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Silicotuberculosis and Silicosis as Occupational Diseases: Report of Two Cases

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SUMMARY

Introduction Silicosis, the most prevalent of the pneumoconioses, is caused by inhalation of crystalline silica particles. Silica-exposed workers are at increased risk for tuberculosis and other mycobacterium-related diseases. The risk of a patient with silicosis developing tuberculosis is higher (2.8 to 39 fold higher, depending on the severity of silicosis) than that found in healthy controls.

Outline of Cases The first patient was a 52-year-old male who was admitted in 2002 for the second time with dyspnoea, wheezing and fatigue over the last 11 years. He had worked in an iron smelting factory and was exposed to silica dust for 20 years. First hospitalization chest radiography showed bilateral pleural adhesions, diffuse lung fibrosis with signs of a specific lung process. Second hospitalization chest radiography showed bilateral massive irregular, non-homogenous calcified changes in the upper and middle parts of lungs. The patient died due to respiratory failure and chronic pulmonary heart in 2007. The main causes of his death were silicotuberculosis and chronic obstructive pulmonary disease. The second patient was a 50-year-old male who was admitted in 2005 for the second time with chest tightness, dyspnoea, wheezing and fatigue over the last 10 years. He had worked in an iron smelting factory and was exposed to silica dust for 30 years. First hospitalization chest radiography showed diffuse lung fibrosis and small nodular opacities. The patient was diagnosed with silicosis, small opacities sized level p/q, and profusion level 2/3. Second hospitalization chest radiography and CT showed diffuse lung fibrosis and small nodular opacities predominantly in the upper lobes. The patient was recognized as having an occupational disease, and received early retirement due to disability.

Conclusion In low-income countries, new cases of silicosis and associated lung cancer, chronic obstructive pulmonary disease and tuberculosis are likely to be seen for decades because necessary reduction of silica use will take time to be achieved.

Keywords: silica dust; silicosis; silicotuberculosis; occupational disease; fatal outcome

INTRODUCTION

Tuberculosis as a complication of silicosis has been a historical focus of attention over the last centuries [1, 2]. Beside that fact, there still persists a possible positive association between silicosis and lung cancer [3]. The association between silicosis and tuberculosis has been studied since the beginning of the twentieth century [4]. The risk of developing pulmonary tuberculosis is reported to be 2.8 to 39 fold higher for patients with silicosis than for healthy controls [4, 5, 6]. The risk of a patient with silicosis developing extra pulmonary tuberculosis is also as much as 3.7 times higher than in healthy controls [5]. The pleural form is most common, accounting for 61% of the cases, followed by the pericardial form and the lymph node form [5]. Regarding the relationship between mycobacterium-related diseases and different forms of silicosis, studies in the international literature have shown that the acute and accelerated forms present with the highest incidence [7]. In our two case reports, there is a chronic form of silicosis present, which develops more than 10 years after exposure, and is typically oligosymptomatic. However, it can evolve to progressive dyspnoea on exertion. In patients with the chronic form, the progression of the disease can be rapid, leading to death within a few months or years. From a histopathological point of view, silicosis is characterized by the presence of granulomas, with collagen nuclei surrounded by epithelioid cells,

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giving rise to silicotic nodules, which are diffusely distributed in the lungs and, with the progression of the disease, can coalesce and form large masses, distorting the parenchyma [2, 8]. In addition to its importance as an occupational disease, silicosis or even exposure to silica without established disease is associated with increased risk of developing various pulmonary and systemic co- morbidities. Higher prevalence of chronic obstructive pulmonary disease, lung cancer, tuberculosis, non-tuberculous mycobacterium-related diseases, glomerulonephritis, rheumatoid arthritis, scleroderma, and other autoimmune diseases have been documented among patients with silicosis [4, 9]. These two case reports focus on the association between silicosis and the development of tuberculosis, as well as on evaluation and appropriate preventive measures.

CASE REPORT 1

A 52-year-old male patient was admitted to our clinic in August 2002 for the second time. The first time of hospitalization was in October 1991. During the first hospitalization main complaints were chest pain, dyspnoea, fatigue and malaise lasting more than one year. During the second hospitalization his main complaints were dyspnoea, wheezing and fatigue over the last 11 years. He had worked in an iron smelting factory for 34 years but his exposure to dust and silica was 20 years. He had smoked 20 cigarettes per day for the last 30 years. His physical examination revealed blood pressure of 120/75 mm Hg, pulse rate of 80/min. The only abnormal physical examination finding was bilaterally decreased breath sounds. Pulmonary function tests revealed very severe obstruction (Table 1). His blood analysis results are also shown in Table 1.

