A CASE OF ABDOMINAL MESOTHELIOMA

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SYNOPSIS

A 48 year old man with pulmonary fibrosis and a previous history of asbestos exposure presented 6 years later with abdominal pain and ascites and was found to have peritoneal mesothelioma. This is the first such case reported in Singapore.

INTRODUCTION

Accumulated knowledge of asbestos-related diseases has expanded greatly over the past 2 decades. Wagner et al first drew attention to the strong association of malignant mesothelioma with asbestos exposure in the north-western Cape province of South Africa (1). Since then, numerous workers have confirmed this observation. We report here the first case of asbestos-related malignant mesothelioma of the abdomen in Singapore.

CASE REPORT

A 48 year old Javanese man was seen in 1978 for pulmonary fibrosis, detected on pre-employment chest radiography. He was essentially asymptomatic. He had a past history of pulmonary tuberculosis treated from 1956 to 1957. He smoked 40 cigarettes daily for 30 years. From 1950 to 1967, he worked in an asbestos cement factory. His work consisted of tipping sacks of unmilled asbestos fibres (chrysotile, crocidolite and amosite) into a hopper, which fed the materials into a mixing machine. The work environment was extremely dusty and he did not wear any protective mask.

Physical examination revealed only marked digital clubbing and crepitations on chest auscultation. His chest radiograph showed bilateral reticular opacities, mainly in the lower zones. Lung function tests showed a restrictive ventilatory pattern (observed FVC 2.02, predicted FVC 3.47 litres) and a reduced transfer factor (observed TL_{CO} 10.5, predicted TL_{CO} 17.7). Sputum for acid-fast bacilli and asbestos bodies was negative.

He first noticed exertional dyspnoea in 1983. Thereafter, his effort tolerance deteriorated rapidly and by May 84, his effort tolerance was limited to 30 metres on ground level. His chest radiography in May 1984 showed reticular opacities of the lower zones and bilateral pleural thickening (Fig. 1). Ventilatory lung function had deteriorated (observed FVC 1.22L, predicted FVC 3.38L) but transfer factor was unchanged. In July 84, an open lung biopsy showed interstitial and peri-bronchiolar fibrosis. Numerous ferruginous (asbestos) bodies were seen (Fig. 2).

