Case Report

Silicosis in a housewife

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ABSTRACT

Silicosis is an occupational respiratory disease caused by inhaling respirable crystalline silica dust. We report the case of silicosis in a 50-year-old woman that presented with pulmonary fibrosis and later developed pulmonary tuberculosis. Her condition is believed to be due to inhalation of dust produced by scrubbing a silica rich surface during usual daily housework

Saudi Med J 2003; Vol. 24 (1): 101-103

Silicosis is usually an occupational lung disease. It is irreversible, often progressive and potentially fatal.[1] The disease occurs as a consequence of cumulative exposure to crystalline silica dust over the years. It may result from accidental exposure to silica dust or silica containing commercial products.[2,3] We present the case of silicosis in a 50-years-old housewife. The clinical, radiological and histopathological features are describe. Various forms of silicosis are discuss.

Case Report. A 50-years-old Afghani housewife was referred to the Chest clinic complaining of dry cough and progressive dyspnea on exertion. Her symptoms started when she came to live in the United Arab Emirates (UAE) one year previously. She had 6 years history of arthritic pains and diabetes mellitus. She is a non-smoker. She immigrated from "Khost" in Afghanistan to Pakistan and lived there for 12 years before moving to the UAE. Her clinical examination was unremarkable except for being obese and diminished chest expansion. Blood tests revealed hemoglobulin 15.8 g/dl, white cell count 12,400/cmm, neutrophils 54%, lymphocytes 39%, monocytes 4%, eosinophils 3%. Urea and electrolytes, serum transaminases, bilirubin, lactate dehydrogenase, total proteins/albumin, calcium, phosphorus were all within normal limits. Blood sugar 14 mmol/lt. Hepatitis serology, rheumatoid factor and anti-native DNA antibodies were negative. Sputum for acid-fast bacillus (AFB) x3 was negative on direct microscopy and culture. Sputum cytology was negative. Blood gases on room air pH 7.43, Pco2 35 mm Hg, Po2 60 mm Hg, and electrocardiogram showed sinus tachycardia at 120/minute, left axis deviation and poor R-wave progression. The x-ray of the knees revealed osteoarthritic changes. Chest x-ray and computed tomography (CT) chest showed prominent pulmonary arteries, bilateral hilar lymph nodes enlargement **(Figure 1)**, nodular and diffuse reticular infiltrates most prominent on the midfields and basally **(Figure 2)**. Pulmonary functions tests showed forced vital capacity (FVC) 2.2L (2.88), forced expiratory volume in one second (FEV1) 1.5L (2.36), FEV1/FVC 68% (82%), no response to salbutamol nebulization, indicating obstructive lung disease (equipment for total lung capacity, functional residual capacity and residual volume was not available). Bronchoscopy was negative except for black discoloration throughout the visualized bronchial tree. Transbronchial lung biopsy was normal except for multiple dark pigments; lavage was negative for AFB and malignant cells. On open lung biopsy, the entire left lung was extremely loaded with black pigments. A piece of the lingula and the largest 2 lymph nodes were removed. Histological examination of the lung and lymph nodes revealed moderate to marked

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Received 22nd April 2002. Accepted for publication in final form 7th September 2002.

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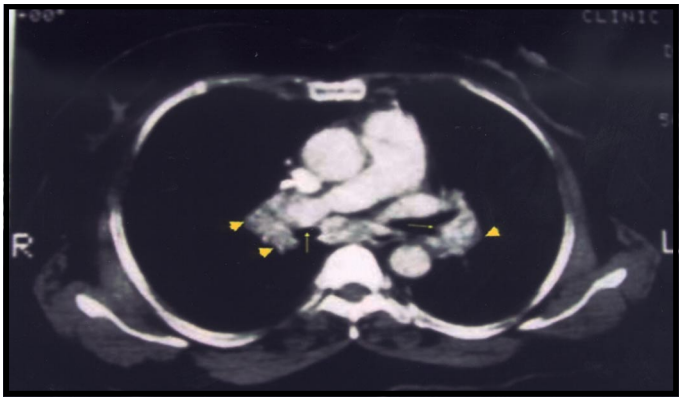

Figure 1 - Computed tomography chest mediastinal window. Bulky hilar areas bilaterally, more marked on the right. Arrow head pointing to enlarged hilar lymph nodes. Arrows pointing to pulmonary arteries.

