Tanaffos (2007) 6(2), 73-76

Rapidly Progressive Silicosis

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ABSTRACT

The control and reduction of silica dust exposure in developed countries have resulted in a remarkable decrease in morbidity and mortality due to silicosis but exposure risks have remained high in other countries. Here, we present a fatal case of silicosis in a 27 year-old man with exposure duration of less than one year. This case indicated that intense exposure to silica dust can cause significant fibrotic disease after a short latency period. ***(Tanaffos 2007; 6(2): 73-76)***

Key words: *Silica, Silicosis, Pathology, Radiology*

CASE REPORT

A 27 year-old man was admitted to our medical center with anorexia, weight loss, and exertional dyspnea in August, 2004. The patient had worked as a packer in a silica powder production workplace at the age of 24 for one year and, thereafter, in an agricultural farm for 2 years. The patient's complaints had begun one year after termination of silica dust exposure. He had no previous history of respiratory symptoms and had never smoked. The patient stated that the workplace had no ventilation. Abnormalities on his chest x-ray had been identified at the age of 25. He became symptomatic at the age of 26 with complaints of dry cough and shortness of breath. Initial investigations showed normal complete blood count (CBC), urea, and electrolytes. IgG level had been raised to 2019 mg/l, and c-reactive protein (CRP) was 7 mg/l (+++). Spirometry demonstrated a very severe restrictive pattern [FEV1= 1.56L (39% of predicted), FVC= 1.62L (34% of predicted), FEV1/ FVC = 96%]. Transfer factor (TLCO) was 3.17 mmol/kp/m (29% of the predicted). Total lung capacity (TLC) was 3.32 L (51% of predicted) and residual volume (RV) was 1.69 L (105% of predicted). Chest x-ray showed small opacities with radiological profusion category 3/3, large opacity with B-scale, and evidence of costophrenic angles blunting and diffuse bilateral pleural thickening. Additional findings on chest x-ray according to International Labor Office (ILO) classification of radiographs of the pneumoconiosis (revised edition 2000) included coalescence of small pneumoconiotic opacities, bulla, marked emphysema, enlargement of hilar nodes, honeycomb lung and shaggy heart. (Fig.1). Two years later, the patient developed extensive spontaneous pneumothorax confirmed by CT-scan of the lung (Fig. 2). He consequently died as a result of ARDS. The autopsy revealed collapsed lung (Fig. 3), pleural thickening, pigmented masses and advanced fibrosis (Fig.4). Pathological investigation showed well-

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Received: 24 February 2007

Accepted: 22 May 2007

developed silicotic nodules with central necrosis and hyalinization, surrounded by many concentrically-arranged fibroblasts and histiocytes containing abundant silica particles revealed under polarized light microscopic examination (Figure 5, Figure 6).

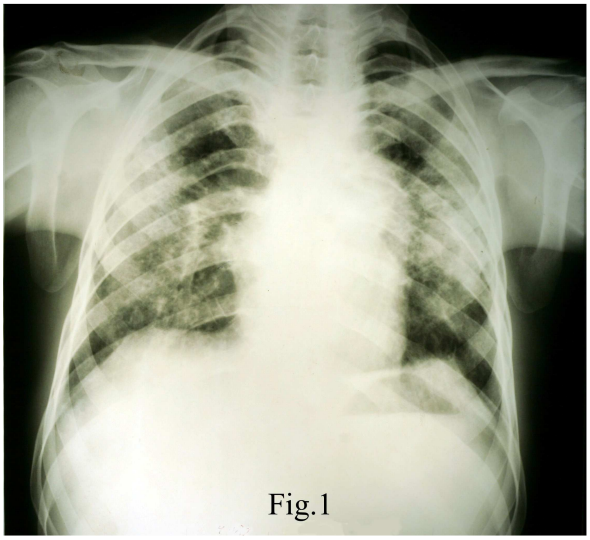

Figure 1. Chest x-ray of the patient showing 1) shaggy heart, 2) diaphragmatic tenting in both diaphragms, 3)blunting of left and right costophrenic angles and pleural thickening, 4) mixed alveolar and interstitial shadowing in both middle lobes and apical segments of both lower lobes, 5) airbronchogram pattern and peribronchial thickening, 6) prominent hilar areas, and, 7) alveolar shadowing in the left upper lobe.

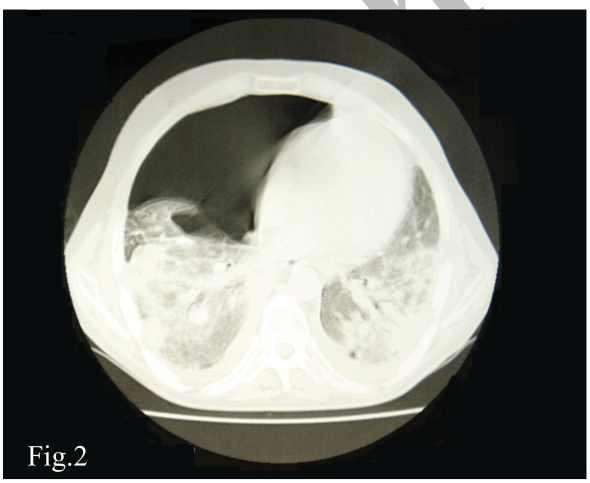

Figure 2. Lung CT illustrates 1) tension pneumothorax on the right, 2) bilateral alveolar shadowing with airbronchogram in both apical segments of lower lobes, 3) bilateral pleural thickening, 4) bilateral interstitial shadowing with reticulonodular pattern, 5) honey-combing pattern in the apical segment of left lower lobe.