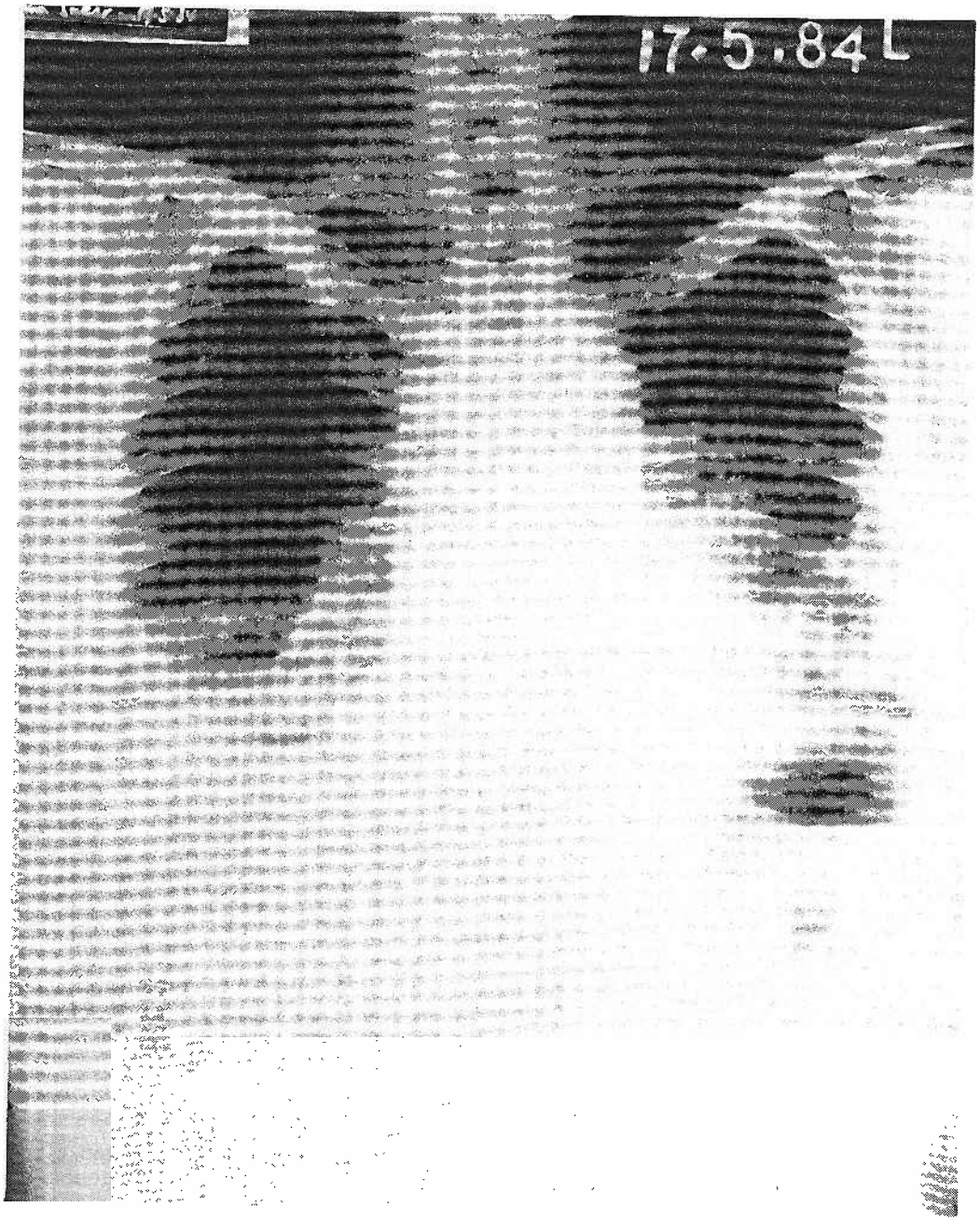

Figure 1 — Chest x-ray showing bilateral reticular opacities of lower zones, pleural thickening and shagginess of left heart border.

