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Case Study

Silicosis Caused by Sandblasting of Jeans in Turkey: A Report of Two Concomitant Cases

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Silicosis is a fibrotic disease of the lungs caused by inhalation, retention, and pulmonary reaction to crystalline silica. Crystalline silica exposure is widespread, and silica sand is an inexpensive and versatile component of many manufacturing processes[1)]. Its use and the incidence of silicosis are increased in industrial operations by the mechanization and the use of sandblasting, drilling, pulverizing, cutting, grinding tools and other pneumatic equipment[2)].

Sandblasting involves forcefully projecting a stream of abrasive particles onto a surface, usually with compressed air or steam. Mostly, silica sand is forced by compressed air onto the target surface. It has been commonly used in abrading metal or glass[3)]. Recently, it has been widely and increasingly used in sandblasting of jeans in Turkey. In this report, we present two concomitant cases of silicosis occurring in the same workplace, located in Istanbul, and caused by sandblasting of jeans. To our knowledge, there is no previous description of silicosis in such an occupation.

Case Reports

Case 1

A 19-yr-old man was admitted to our hospital in August 2004 with a history of 3 months of malaise, dry cough, sweating and loss of weight. A month prior to the admission he had been admitted to the local government hospital with these complaints, and tuberculosis (TB) treatment had been given without any bacteriologic proof. On admission he was continuing to take anti-tuberculous medication. He also reported he did not have any benefit from the medication and had had dyspnea for a week. He had no smoking history. He initially stated he had worked in a textile factory between March 2000 and November 2003. When he was asked to detail his work, he explained that he had worked in a small-scale workplace producing sandblasted jeans. Physical examination showed a non-productive cough, fever (38°C), dyspnea, tachypnea, and bibasilar coarse crackles. His initial investigation revealed WBC of 11,300 × 10^6 cells per liter, an erythrocyte sedimentation rate (ESR) of 55 mm/h, an albumin level of 2.7 mg/d*l* (normal range: 3.5–5.5) a CRP level of 21.8 mg/d*l* (upper limit: 0.5), and a LDH level of 2,103 U/m*l* (normal range: 250–500). On hospital admission arterial blood gas values were pH=7.53, arterial oxygen tension (PaO_2) 49 mmHg (FIO_2=0.21), arterial carbon dioxide tension ($PaCO_2$) 21.8 mmHg and percent of arterial oxygen saturation (SaO_2) 70.6%. Pulmonary function testing (PFT) was not performed because of TB or other infection suspicion. Sputum smears for TB and blood cultures were negative. Other laboratory tests were unremarkable. Chest radiography showed bilateral reticulonodular infiltrates (Fig. 1a). High resolution computer tomography (HRCT) revealed diffuse intralobular micronodules, which were prominent in the mid-to-lower lung zones (Fig. 2a, 2b and 2c). The patient underwent bronchoscopy with BAL and TBB, but no clue was identified. Anti-tuberculous treatment was stopped due to negative confirmation of the diagnosis and no substantial improvement of his symptoms, and antibiotic treatment with levofloxacin 500 mg once daily was initiated empirically due to clinical findings of infection. Although the findings of infection after the treatment were partially improved, the respiratory symptoms continued to worsen. An open lung biopsy specimen lead us to the diagnosis of silicosis (Fig. 3a).

Case 2

An 18-yr-old non-smoking man was admitted to our hospital in September 2004 with a history of dyspnea for 4 yr. His symptoms had started a short time after beginning work in the same workplace as Case 1. He had worked in that place between March 2000 and December 2003 years. Later, non-productive coughing, malaise, and weight loss had developed. He was seen by a physician on two occasions, May 2004 and August 2004, and bronchodilator drugs were prescribed. However, the diagnosis was not proven nor was there any benefit from the medications. Physical examination showed a non-productive cough, dyspnea, and fine crackles at the end of inspiration. Chest radiography showed diffuse interstitial infiltrates (Fig. 1b). HRCT revealed diffuse intralobular micronodules in the upper zones and fibrotic changes, which were prominent in the mid-to-lower zones of the lung, with bilateral minimal pneumothorax (Fig. 2d, 2e and 2f). The laboratory tests revealed an ESR of 20 mm/h and increased LDH levels of 1,426 U/m*l*. Sputum smears for tuberculosis were negative. Other laboratory tests were unremarkable. On hospital

