A STONE MINER WITH SILICOSIS- CASE REPORT

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PRESENTATION OF CASE

A 45 years old male who used to work as a stone miner in Potka Block of East Singhbhum for 20 years complained of dry cough, shortness of breath, dyspnoea and severe weight loss for months and worsen 5 days before admission at MGM Medical College. At the time of admission, patient was dyspnoeic even at rest. He had also pedal oedema. Patient reported a smoking history of 30 years. In the meantime, patient died due to respiratory failure. For exact diagnosis, the post-mortem of the patient was done under a board of three doctors.

Mantoux test and sputum for AFB were negative. Autopsy report- both lungs were small and firm. Multiple bilateral small to large fibrotic nodules were present in the lungs predominantly at the upper and mid zones (Figure 2).

Pleural thickening was seen on both sides. Lungs were preserved in 10% formalin and sent to Pathology Department, MGM Medical College and Hospital, Jamshedpur, for HPE. Microscopic examination of supplied tissue section showed areas of multiple nodular lesions, which have concentric layers of hyalinised collagen surrounded by more condensed collagen (Figure 3). It also showed the areas of emphysematous changes in lung parenchyma and infiltrated with inflammatory cells and fibroblasts.

DIFFERENTIAL DIAGNOSIS

By the history and the clinical features (signs and symptoms), which are written in the case presentation, the differential diagnosis of the patient were-

a. Silicosis.
b. COPD with cor pulmonale.
c. Pulmonary tuberculosis.
d. Congestive cardiac failure.

CLINICAL DIAGNOSIS

Initial investigations including 10.5 gm/dL; ESR 30 mm; total leucocyte count 8500/cumm; x-ray chest showed bilateral diffuse small-to-large nodular opacities throughout the lung fields. These nodules were more at the upper zone of lungs (Figure 1). Pulmonary function test revealed a restrictive defect. Heart shadow was enlarged with right ventricular dilatation. Mantoux test and sputum for AFB were negative.

On the basis of history, clinical features and the above investigation, the clinical diagnosis of the patient was "chronic complicated silicosis."

PATHOLOGICAL DISCUSSION

Silicosis is the most prevalent occupational lung disease in Jharkhand. It is caused by inhalation of crystalline silica dust (silicon dioxide), which is marked by inflammation and nodular fibrosis in the lung. Pulmonary tuberculosis is also very common in the rural areas of East Singhbhum, Jharkhand, and the risk of tuberculosis is increased by about 20-30 folds in silicosis.

Silicosis is a pneumoconiosis. After inhalation, the silica dust particles are engulfed by alveolar macrophages and released various cytokines, oxidants, growth factors, which injured the epithelial cells and causes fibrosis mainly in the uppers and the middle zone of the lungs. Work place activities such as cutting, grinding and polishing materials that contain respirable crystalline silica produce fine dusts and are associated with an increased risk of developing silicosis. With the advance of industrialisation, especially after introduction of pneumatic hammer drill and sandblasting, the prevalence of silicosis was increased significantly.[1] Still it is the most important occupational lung disease for the developing countries like India. Pathogenesis of silicosis is still not clear, but some believed the silica deposits could cause immune response, which was responsible for silicosis progression.[2,3] While some others believed the trace metals found on silica dusts played the major role in silicosis pathogenesis.[4,5] Risk of lung cancer is 3 folds more in silicosis than in general population.[6] Patients with silicosis are 20-30 folds more susceptible to pulmonary tuberculosis.[7,8]

After inhalation of the silica dust particles, the macrophages ingest them, release cytokines and initiate an inflammatory reaction and stimulate fibroblasts ultimately leading to fibrosis and formation of pulmonary nodular lesions. Silicosis develops slowly progressive shortness of breath in the involved persons. Other symptoms and signs include persistent by cough, tachypnoea, fatigue, weight loss, chest pain and fever. In advanced cases, there may be cyanosis, cor pulmonale and respiratory failure.

Diagnosis of silicosis depends on-

a. History of exposure.
b. Chest x-ray findings.
c. Exclusion of other illness.

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The classical silicosis is most common and usually manifests after 10-20 years of continuous silica exposure, during which time, the patient remains asymptomatic. Clinically, the onset of the disease is insidious and Total Lung Capacity (TLC) is reduced. An x-ray of chest shows "snow storm" appearance in the lung fields. Post-mortems on silicotuberculosis failed to prove the existence of tuberculosis, but showed them to be the cases of pure silicosis. The radiological evidence in two conditions (silicosis and tuberculosis) is so similar that one is apt to mistake a case of silicotic to be a case of tuberculosis of lungs.[9] In few cases, thin sheets of calcification occur in the hilar lymph nodes. Radiologically, these are eggshell calcification, which is characteristic, but not diagnostic of silicosis.[10,11]

There is no effective treatment for silicosis. Fibrotic changes is irreversible and T/t is mainly symptomatic. Further exposure to silica dust must be restricted. The silicosis can be controlled by-

a) Rigorous dust control measures and b) Regular physical examination of workers.[12] Lung transplantation is the most effective T/t, but caries high risk.