Chest radiography (PA and tomography) performed during the first hospitalization (Figure 1A) showed bilateral pleural adhesions and diffuse lung fibrosis with signs of specific lung process. These findings led to the recommendation of treatment with antituberculotic drugs, which unfortunately proved to be ineffective in this case. Chest radiography during the second hospitalization (Figure 1B) showed bilateral massive irregular and non-homogenous calcified changes in the upper parts and partially in the middle parts of lungs. Both hiluses presented more volume and signs of enlarged paratracheal and peribronchial lymph glands.

Table 1. First and second hospitalization pulmonary function tests, sedimentation rate, leukocyte counts of case report patients – progression of the disease

Parameter	Case 1 patient		Case 2 patient	
	1st H	2nd H	1st H	2nd H
FVC (L)	4.69	2.09	4.34	4.59
FEV_1 (L)	3.82	1.00	3.36	3.35
PEF (L/s)	11.20	2.37	6.98	6.10
FEV_1/FVC (%)	82	48	77	73
Leukocyte count ($\times 10^9$/L)	5.4	5.0	6.2	8.4
Sedimentation rate (mm/h)	15	24	20	22

1st H – first hospitalization; 2nd H – second hospitalization

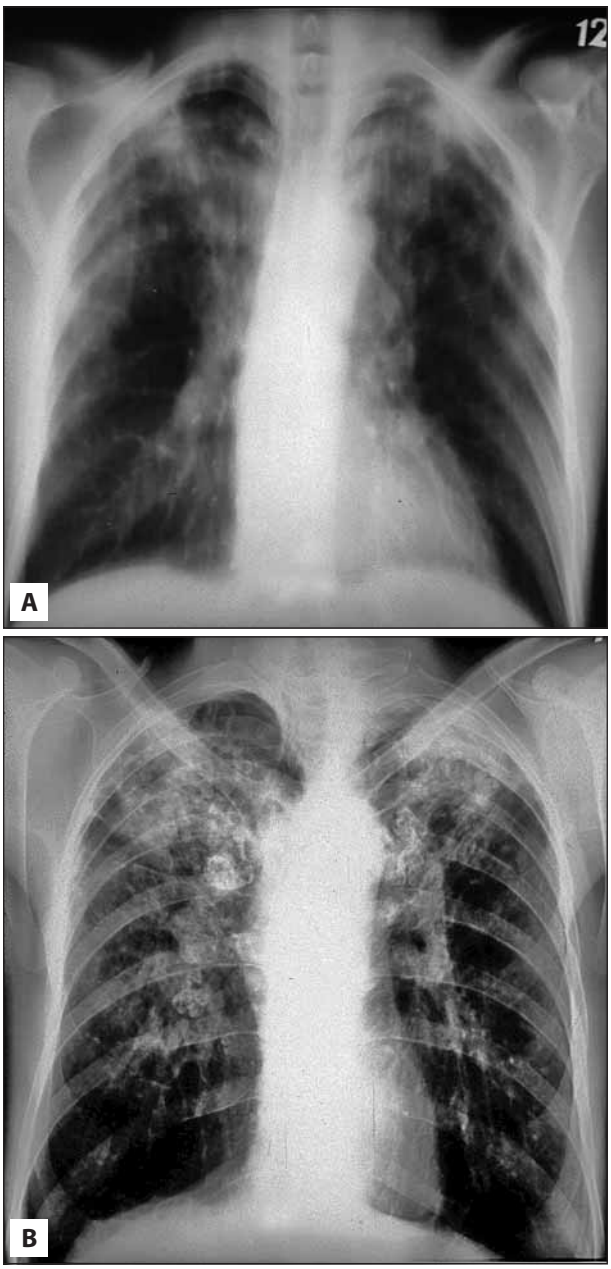

Figure 1. Chest X-ray (PA) of case report 1 patient: A. First hospitalization (1991) – bilateral pleural adhesions and diffuse lung fibrosis with signs of specific lung process; B. Second hospitalization (2002) – bilateral massive irregular, non-homogenous calcified changes in upper and middle parts of lungs

The patient died due to respiratory failure and chronic pulmonary heart in 2007. After his death his family went to court demanding material compensation from the iron smelting factory he worked for over 30 years. Official court medical experts stated that the main causes of his death were silicotuberculosis and chronic obstructive pulmonary disease.