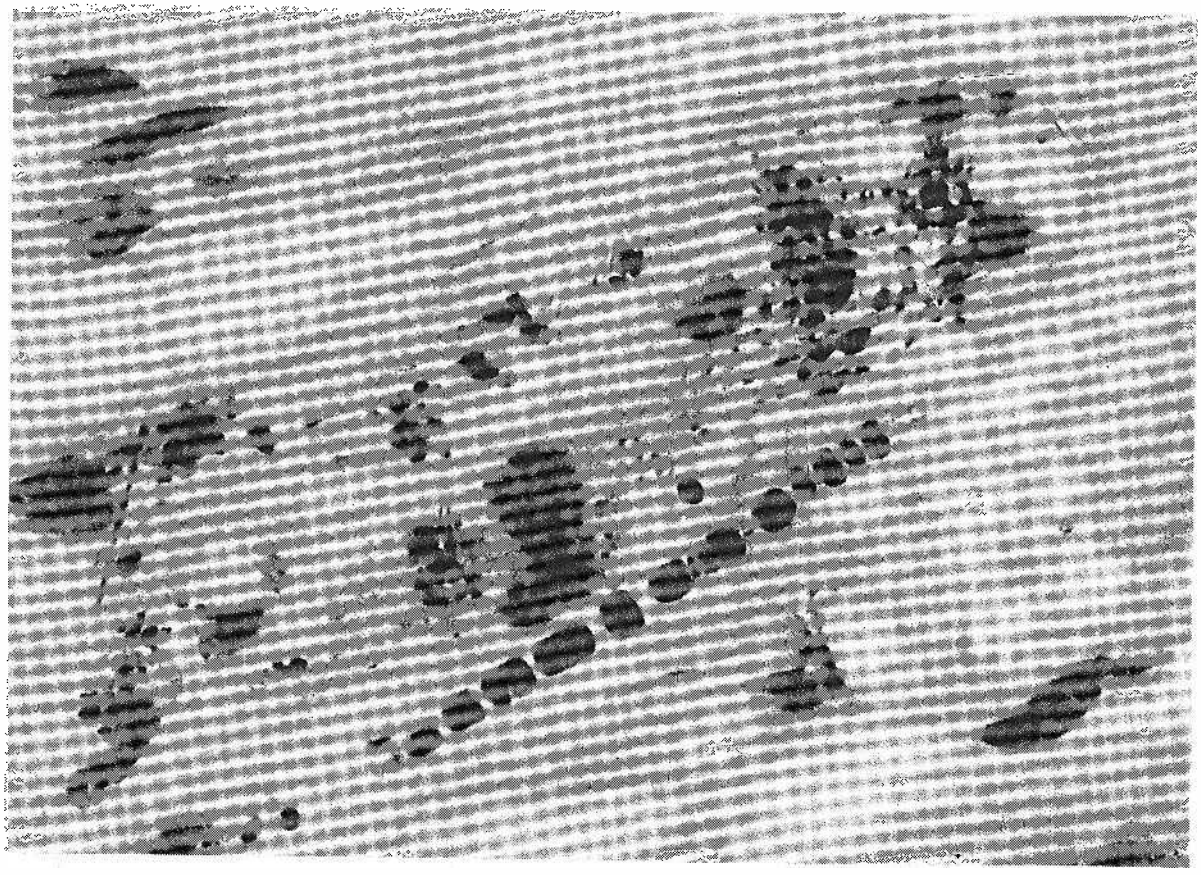

Figure 2 — H & E (Magnification × 1000)
Typical asbestos body with thin, clear fibrous core and segmented iron-protein coat.

In early Sept 1984, he was warded for abdominal pain, constipation, gross ascites and severe weight loss. Apart from erythrocyte sedimentation rate 58 mm/lst hour, serum glutamate pyruvate transaminase 82 u/L and serum glutamate oxaloacetate transaminase 150 u/L, laboratory investigations were essentially normal. Arterial blood gases analysis showed pH 7.37, P_aO_2 74 mmHg, P_aCO_2 38 mmHg and O_2 saturation 93.3%. An abdominal tap drew straw-coloured peritoneal fluid of SG 1.031 and protein content 4.9 g/dl. Clusters of malignant mesothelial cells were seen on microscopic examination of the peritoneal fluid (Fig. 3). Computerised tomography scan of the abdomen showed gross ascites; two dense areas representing tumours were seen in the right hypochondrium just below the liver and in the pelvic mesentery. Computerised tomography scan of the chest showed mediastinal nodes, basal fibrosis and pleural thickening. He was treated symptomatically and subsequently was discharged.

On 19 October 1984, he was warded for sudden dyspnoea and he died soon after hospitalisation.

An autopsy was performed. Both lungs showed honey-combing, prominent over the lower lobes and right middle lobe. Extensive pleural plagues were noted over the right lung. The abdominal cavity contained over 7000 ml of thick yellow exudate. There was extensive tumour involvement of both visceral and parietal peritoneum, with multiple nodules varying from 2 cm to 9 cm in diameter. Three large prominent nodules were noted over the sigmoid mesocolon, in the right hypochondrium below the liver and in the left upper posterior abdominal wall. Microscopically, the tumour was an epithelial variant of malignant mesothelioma, with sheet-like pattern showing few tubular or papillary areas. There were frequent mitosis, atypism and anaplastic areas (Fig. 4).