DISCUSSION OF MANAGEMENT

A fair number of people in Jharkhand are exposed to silica dust in their working places. Silicosis is caused by inhalation of crystalline silicon dioxide for several years. Inhaled silica particles are engulfed by alveolar macrophages and starts an inflammatory response by releasing TNF (tumour necrosis factors), interleukin-1, leukotriene B4 and other cytokines (various cytokines). The interleukin-4 and the growth factors stimulate fibroblasts to proliferate and produce collagen around the silica particle, thus resulting in fibrosis and the formation of nodular lesions.[3,4]

In this report, our patient with a typical occupational history of stone mining for a decade and chest imaging result was diagnosed silicosis by definite histopathology result.

The occupational history of exposure to silica dusts, progressive nature of breathlessness and the classical chest x-ray findings are the main clues for the diagnosis.[13]

The natural history of the disease varies greatly even in chronic silicosis. One study revealed that progression of the disease was more likely to occur if exposure continued for more than 2 years. The reported case had history of working as a stone miner for more than 20 years developed shortness of breath 10 years back and continued the job till death. Exposures to low levels of crystalline silica dust are not felt to produce clinically significant lung disease. Silicosis is not permanently cured, so preventive measures are the principal support of management. The World Health Organization (WHO) recommends all workers exposed to crystalline silica should undergo lifelong health surveillance. They advise that a plain chest x-ray should be obtained at the start of service and then at the regular interval, e.g. every 2-5 years. Tight enforcement and fidelity to dust control measures remain the best method of preventing the development of silicosis. Water spray and dry air filtering can control dust.

In USA, protective measures such as respirators have brought a steady decline in death rates due to silicosis. But, in our country, this is not true, where working condition are poor and respiratory equipment is seldom used.

Those who are exposed to the rise of silicosis can use protective masks. If silicosis is diagnosed in the early stage, the worker should be protected from further exposure to prevent further progression and the development of complicated silicosis.[14]

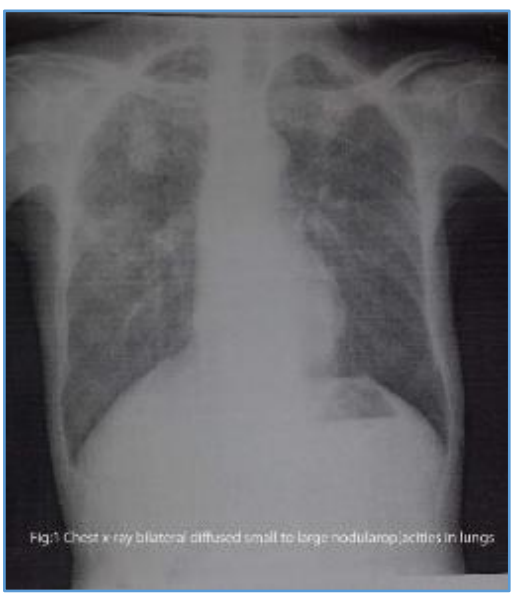

Figure 1. Chest X-Ray, Bilateral Diffuse Small-to-Large Nodular Opacities in Lungs

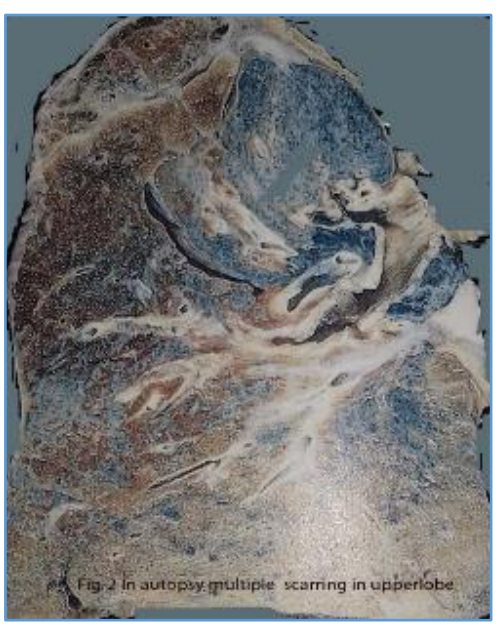

Figure 2. In Autopsy, Multiple Scarring in Upper Lobe

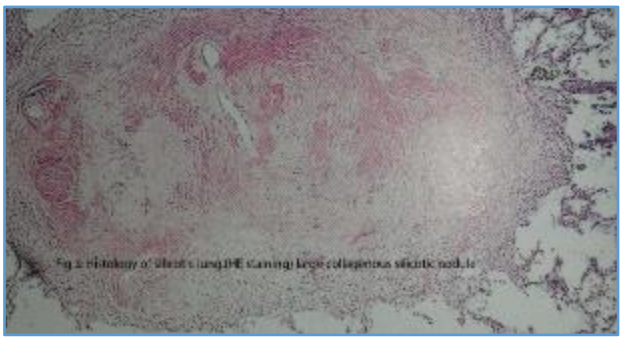

Figure 3. Histology of Silicotic Lung (HE Staining) Large Collagenous Silicotic Nodule

FINAL DIAGNOSIS

According to the history, clinical findings, laboratory investigations and the reports of autopsy and histopathological examination (microscopic examination of lung tissue), the final diagnosis of the patient is "chronic complicated silicosis with COPD" and the patient died due to respiratory failure and cor pulmonale.

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