CASE REPORT

Asbestosis in Malaysia: Report on First Two Cases

H H Lim, FAFOM*, K G Rampal, PhD, S Joginder, FRCR***, C M Abu Bakar, MSc****, K H Chan, MBBS*, T N Vivek, MBBS*, *Mediviron Consultants Sdn Bhd, 8 Jalan 21/12, 46300 Petaling Jaya, **Department of Community Medicine, Universiti Kebangsaan Malaysia, ***Department of Radiology, Pantai Medical Centre, Kuala Lumpur, ****Department of Occupational Safety and Health Ministry of Human Resources**

Summary

The first two cases of asbestosis in Malaysia are reported. Both had considerable occupational exposure to asbestos dust in the past, with a long latency period exceeding 30 years. One case presented with distinctive clinical and radiological features, while the other case was only confirmed by histological diagnosis. The usefulness of modern investigation techniques such as CT scan in the diagnosis of asbestosis is also illustrated.

Key Words: Asbestosis, Malaysia, Occupational disease

Introduction

Asbestos is a collective generic term referring to naturally occurring fibrous mineral substances, which include two main groups - serpentine (e.g. chrysotiles) and amphiboles (e.g. crocidolite). The wide commercial application of asbestos is mostly due to its excellent heat resistant and insulation properties, particularly in asbestos cement products. Health effects from asbestos exposure are mainly associated with inhalation resulting in four conditions - asbestosis, bronchial carcinoma, malignant mesothelioma and benign pleural thickening[1]. A review of the medical literature and examination of records from relevant organizations such as the Department of Occupational Safety and Health and Employees Social Security Organization (SOCSO) revealed no cases of asbestosis reported so far. This is a report of the first two cases of asbestosis notified in the country.

Case Report

Case I

The first case, a 54 year old Malay male, presented with non-productive cough of three months' duration, associated with slight dyspnoea on effort. There was no history of cigarette smoking or bronchial asthma. He worked as a production worker in a factory manufacturing asbestos cement pipes and building materials since 1963.

On examination, there was no cyanosis, clubbing or edema. Blood pressure was 110/90mmHg. The cardiovascular system was normal. His chest expansion was slightly reduced bilaterally. Crepitations were heard in both lower zones. There was no lymphadenopathy.

Sputum for Acid Fast bacilli were negative on three occasions, while sputum culture grew no pathogens. Lung function test showed a mild restrictive ventilatory defect.

The chest radiograph showed normal heart size and shape. Thickened bronchi were seen at both hilar and right lower zones. There was minimal pleural thickening at the right costophrenic angle.

A high resolution computed tomographic (HRCT) scan of the thorax showed bilateral pleural thickening and calcification, mainly in the mid and lower zones posteriorly (Fig. 1). No mediastinal or hilar abnormalities were seen. Lungs were well expanded and clear (International Labour Office Classification, Grade 0/0).

A biopsy of lung tissue from the right lower lobe was performed which revealed fairly uniform alveoli spaces with congested blood vessels in the alveolar wall. Interstitial tissue revealed edema and fibrosis. No granulomas or malignancy were seen. No asbestos bodies were identified. Biopsy of the right pleura and pleural nodules showed partly denuded mesothelial cells on the surface, comprising of hyalinized fibrous tissue with several compressed blood vessels. No granulomas or asbestos bodies were seen. The diagnosis of asbestosis was based on the histological evidence of interstitial fibrosis in the lower lobes with history of prolonged and heavy occupational exposure to asbestos.

Case 2

The second case, a 55 year old Malay male, complained of dyspnoea on effort for the past one year. He had no cough, phlegm or chest pain. There was no history of bronchial asthma, but he smoked 20 cigaretttes/day since 1965. He worked as a production worker in a factory manufacturing asbestos cement pipes and building materials since 1965.

On examination, he had no cyanosis, clubbing or edema. Blood pressure was 110/80mmHg. His cardiovascular system was normal. Chest expansion was reduced bilaterally. There were reduced breath sounds at the right apex with bilateral crepitations heard over both lower zones. There was no lymphadenopathy.

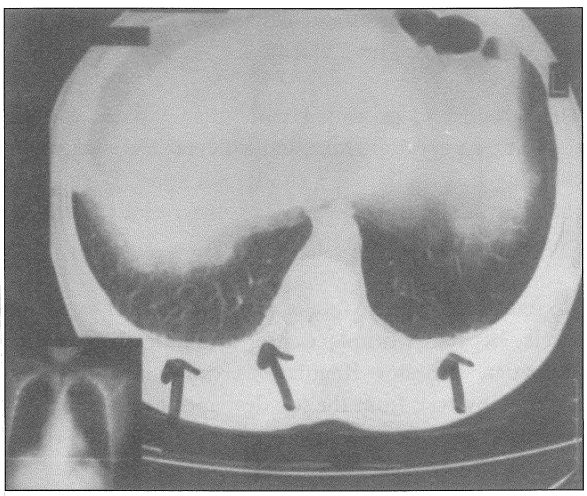

Fig. 1 : HRCT of the thorax of patient 1 showing bilateral pleural thickening and calcifications, mainly in the mid and lower zones posteriorly.

