

AN UNUSUAL FORM OF SILICOSIS IN A STONE-CUTTER

S. A. Sharifian*, A. H. Mehrparvar and S. Mohammadi

Department of Occupational Medicine, School of Medicine, Medical Sciences/University of Tehran, Tehran, Iran

Abstract- Silicon dioxide or silica is the earth's most abundant mineral. The primary pulmonary illness attributable to silica exposure is silicosis, which is observed in three forms: classic, accelerated and acute. A case of silicosis is presented in this article who has been working in a stone-cutting plant. He had suffered from symptoms of silicosis one year after employment in this plant (acute form), but his radiologic findings were compatible with accelerated or chronic silicosis. One of the important occupational diseases is silicosis, which is not treatable but is preventable. However, because of lack of appropriate engineering controls or personal protective devices in some workplaces we observe cases of silicosis yet.

© 2007 Tehran University of Medical Sciences. All rights reserved.

Acta Medica Iranica, 45(2): 158-160; 2007

Key words: Silicosis, stone cutter, dyspnea

INTRODUCTION

Silicon dioxide or silica is the earth's most abundant mineral (1). The primary pulmonary illness attributable to silica exposure, silicosis (which is the most common pneumoconiosis worldwide), is caused by the inhalation of respirable size silica particles, and can be categorized by recognizable findings on the chest radiograph and also the time from initial exposure to silica into 3 types: simple or classic, accelerated or subacute and acute silicosis (1-3). Here we present a case of silicosis in a man working in a stone-cutting plant.

CASE REPORT

Our patient was a 40 years-old stone-cutter male living in a village near Hamadan (west of Iran). He was referred to our occupational medicine clinic in Tehran University of Medical Sciences with chief complaint of exertional dyspnea.

He had a progressive dyspnea beginning 2 years before referring to us (1 year after employment in a stone-cutting plant), with cough (first productive and then without sputum). When we visited him, he had dyspnea (functional class II to III) and non-productive cough. In his medical history he didn't have any other diseases and hospital admission, besides he was non-smoker.

Our patient had worked as a miller in a stone-cutting plant from 2002 till 2005 (14 hours a day). The product of this plant is used for glass-making. The plant has had a closed environment with general ventilation, and he has used only paper masks as a personal protective device. He mentioned that his 14 co-workers also had the same feature. He had also worked as a farmer (in a farm in which wheat, barley, and hay were planted) from 1991 till 2002.

He was found to be tachycardic (110 beats/min.), afebrile (37.2 °C oral), with respiratory rate of 22 breaths/min., and blood pressure of 110/80 mmHg. In chest auscultation, we heard a diffused coarse crackle bilaterally. He mentioned that he hasn't have any pre-employment examinations and radiography. He had normal chest radiography in 2002, but after 2 years his chest radiography revealed reticulonodular pattern and round opacities especially in upper lung zones (Fig. 1).

Received: 10 Jul. 2006, Revised: 20 Sep. 2006, Accepted: 20 Nov. 2006

* Corresponding Author:

A. H. Sharifian, Department of Occupational Medicine, School of Medicine, Medical Sciences/University of Tehran, Tehran, Iran
Tel: +98 21 66405588
Fax: +98 21 66405588
E-mail: ahmehrparvar@razi.tums.ac.ir



Fig. 1. Chest radiography of the case.

We performed pulmonary function tests for him, with following results: forced vital capacity (FVC): 1.5 lit (39% predicted), forced expiratory volume in 1 s (FEV₁): 1.07 lit (32% predicted), FEV₁/FVC: 73.6 (92% predicted), forced expiratory flow (FEF): 1.74 lit/s (21% predicted), forced expiratory flow between 25 and 75% of the vital capacity (FEF₂₅₋₇₅): 0.92 lit/s (22% predicted), total lung capacity (TLC): 2.7 lit (41% predicted).

Computed tomography (CT) of chest in 2004 revealed bilateral fibrocystic changes, especially in upper lobes, besides emphysematous changes and bilateral pleural thickening. High resolution computed tomography (HRCT) of chest in 2004 revealed fine nodules in upper lobes, besides a conglomerated mass in apex of right upper lobe (Fig. 2).

We obtained informed consent to publish details of our patient's history.