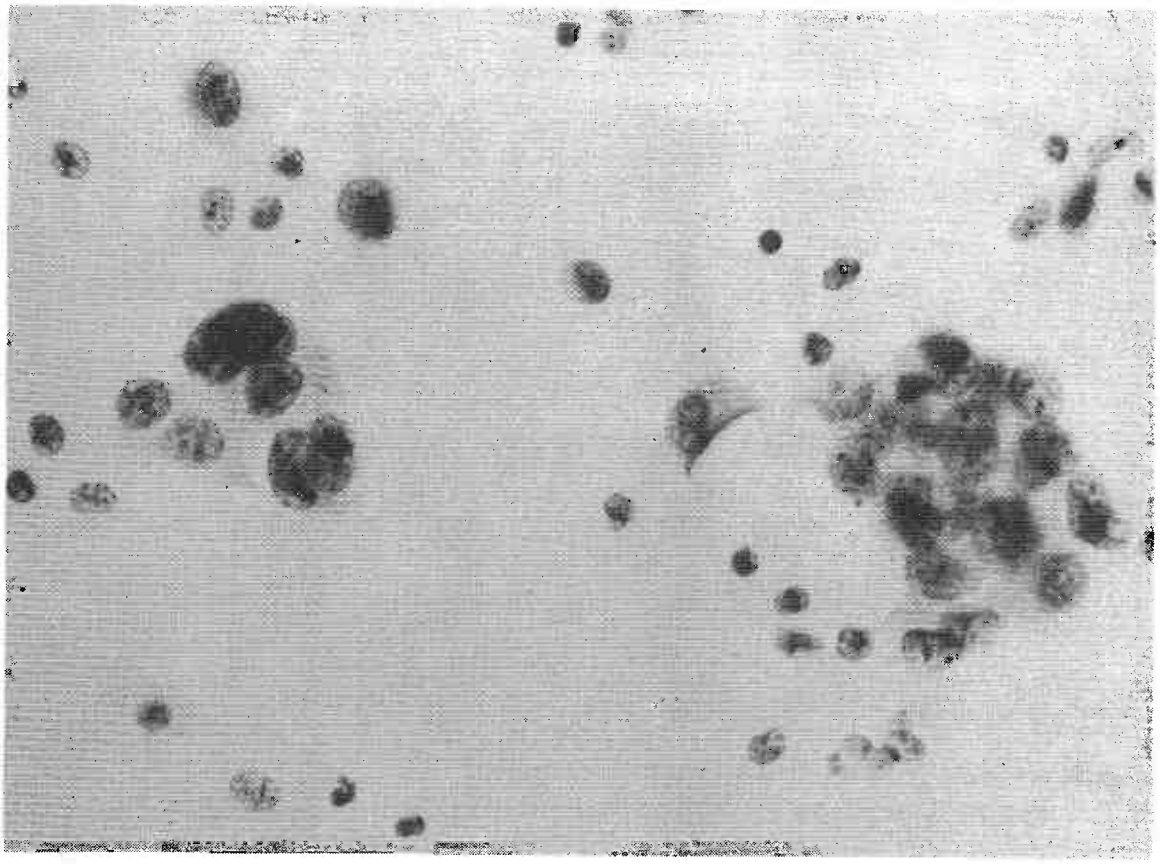

Figure 3 — Papanicolou Stain (Magnification × 1000)
Peritoneal fluid showing clusters of malignant mesothelial cells.