CASE REPORT 2

A 50-year-old male patient was admitted to our clinic in June 2005 for the second time. First time of hospitalization was in August 2003. During the first hospitalization

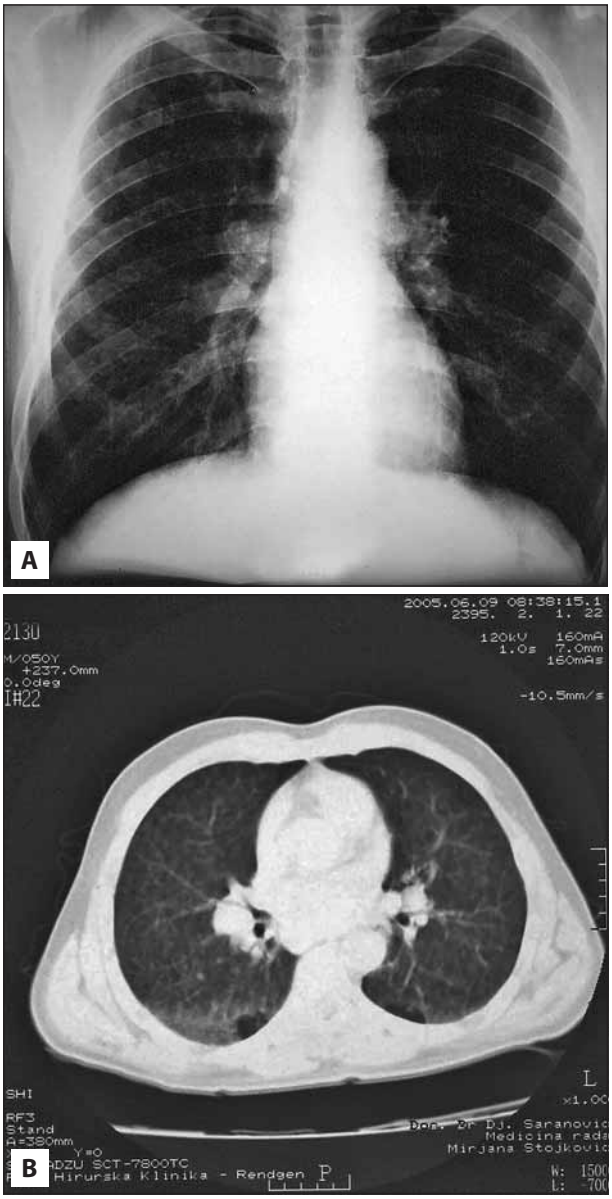

Figure 2. A.Chest X-ray (PA) of case report 2 patient (2005) – bilateral diffuse lung fibrosis and small nodular opacities; B. Chest CT of case report 2 patient (2005) – diffuse micro-nodular changes predominantly in upper lobes, interstitial fibrosis

main complaints were chest tightness, cough, dyspnoea, fatigue lasting more than eight years. During the second hospitalization his main complaints were chest tightness, dyspnoea, wheezing and fatigue over the last 10 years. He had worked in an iron smelting factory for 30 years and had been exposed to dust and silica for 30 years. He had smoked 10 cigarettes per day for last 20 years. His physical examination revealed blood pressure of 120/80 mm Hg, pulse rate of 72/min. Abnormal physical examination findings included bilaterally intensified breathing sounds with prolonged expiration. Pulmonary function tests revealed no signs of respiratory insufficiency, except lowered forced expiratory flow in small respiratory airways (Table 1). His blood analysis results revealed no pathological findings (Table 1).