DISCUSSION

As mentioned, there are three forms of silicosis. Simple or classic silicosis results from low to moderate exposure to silica dust and have features usually develop slowly and frequently require a working lifetime to develop (20 years or more) (1-3). It typically appears as an upper zone distribution of rounded opacities (1, 2, 4). Accelerated or subacute silicosis is characterized by the same features as classic silicosis except that the time from initial exposure to silica is much shorter. The chest radiograph may demonstrate rounded opacities as early as 4 years after the first exposure (usually 5 to 10 years) (1, 2, 5).

Acute silicosis follows a short duration of exposure to overwhelmingly high concentrations of respirable free silica. The onset of symptoms usually is 1-3 years after the initial exposure (1, 2, 6). The chest radiograph typically reveals diffuse alveolar infiltration usually associated with air bronchograms (1, 2, 6-8). The alveoli are filled with lipid and proteinaceous exudative material (1, 2).

Our patient had the typical signs and symptoms of silicosis. He also had a pure severe restrictive pattern in pulmonary function tests. He was a stone-cutter. The changes in simple chest X ray and also CT and HRCT are completely compatible with classic or subacute silicosis (interstitial fibrosis), but the symptoms have begun only 1 year after exposure to silica dust, which is compatible with acute silicosis and he did not have any alveolar filling and infiltration as usually is seen in acute silicosis. So he is an unusual case of silicosis, with history of acute but radiography of classic silicosis.

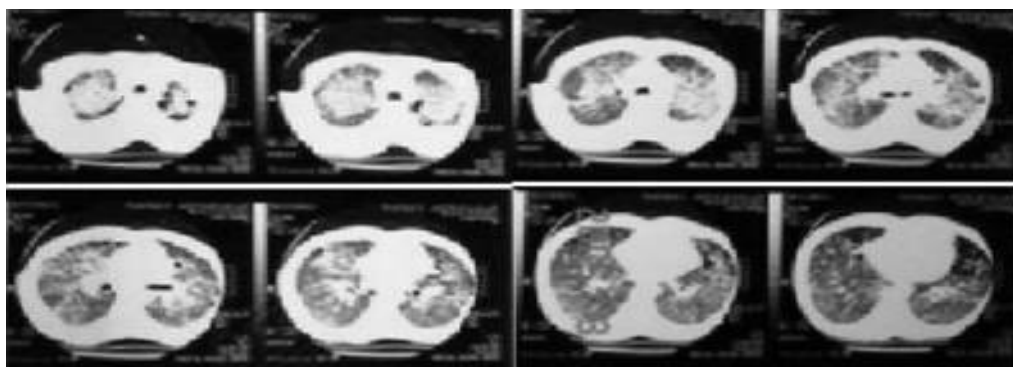


Fig. 2. Chest high resolution computed tomography of the case.

REFERENCES

1. James LF, Morris G, Youngblood, DA. Textbook of clinical occupational and environmental medicine. 2nd edition. Philadelphia: Elsevier Saunders; 2005.
2. Rom WN. Environmental and occupational medicine. 3rd edition, New York: Lippincott-Raven; 1997.
3. Castranova V, Vallyathan V. Silicosis and coal workers' pneumoconiosis. Environ Health Perspect. 2000 Aug; 108 Suppl 4:675-684.
4. Graham WG, Vacek PM, Morgan WK, Muir DC, Sisco-Cheng B. Radiographic abnormalities in long-tenure Vermont granite workers and the permissible exposure limit for crystalline silica. J Occup Environ Med. 2001 Apr; 43(4):412-417.
5. Seaton A, Legge JS, Henderson J, Kerr KM. Accelerated silicosis in Scottish stonemasons. Lancet. 1991 Feb 9; 337(8737): 341-344.
6. Lugano EM, Dauber JH, Daniele RP. Acute experimental silicosis. Lung morphology, histology, and macrophage chemotaxin secretion. Am J Pathol. 1982 Oct; 109(1): 27-36.
7. Bergin CJ, Muller NL, Vedal S, Chan-Yeung M. CT in silicosis: correlation with plain films and pulmonary function tests. AJR Am J Roentgenol. 1986 Mar; 146(3):477-483.
8. Talini D, Paggiaro PL, Falaschi F, Battolla L, Carrara M, Petrozzino M, Begliomini E, Bartolozzi C, Giuntini C. Chest radiography and high resolution computed tomography in the evaluation of workers exposed to silica dust: relation with functional findings. Occup Environ Med. 1995 Apr; 52(4):262-267.