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Case Report

Silicosis - A Case Report

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Abstract

Silicosis is not an uncommon disease in Bangladesh as a good number of people are exposed to silica dust in their working places. Again pulmonary tuberculosis is also common here, the risk of which is increased by about 30 folds in silicosis. In the reported case, a young stone cutter of 40 years was admitted to Faridpur Medical College Hospital with progressive dyspnoea, dry cough and radiological appearance of multiple small and a large nodular pulmonary mass. Two of his brothers, also stonecutter, died of similar disease. In this context the patient was diagnosed as a case of chronic complicated silicosis and treated symptomatically as there is no curative treatment. A good number of people are engaged in stone cutting in our country including Faridpur district and are vulnerable to this progressive and non-curable disease. To aim of this case report is to make those people to be aware about the condition, so that they can protect themselves by taking appropriate measures (i.e. using protective mask) and should monitored their condition by regular chest x-ray. If early signs of silicosis is detected the worker should changed their job.

Introduction

Silicosis, a pneumoconiosis, is an occupational lung disease caused by inhalation of crystalline silica dust. It is marked by inflammation and scarring in the form of nodular lesions especially in the upper lobes of the lungs. Respiratory problems arising from breathing in dust in miners was known to ancient Greeks and Romans[1].Visconti in 1870 originally used the term `silicosis' from Latin Silex or Flint. With the advancement of industrialization, especially after introduction of pneumatic hammer drill and sandblasting the prevalence of silicosis was increased significantly[2]. Now-a-days although the disease is found less commonly in developed world, it continues to be an important problem for the developing countries like Bangladesh.

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The risk of developing silicosis is determined by the lung dust burden dependant further on the intensity, nature and duration of exposure. Depending on these factors different types of silicosis have been described[3]. "Chronic simple silicosis", the commonest form, results from long term exposure usually appearing 10-30 years after exposure[4]. Chronic cough and exertional dyspnoea are common symptoms. Chest X-ray reveals profuse small mottled opacities predominantly in the upper lung zones. "Accelerated silicosis" develops 5-10 years after exposure, progresses rapidly and is at higher risk for complicated disease. "Acute silicosis" also called Silicoproteinosis develops a few weeks to 5 years after exposure to high concentration of silica dust. Rapid onset of severe dyspnoea, cough and ground-glass chest x-ray appearance are the features of acute silicosis which may lead rapidly to death. When severe scarring leads to confluence of small nodules into a larger lesion with more severe symptoms and respiratory impairment, it is termed as "Complicated silicosis". It is more common in accelerated type than with the chronic variety.

Silicosis may be complicated with other lung diseases including lung cancer and autoimmune diseases. Patients with silicosis are 10-30 fold more susceptible to pulmonary tuburculosis known as "silicotuberculosis"[5]. The exact reason is not known but may be due to damage to the macrophages by silica causing inhibition of their ability to kill mycobacterium. Other pulmonary complications include chronic bronchitis, non-tuberculous

mycobacterial infection, fungal infection, emphysema and pneumothorax. Risk of lung cancer is 2-4 fold more in silicosis patients than in general population[6]. Some data reveals an association between silicosis and certain autoimmune diseases like nephritis, scleroderma and SLE especially in acute and accelerated variety of silicosis. When silica dust particles are deposited in the lung, macrophages ingest it, release cytokines, and initiate an inflammatory response and stimulate fibroblasts ultimately leading to fibrosis and formation of pulmonary nodular lesions. Slowly developing progressive shortness of breath is the main symptom of chronic silicosis. Other symptoms and signs include persistent 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 history of exposure to sufficient silica dust, chest x-ray findings consistent with silicosis and exclusion of other illnesses causing similar abnormalities[7]. Physical findings are unremarkable and nonspecific in uncomplicated cases. Pulmonary Function Tests may be normal or obstructive and/or restrictive abnormalities may be present. Chest x-ray reveals small nodules (<10 mm) in early stage that later coalesce to form larger nodule (>1cm) especially in upper lung zone. In 5-10% cases, hilar nodes may calcify producing so called "eggshell" calcification, which is strongly suggestive, although not pathognomonic of silicosis. Tissue biopsy is not usually needed for diagnosis except in certain cases to exclude other conditions. CT scan is sometimes done for more detail findings of the lung lesion. Silicosis is an irreversible condition with no cure. Currently treatment is mainly symptomatic with antibiotics, bronchodilator, cough suppressant, anti-tuberculous drug, oxygen and physiotherapy- as and when appropriate. Further exposure to silica dust, other irritants and smoking must be prohibited.

Lung transplantation is the most effective treatment but carries high risk. Corticosteroid therapy, inhalational Aluminium, D-penicillamin is some of the experimental treatment.

Case Report

A young man of 40 years, nonsmoker, nondiabetic, normotensive, was a stonecutter by profession, hailing from Madaripur was admitted to FMCH with the complains of progressive shortness of breath for 10 years, dry cough for 7 months and generalized weakness for 7 months. Initially he had exertional dyspnoea but for last one year he has dyspnoea even at rest. He received 9-months anti-TB drugs 5 years back without significant improvement. He developed swelling of legs on several occasions that subsided with diuretics. While he was a stonecutter, he used to work approximately 10 hours in a day for 20 years then he changed the occupation for the last 7 years and working in a grosser shop. Two of his elder brothers, also stonecutter, died of similar illness few years back.