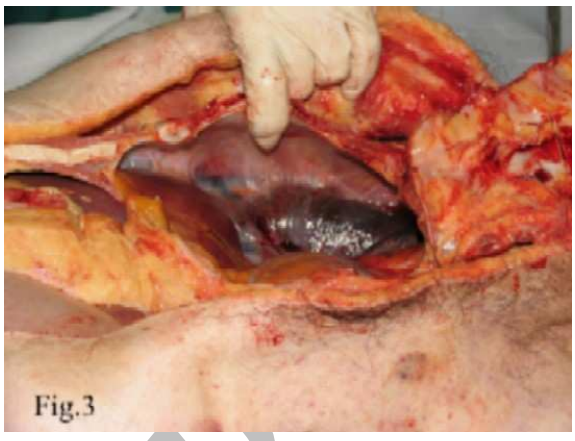

Figure 3. Autopsy demonstrates collapsed lung

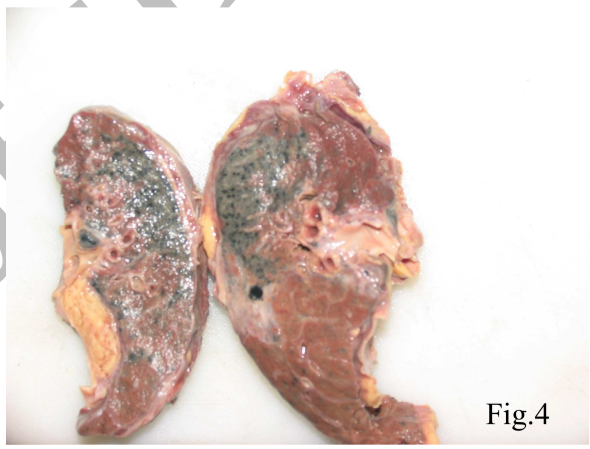

Figure 4. Autopsy also reveals pleural thickening, pigmented masses and advanced fibrosis.

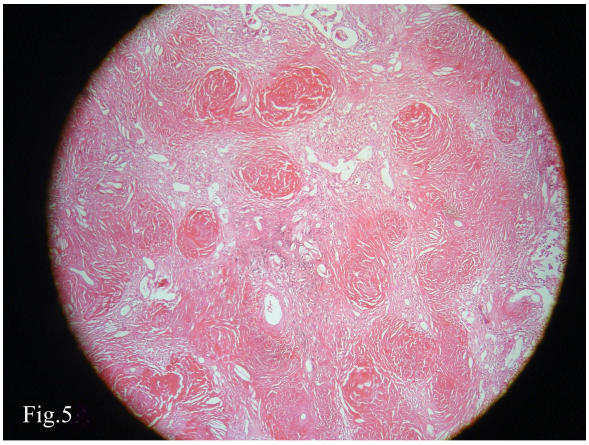

Figure 5. Autopsy lung specimen shows confluent nodules with central Necrosis and hyalinization surrounded by many concentrically arranged fibroblasts.

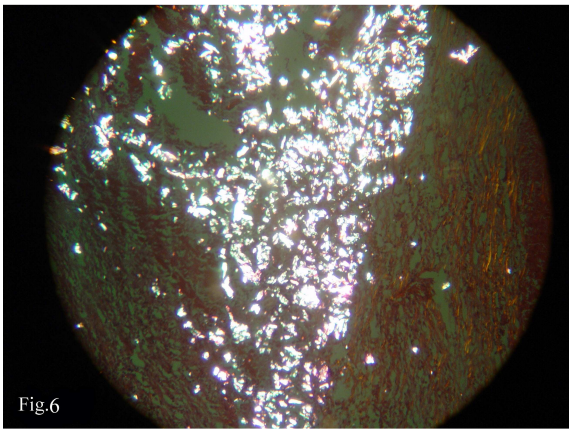

Figure 6. Lung specimen shows silica deposition revealed under polarized light.

DISCUSSION

Silicosis, a form of pulmonary fibrosis, is a well-known occupational disease that results from acute or chronic exposure to silica or silica-containing dusts (1, 2). Pathologic diagnosis of pneumoconiosis is based on the identification of several exposure-specific lesions, which include dust macules and nodules. Death or activation of macrophages and mediator release due to the toxic effects of silica consequently leads to nodule formation (3, 4, 5).

The current diagnostic criteria of pneumoconiosis are based on 1) exposure history, and 2) chest x-ray findings according to the ILO system (5). The sources of occupational exposure to silica dust are diverse and include many manufacturing industries in which silica is used, mining and processing of silica-containing rocks. Silica can be harmful when inhaled as dust in the respirable range (< 5-µm particles) (6, 7, 8, 9).

Death due to silicosis in young adults reflects relatively recent overexposures in a short period of time (10). It is well recognized that severe silicosis can cause significant lung function impairment (11). In this case, all of the previously-mentioned pathological changes due to silicosis were observed. With respect to our literature review, acute silicosis was found to occur in a few weeks to five years after exposure, and accelerated silicosis develops 5 to 10 years after exposure; however, this patient's occupational history, symptoms, chest x-rays, and pathological manifestations showed that after only one year of exposure to silica dust, rapidly progressive silicosis had occurred. This case shows that a brief, but intense exposure can cause significant fibrotic disease after a short latency period. Historically, people exposed to silica are from lower-income backgrounds and have not had the advantage of regular medical surveillance or specialized care until their conditions become very advanced. Therefore, professional surveillance is important in implementing effective hygiene and ensuring the health of these workers. Stringent controls of occupational exposure to silica dust are imperative to prevent future mortalities of industrial workers. This case of silicosis in a young adult male was reported due to the rarity of acute and accelerated silicosis in the world (10, 11).

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