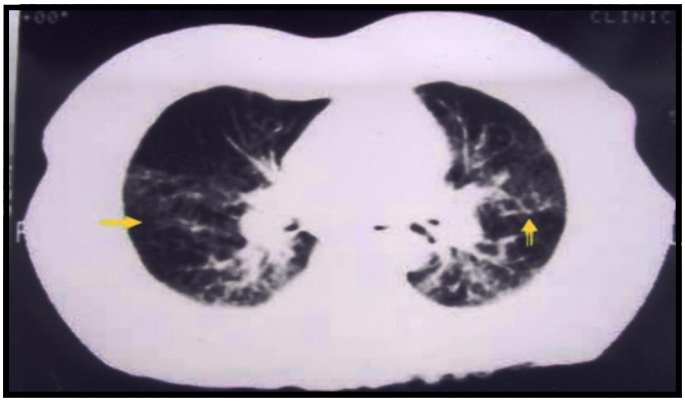

Figure 2 - Computed tomography chest - nodular and diffuse reticular infiltrates most prominent in the midfields and basally, marked by arrows.

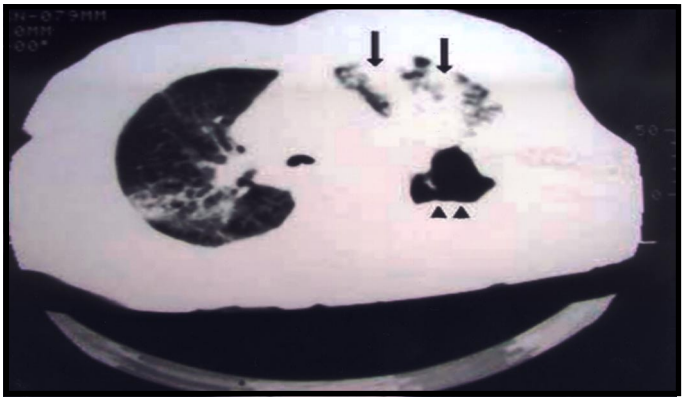

Figure 3 - Computed tomography chest consolidation in the left upper lobe marked by arrows and a large cavity marked by arrow heads. Note also increased interstitial shadowing on the right (see text).

fibrosis, multiple foci showing whorls appearance with hyalinization and marked anthracosis (macrophages laden with pigments). Birefringent silica particles seen on polarized microscopy, no evidence of granuloma or malignancy. The patient was diagnosed to have chronic silicosis with symptoms developing 12 years after cessation of exposure. She was questioned regarding exposure to silica and carbon particles. She reported that when she was in Afghanistan, since her childhood and until she left 13 years previously, she lived in a hut made of mud. Daily she cleaned the floor and the walls of the hut by scrubbing them with a hard brush. All her cooking was on wood fires. She never used coal.

Eighteen months after presentation she developed fever, and her cough became productive. Chest x-ray revealed necrotic area in the left upper lobe not noted on a previous study 4 months previously. The CT chest confirmed a large cavity plus consolidation in the left upper lobe and progression of fibrosis bilaterally **(Figure 3)**. Sputum was positive for mycobacterium tuberculosis sensitive to first line drugs. She received isoniazid (INH), Rifampicin, and Pyrazinamide for a period of 4 months (until the sputum became negative for AFB). This was followed by INH and Rifampicin for 6 months. The patient moved to another area, where, one month later she developed hemoptysis and was restarted there on anti-TB treatment for another year. Her respiratory functions, however, continued to deteriorate and she died from respiratory failure 3 years later.