On physical examination, he was ill-looking, cachectic, mildly anaemic and non-oedematous. Chest examination revealed evidence of consolidation on the

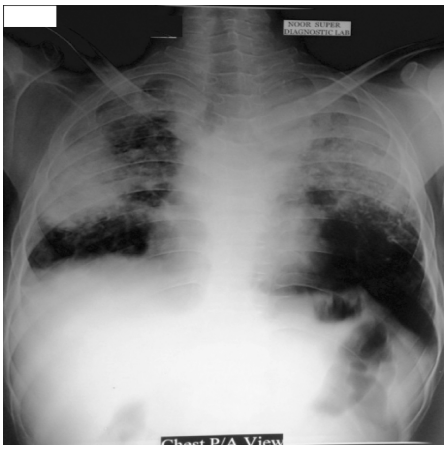

Figure 1: Chest X ray P/A view, Bilateral mottled opacities and a large nodular mass in the right upper and mid zone

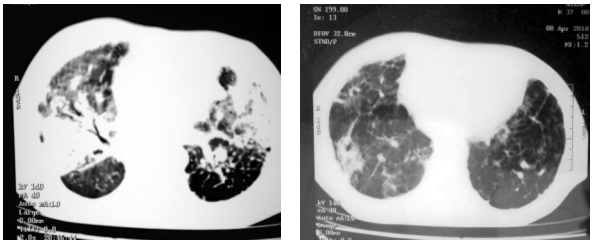

Figure 2: CT scan of the chest, bilateral pulmonary fibrotic masses, larger on right side, predominantly involving upper and mid zones and bilateral pulmonary thickening.

right upper chest with few rhonchi all over the chest. Investigations revealed Hb 60%, ESR 45mm in the 1st hour, TC 9700/cu-mm, x-ray chest showed bilateral mottled opacities and a large nodular mass in the right upper and mid zone. His oxygen saturation was 96% on pulse oximetry. Pulmonary Function Test showed mixed obstructive and restrictive type of abnormalities. Tuberculin test and sputum for AFB were negative. CT scan of the chest showed bilateral pulmonary fibrotic masses, larger on right side, fibrosis is predominantly at upper and mid zones, pulmonary thickening was seen on both sides. With the above findings, the patient was diagnosed as a case of "Chronic Complicated Silicosis".

As there is no specific treatment, the patient was managed symptomatically with antibiotic, bronchodilator and low flow oxygen. With this treatment the patient improvement was not significant. The progressive nature of the disease was discussed with the patient and he was discharged with advice for further follow up.

Discussion

A good number of people in Bangladesh are exposed to silica dust in their working places especially in stonecutting, brick-making, ceramic and other industries. Although there is no definite statistics in Bangladesh, it appears from clinical experience that silicosis is not uncommon in our country[8]. As pulmonary TB is also common many of the silicosis cases may be misdiagnosed as pulmonary TB. Moreover increased frequency of tuberculosis in silicosis patients complicates the situation further. In this reported case the patient also received a course of anti-TB treatment without satisfactory improvement. It may be that his occupational history was not considered with due importance at the time of diagnosis of tuberculosis.

The occupational history of exposure to silica dusts, progressive nature of breathlessness and the classical radiological findings are the main clues for the diagnosis[7]. Exclusion of other conditions especially pulmonary tuberculosis is important before confirmatory diagnosis of silicosis is made. In this patient pulmonary tuberculosis was excluded by relevant investigations and he had almost all other points in favor of silicosis. In complicated silicosis sometimes bronchoscopic specimen is studied for detection of mycobacteria. In selected cases of mixed dust exposure or suspicion of malignancy, lung biopsy may also be considered[9].

The natural history of the disease varies greatly even in chronic silicosis. One study[10] revealed that progression of disease was more likely to occur if exposure continued for more than 02 years after the earliest radiological abnormality was detected than in those where exposure was discontinued within 02 years of detection. The reported patient had history of working as a stonecutter for more than 20 years, developed shortness of breath 10 years back and continued the job for further 03 years. Despite leaving stonecutting his symptoms progressed relentlessly over the next 07 years, later complicated by appearance of large pulmonary nodule and cor-pulmonale. He had no history of hemoptysis or chest pain, no clubbing, the progression of the disease continued for longer period-all making the chance of any malignancy less likely. Silicosis, although mostly an occupational lung disease, a non-occupational form has also been described caused by long term exposure to sand dust in desert areas, with cases reported form Sahara, Libyan desert and the Neger[11]. The disease is caused by deposition of sand dust in the lung. Brief or causal exposures to low levels of crystalline silica dust are not felt to produce clinically significant lung disease.

As there is no cure, preventive measures are the mainstay of management. The best way to prevent Silicosis is to identify working place activities that produce respirable silica dust and then to eliminate or control the dust. Water spray and dry air filtering can also control dust. In western countries, protective measures such as respirators have brought a steady decline in death rates due to silicosis. Unfortunately, this is not true for less developed countries like Bangladesh, where Working conditions are poor and respiratory equipment is seldom used. . Those who are exposed to the risk of silicosis can use protective masks and should be monitored by chest radiography at regular interval e.g. every four years. If early signs of silicosis are detected, the worker should be protected from further exposure to prevent further progression and the development of complicated disease[12].

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