Chest radiography (PA) performed during the first hospitalization showed bilateral diffuse lung fibrosis and small nodular opacities indicating an occupation-specific lung process. Using the International Classification of Radiographs of Pneumoconiosis [10], published by the International Labour Office (ILO) in 2002, the patient was diagnosed with pneumoconiosis, in this case silicosis, with small opacities level size level p/q, and profusion level 2/3. Chest radiography during the second hospitalization (Figure 2A) showed bilateral diffuse lung fibrosis and small nodular opacities. Both hiluses were with more volume. Computed tomography (CT) of the chest, with high resolution CT of apical and hilar regions showed signs of diffuse micro-nodular changes predominantly in the upper lobes, with the presence of interstitial fibrosis, apical pleural thickening, and enlarged hilar lymph glands (Figure 2B).

The patient was legally recognized as having an occupational disease, and was approved early retirement due to disability.

DISCUSSION

Silicosis and pulmonary tuberculosis are not uncommon diseases in low income countries [11]. As both diseases have a similar initial presentation, and there is a strong possibility of concurrent existence of both diseases in one patient [4], it is very important that the physician in charge has adequate knowledge of diagnostic techniques in order to evaluate the patient. The occupational history of exposure to silica dust, progressive nature of breathlessness and classical radiological findings are the main clues for the diagnosis of silicosis. Exclusion of other conditions, especially pulmonary tuberculosis, is important before confirmatory diagnosis of silicosis is made. Patients suffering from silicotuberculosis are often misdiagnosed leading to late onset of adequate treatment and multiple complications and possible fatal outcome. There are strong suggestions for use of anti-tuberculotic chemoprophylaxis in order to prevent complications and fatalities in silica workers [12].

Silica-associated diseases can be prevented, as has been shown in some high-income countries. However, even with a focus on primary prevention, silica-associated diseases with long latency will occur well into the future due to contemporary exposure. In low-income countries, new cases of silicosis and associated lung cancer, chronic obstructive pulmonary disease and pulmonary tuberculosis are likely to occur for decades because reduction to very low concentrations of silica, necessary to prevent the disease, will take time to be achieved, while protective standards have not even been established for some silica-associated diseases such as tuberculosis.

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Силикотуберкулоза и силикоза као професионална обољења: приказ два болесника

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КРАТАК САДРЖАЈ

Увод Силикоза је најчешће обољење из групе пнеумокониоза, а узрокована је удисањем честица силицијум-диоксида. Код радника изложених силицијум-диоксиду повећан је ризик од настанка туберкулозе и других обољења изазваних микобактеријама. Код особа оболелих од силикозе ризик оболевања од туберкулозе је од 2,8 до чак 39 пута већи (у зависности од тежине силикозе) него код здравих људи.

Прикази болесника Први болесник, стар 52 године, примљен је на лечење у Институту за медицину рада Србије други пут током 2002. године са симптомима диспнеје, шиштања у грудима (визинга) и изражене малаксалости, који су били заступљени последњих 11 година. Радио је у ливници и био изложен прашини силицијум-диоксида 20 година. На радиографском снимку грудног коша начињеном при првом пријему у болницу утврђени су: обостране адхезије плеуре, дифузна фиброза и специфични процес паренхима плућа. На рендгенограму који је урађен током друге хоспитализације утврђене су обостране масивне, нехомогене, калцификоване промене неправилног облика у горњим и средњим деловима плућа. Болесник је умро 2007. године услед респираторне инсуфицијенције и хроничног плућног срца због силикотуберкулозе и хроничне опструктивне болести плућа (ХОБП). Други болесник, стар 50 година, примљен је на лечење у истој установи други пут током 2005. године са симптомима стезања у грудима, диспнеје, визинга и малаксалости, који су трајали претходних десет година. Радио је у ливници и био изложен прашини силицијум-диоксида 30 година. На рендгенограму грудног коша при првом пријему у болницу установљене су дифузна фиброза плућа и мале нодуларне промене у паренхиму. Постављена је дијагноза силикозе, величине нодуларних промена *p/q*, профузије промена 2/3. Приликом друге хоспитализације, радиографијом и компјутеризованом томографијом грудног коша утврђене су дифузна фиброза и мале нодуларне промене, превасходно у горњим деловима плућа. Болеснику је потврђено професионално обољење и одобрена инвалидска пензија.

Закључак Употреба силицијум-диоксида у земљама у развоју постепено се смањује, тако да се појава нових случајева силикозе и придружених обољења (малигнитети плућа, ХОБП, туберкулоза) може очекивати и током наредних година.

Кључне речи: силицијум-диоксид; силикоза; силикотуберкулоза; професионално обољење; смртни исход

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