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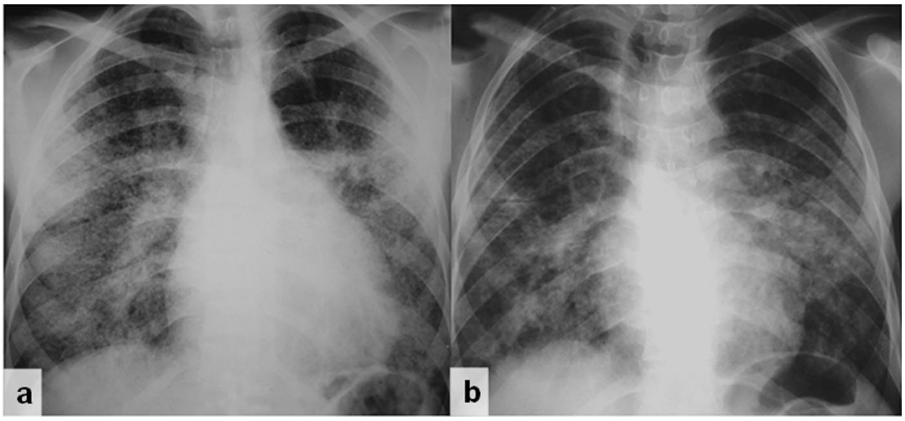

Fig. 1. Chest X-ray images of the patients showing bilateral interstitial infiltrates, which are prominent in the mid-to-lower zones. (a) Case 1, (b) Case 2.

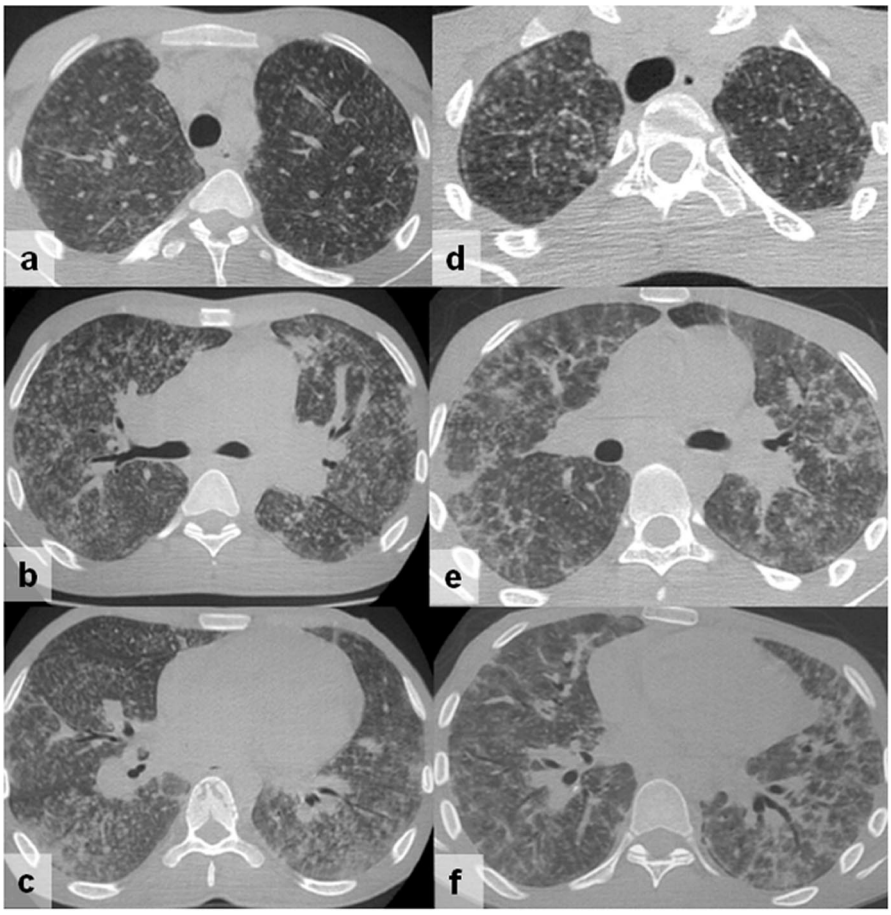

Fig. 2. HRCT images of the cases. Diffuse intralobular micronodules are prominent in the mid and lower zones (a, b and c). Intralobular micronodules in the upper zones and fibrotic changes in the mid-to-lower lung zones with bilateral minimal pneumothorax (d, e and f).