Discussion. Silicosis is an occupational respiratory disease caused by inhaling respirable crystalline silica dust. Silicosis is irreversible, often progressive (even after exposure has ceased), and potentially fatal.[1] It is associated with higher incidence of mycobacterium tuberculosis infection, which developed in this patient.[2] Silicosis occurrence is predictable among people exposed to silica dust in various industries and occupations such as mining, construction, manufacturing and building maintenance.[1] However, patients are inadvertently exposed to the inhalation of silica dust when using silica rich domestic cleaning powder,[2] through ingestion of silica-containing natural health products[3] and in this patient, who was naïve of the dangers of scrubbing a probable silica rich surface and what was, to her, ordinary housework. It is associated with higher incidence of mycobacterium tuberculosis infection, which developed in this patients.[4] Silicon dioxide or silica is one of the most abundant materials in the earth's crust. The most frequent naturally occurring form of crystalline silica is quartz, a compound of many rock types. Sandstone may contain almost 100% quartz, slate and shale up to 40% and granite 10-30%. Smectite clays (for example fuller's earth) may contain up to 10% of quartz. A hazard to health should always be suspected whenever dust containing free silica is likely to be liberated into the air. Indians making slate pencils[5] contract silicosis, so do Nigerians carving sand stone grinding wheels.[6]

Silicosis was described in 3 different types: chronic, accelerated and acute. The type of presentation depends on the length and intensity of exposure. Chronic silicosis is the most usual form of silicosis, occurring after many years of exposure to relatively low levels of dust. It is usually a progressive disease, even in the absence of further dust exposure. The main symptom is dyspnea, which is progressive and ultimately disabling, ending in respiratory failure, cough and sputum production are also common. Radiologically it starts as a simple nodular form, which frequently develops into progressive massive fibrosis. Accelerated silicosis[7] is due to exposure to a high concentration of silica over a period of as little as 5 years resulting in a more rapidly progressive disease. Symptoms present early, and the patient deteriorates rapidly to respiratory failure. While the acute form[8,9] occurs in subjects exposed to very high concentrations of silica over a period of as little as a few weeks. The presentation is that of progressive dyspnea, fever, cough and weight loss after a heavy short exposure to silica (few weeks to 5 years). Death occurs in hypoxic respiratory failure.

In conclusion, silicosis is relatively rare and is often initially misdiagnosed. The key to diagnosis is to take an occupational history in any patient, with diffuse radiological changes who has worked for several years in an environment where stone dust could get into the air, and should be suspected of having the disease. Confirmation by open lung biopsy is mandatory in atypical presentations or absent history of exposure. This patient's history, which was taken from the young as well as the senior members of her family, did not disclose any possible source of exposure to silica. Scrubbing the walls of her mud house was thought to be the most likely cause, however, this can be proven only through analysis of the mud for silica contents, which, was not possible to do. There was neither mining nor quarrying in that region nor any industries where slate or sandstones are used.

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Title: Silicosis of the lung in Saudi Arabia - Role of traditional occupations and environmental factors

Source: Saudi Med J 1991; Vol. 12 (5): 384-388

Abstract

As pneumoconiosis is usually linked with industrialized societies, we undertook a search for pneumoconiosis in Saudis engaged in traditional occupations. Three cases of advanced silicosis of the lung with progressive massive fibrosis were detected. In 2 cases, 2 patients were life-long diggers of wells, and one a grave-digger. The search also detected that 13 cases of desert lung (simple siliceous pneumoconiosis); 11 of the patients were females engaged in traditional women's chores like dusting tents. However, 2 patients were men with no such history, which supports the view that desert lung could be an environmental lung disease; the suspended dust in the periphery of the city of Riyadh, Saudi Arabia averaged 3810μg/m^3. Although desert lung was thought to be free of significant fibrinogenic activity, we document progressive massive fibrosis in desert lung by computerized tomography scanning. Finally, 10 of the 11 women with desert lung had cataract of the eye which lends support to the previously described association between the 2 conditions. The basis for such an association is discussed.