view of occupational history, considered as pneumoconiosis. CT guided lung biopsy of right upper lobe lesion was done and sent for HPE-reported as Tuberculosis. ATT was started under RNTCP along with supportive treatment.

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#### Introduction:-

Silicosis is a chronic lung disease caused by breathing of silica dust. Silica is one of the common mineral in the earth's crust. Among the clinical complications of silicosis tuberculosis (called silico-tuberculosis), is still a major public health concern in low- and middle-income countries. Prevalence of pulmonary tuberculosis in Silicosis is 3-7 times more common when compared to prevalence in general population. Overall, it appears that TB occurs in as many as 20 to 25% of all silicosis patients in their lifetime.

#### Pathogenesis:

Experimental studies showed that silica impairs the function of alveolar macrophages. The silica particles phagocytosed by macrophages are engulfed in phagolysosomes. Silica has the capacity to cause damage of macrophages with re-exposure of the other macrophages to the same particle. Cytokines, IL-1 beta, TNF-alpha, released by macrophages cause local damage, fibroblast activation and fibrosis. Lung fibrosis occurs in both diseases and may interfere with the clearance of dust laden alveolar macrophages & mycobacteria laden material from the lung. Lymphatic obstruction causes macrophages to accumulate in the interstitial tissues resulting in local fibrosis. Silicotuberculosis affects not only the parenchyma but also the arteries and the veins. There is thickenings of the intima, hyaline and lipid degenerations, scars in the vessels, impeding the blood circulation.

Iron hypothesis:

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Mycobacteria are dependent on iron for growth and produce the iron chelators mycobactin and exochelin. Silica particles absorb body iron and act as a reservoir of iron. Silicoto-iron complexes may activate dormant tubercle bacilli.

**Case report:**

A 60year old gentleman, mason by occupation, presented with shortness of breath since 9months, cough since 5months, streaky hemoptysis since 5days. No history of fever, chest pain, loss of appetite and loss of weight. He is a smoker with 20 pack-years. No history of hypertension, diabetes mellitus, tuberculosis. On examination patient is tachypneic and sings of bilateral lung consolidation was present.

**Investigations:**

Complete hemogram was within normal limits, sputum for AFB was negative, viral markers were negative. Chest X-ray showed Non-homogenous opacity in bilateral upper zones and left mid zone and calcified lesions in left paratracheal location and in right cardio phrenic angle. Spiral CT-chest revealed calcified mediastinal lymphnodes and bilateral hilar soft tissue density lesions extending peripherally into posterior segment of upper lobe and superior segment to lower lobe- suggestive of progressive massive fibrosis. In view of occupational history, considered as pneumoconiosis. CT guided lung biopsy of right upper lobe lesion was done and sent for HPE-reported as Tuberculosis.

**Diagnosis:**

Silico-tuberculosis.

**Treatment given:**

CAT-1 ATT under RNTCP, Tranexamic acid, Proton pump inhibitors, bronchodilators and supportive treatment.

**Discussion:-**

Although treatable, tuberculosis in silicosis patients may go undiagnosed because cough, wheeze, expectoration, dyspnoea and vague chest pains are symptoms common to both diseases. Interpretation of the chest X-ray film of patients with silicosis is difficult due to the superimposition of silicotic nodules and tuberculous infiltrations. Mycobacterium tuberculosis bacilli may not be recovered from the sputum of silicotuberculosis patients because silicotic fibrosis prevents the discharge of tubercle bacilli into the sputum. For early and accurate diagnosis, frequent sputum examination for AFB, Mycobacterial culture where high prevalence of atypical mycobacteria, fiberoptic bronchoscopy- bronchoalveolar lavage/trans-bronchial lung biopsy, Transthoracic needle biopsy are helpful.

According to study by Cowie RL et al. the risk of a patient with silicosis developing pulmonary tuberculosis is reported to be 2.8 to 39 times and for extra-pulmonary tuberculosis is also as much as 3.7 times higher than in healthy population. The pleural form is most common, accounting for 61% of the cases, followed by the pericardial form and the lymph node form

**Conclusion:-**

Active surveillance should be carried out in pre and post-employment periods that include clinical examination, chest radiographs and tuberculin skin test. In our case diagnosis was done by transthoracic needle biopsy and histopathological examination.

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