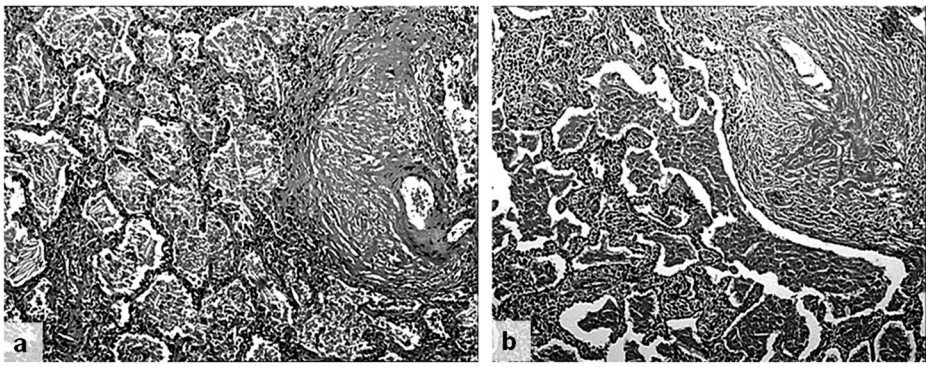

Fig. 3. Open lung biopsy specimens taken from the middle lobe of the cases. (a) A silicotic nodule in the lung is seen here. It is composed mainly of bundles of interlaced collagen. There is a minimal inflammatory reaction. A lesion characterized by the generalized accumulation of lipoproteinaceous material within the alveoli (H&E × 40). (b) It shows exudative alveolar lipoprotenosis associated with chronic inflammation. Alveolar septa are usually thickened with hypertrophic and hyperplastic type II epithelial cells (H&E × 100).

admission arterial blood gas values were pH=7.35, PaO_2=33.7 mmHg (FIO_2=0.21), $PaCO_2$=45.9 mmHg and SaO_2=61.6%. PFT revealed the following (as % predicted): FVC 53%, FEV_1 69%, FEV_1/FVC 96%. The diagnosis of silicosis was confirmed with histopathological examination of an open lung biopsy specimen as for Case 1 (Fig. 3b).

Discussion

The clinical forms of silicosis have been well characterized. These clinical and pathologic expressions of the disease reflect differing exposure intensities, latency periods, and natural histories. The chronic or classical form usually follows one or more decades of exposure to respirable dust containing quartz. The accelerated form results from heavier exposures, often with a duration of 5 to 10 yr. The acute form of silicosis, as in our cases, is a consequence of intense exposure to high levels of respirable dust that contain a significant proportion of silica. The reported exposure period is usually from several months up to about 5 yr[1]. In our cases, the exposure time was less than 5 yr and the clinical course had a rapid progression. Although the two cases had left work approximately a year ago, it is known that the development and progression of silicosis frequently occurs after exposure has ceased[1].

A wide variety of industries are associated with the generation of particulate aerosols with sufficient silica content to induce silicosis. The occupations causing silicosis are well known but they continue to change over time[4]. New reports of silicosis in industries and work settings not previously recognized to be at risk continue to occur[5].

In industrial countries, the disease is well recognized and is adequately prevented by ambient dust control measures. Nevertheless, it continues to occur in developing countries, even developed countries, because of lack of efficient protective measures. Sandblasting, one of the most important risk factors of silicosis, generates respirable aerosols of silica and is associated with a high risk for silicosis even when respiratory protection is used[6]. Sandblasting is still used in many applications where abrasive cleaning of surfaces is required. Although European countries restricted the use of silica for sandblasting 40 yr ago[7], it is widely used in different workplaces in Turkey since quartz can be provided easily and cheaply[3].

Because of the increased popularity of sandblasted jeans, their production is rapidly increasing. It is estimated that there are many small-scale workplaces, which would operate as contractors for bigger companies, producing sandblasted clothes in Turkey. According to information taken from our patients, there were more than 20 similar workplaces with an average of 20 workers only in Istanbul which is one of the most prominent cities in Turkish industry, especially in textiles. Most of their co-workers were as young as the two cases described. They had both worked in a closed and poorly ventilated workplace without any protection other than a simple face mask for 11 h per day. Although we had no opportunity to measure the dust concentrations in their work area, it was assumued that it was similar to those measured in the study by Sevinc *et al.*[3] performed earlier in a different workplace in our country (116.42 ± 15.62 mg/m^3 and 76.45 ± 7.99 mg/m^3, mean respirable dust and free silica concentration, respectively).

There is no data showing the exact incidence of silicosis in our country. In our region, in the eastern part of the

country, there have been no reported silicosis cases because most of the industrial activity occurs in the western part of the country. However, most of the work force is composed of migrants from the eastern part. They not only work in uncontrolled and primitive conditions, but they also live in poor conditions. Sometimes the workers are admitted to our hospital because of their illness when they visit their families. The most common disease in such workers, especially in textile workers is tuberculosis. Its incidence is higher among those workers (19.8% of all diagnosed tuberculosis cases) than others[8].

In conclusion, the two incidentally concomitant cases show that many workers employed in sandblasting of jeans, work in uncontrolled and primitive conditions without protective measures. To our knowledge, these are the first reported cases among workers sandblasting jeans, however, the problem may be more hazardous than our estimates. Close attention and strict measures are urgently needed, especially for small-scale enterprises, to prevent the disease in Turkey. We have informed the relevant official institutions in order to initiate the necessary measures.

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