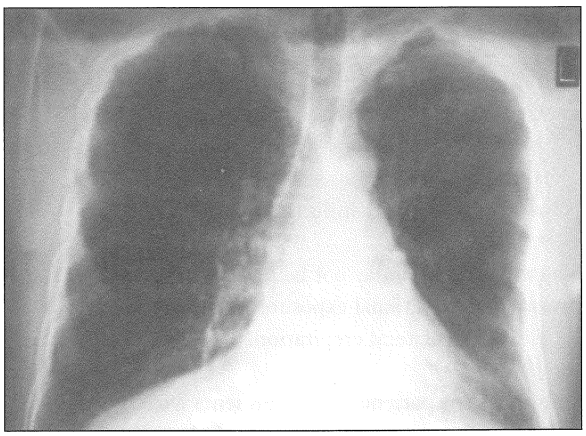

Fig. 2 : Chest radiograph of patient 2 showing pleural thickening, especially the left apex and base, shaggy heart borders and reticulo-nodular changes in the lung bases. The left lung apex also shows changes of old pulmonary tuberculosis.

Investigations showed a normal white count but raised ESR (40mm/hr). Three sputum specimens were negative for acid fast bacilli. Lung function test showed reduced forced expiratory volume (FEV1) in one second, but the forced vital capacity (FVC) was within normal limits.

The chest radiograph showed marked pleural thickening at the left apex and base, and milder pleural thickening along the right hemithorax (Fig. 2). The heart borders were shaggy (left>right) and there were reticulo-nodular changes in the lung bases (ILO Gd 1/0). The left lung apex showed changes of old pulmonary tuberculosis. CT scan of the chest showed reticulo-nodular changes in the left lung apex and pleural thickening, suggestive of old tuberculosis. Marked pleural thickening was seen in both lower zones - more on the left side where there was a calcified plaque. There were also interstitial changes in both lung bases, more on the left, suggesting interstitial fibrosis.

Discussion

The diagnosis of asbestosis is made on a combination of four criteria:

i) history of occupational exposure to asbestos dust, usually for more than 10 years before examination;
ii) radiological changes of basal fibrosis (ILO Classification of category 1/0 and above);
iii) bilateral crepitations at full inspiration, persisting after coughing;
iv) restrictive defects in lung function.

The above criteria may not be present in every case, but a history of occupational exposure (i) is essential. However, occasionally, bilateral crepitations may be the only sign.

Clinically, the patient usually presents with dyspnoea on exertion, chest pain and cough. Other signs include clubbing of the fingers and toes.

The most important single investigative evidence for routine screening is the chest radiograph, showing features of interstitial fibrosis. The ILO 1980 International Classification of Pneumoconiosis records the changes on a 12-point scale of severity (profusion) and extent (zones)[2]. Earliest changes usually occur at the bases. There are no hilar glands enlargement or calcification unless silica dust exposure has also occurred.

Lung function test changes usually reflect restrictive defects with reduction in FVC but normal FEV1/FVC ratio (40% of cases). However, about 10% of cases show an airway obstructive defect, while the remaining 50% show a mixed pattern, mainly due to the confounding effects of cigarette smoking. Small airway obstruction as indicated by Forced Expiratory Flow (FEF) 25 - 75% or FEV_3/FEV_6 can also be an early manifestation of asbestos related lung disease.

The pleura, both visceral and parietal, are also affected by the fibrosis, showing up radiologically as pleural thickening and blunting of one or both costophrenic angles.

Asbestos bodies in the sputum indicate past exposure, but are not necessarily diagnostic of asbestosis.

CT scan or lung biopsy are not routinely done for medical screening of asbestos workers, but may provide conclusive evidence of fibrosis or malignant changes.

The first case probably represents early asbestosis, producing clinical symptoms such as dyspnoea on exertion and non productive cough, with bilateral crepitations. However, his chest radiograph and CT scan showed no evidence of parenchymal fibrosis, which was only confirmed by lung biopsy.

The second case presented with more typical features such as dyspnoea on effort and bilateral crepitations in the lung bases. His chest radiograph and CT scan also showed distinctive features of interstitial fibrosis. Biopsy was not indicated in this case.

No industrial hygiene monitoring data of dust exposure levels in the workplace are available before the Malaysian Asbestos Regulations came into force in 1986[3]. However, from the job descriptions given by the workers and factory management sources, there appeared to be considerable occupational exposure to asbestos dust before 1984. Crocidolite was used until 1975, while amosite was used until 1983. Dust monitoring data after 1986, however, showed the levels to be below the Permissible Exposure Limit of 1 fibre/ml of air (averaged over an eight-hour period), as the factories had implemented various safety measures to comply with the Asbestos Regulations.

The occurrence of these two cases is probably related to their dust exposure levels in the past, presenting after a long latency period of more than 30 years. This is a timely reminder of the constant need for proper and adequate control measures in the safe handling of asbestos in the work place and for medical practitioners to inquire about previous occupational exposures in case of pulmonary fibrosis.

Acknowledgement

This report is part of a study under the Tun Abdul Razak Research Award, Academy of Medicine, 1996. The authors would like to acknowledge the assistance of Mr. Kamarulzaman (UKM) for preparing the photographs.

References

1. World Health Organization (1986). Asbestos and other Natural Mineral Fibres. Environmental Health Criteria 53. WHO, Geneva.
2. International Labour Office (1980). ILO International Classification of Radiographs of Pneumoconiosis, ILO, Geneva.
3. Ministry of Human Resources, Malaysia (1984). Factories and Machinery (Asbestos Process) Regulations, 1986.