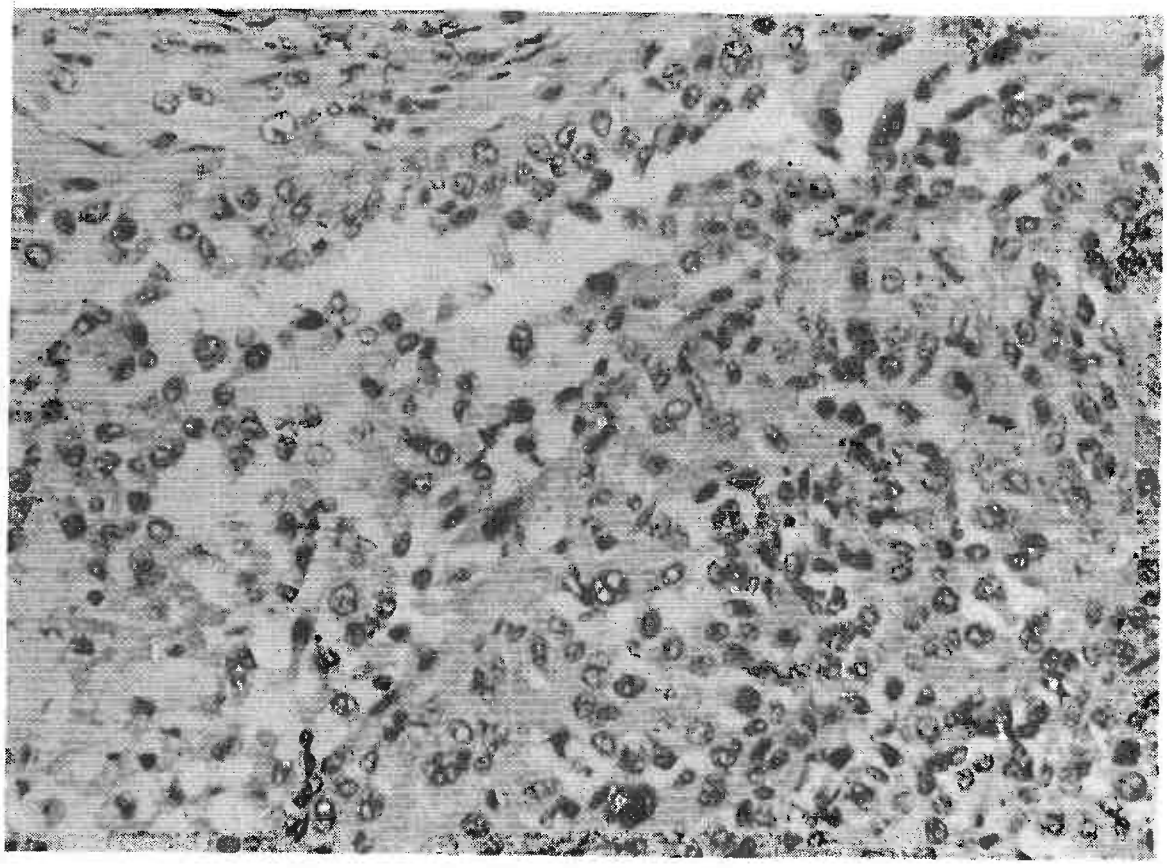

Figure 4 — H & E (Magnification × 400)
Mesothelioma with a poorly formed tubular area. Cells show moderate anaplasia.

DISCUSSION

Over the last 10 years, Singapore consumed about 9,000 tonnes of asbestos annually. In 1984, the consumption was 9,464 tonnes of asbestos (2). All of the asbestos used in Singapore is imported. Important asbestos industries in Singapore include asbestos-cement production, insulation work, shipyard work and construction industry. There is now an increasing awareness of the hazards of asbestos exposure and the Annual Reports of the Ministry of Labour, Singapore, showed a cumulative total of 15 cases of asbestosis from 1973 to 1984. Up to the time of this write-up, a further two cases were reported in 1985.

The long latency period between exposure to respirable asbestos fibres and the onset of symptoms is well established. Lung cancer peaks at 30-35 years from onset of exposure, mesothelioma at 35-40 years and asbestosis at 40-45 years (3). Hence, today's cases are attributable to the working environment 30-40 years ago. Based on this observation, it is reasonable to anticipate more cases of asbestos-related diseases to surface over the next 10 to 20 years as dust control measures of the past were inadequate. Another point to note is the fibre gradient for mesothelioma; crocidolite has the greatest potential for developing mesothelioma, chrysotile the least with amosite somewhere in between (4). For this reason, the use of crocidolite is discouraged in Singapore. The dose-response relationship for mesothelioma is linear with no safe threshold limit (4).

The main point of diagnosis of asbestosis is that the patient may be able to benefit from compensation. Diagnosis of asbestosis may be made from an accurate exposure history, physical findings and radiographic abnormalities. Lung biopsy may be resorted to when pulmonary fibrosis is present without pleural plaques, or when an alternative diagnosis is considered. Diagnosis is sometimes made late, especially in countries where asbestosis is not common. The diagnosis of malignant mesothelioma is by no means straight forward as the morphological distinction between benign and malignant mesothelial proliferation may be difficult. Histological diagnosis of mesothelioma from biopsy specimen is occasionally one of exclusion. Post-mortem examination may be necessary to exclude a primary adenocarcinoma elsewhere. Cytopathological diagnosis of mesothelioma from pleural or ascitic fluid is even more difficult and often not recommended. A recent report (5) that Ca^1 antibody, a very useful marker of human cancer cells, was particularly helpful in distinguishing reactive mesothelial cells from mesothelioma needs further confirmation.

Peritoneal mesothelioma presumably resulted from the migration of asbestosis fibres from the gas exchange portion of the lung across the diaphragm to lodge in the peritoneal cavity.

In 1954 (8) Leicher reported an abdominal tumour in a patient with pulmonary asbestosis and thought that asbestos was also present in the tumour. In Enticknap's series (9) of peritoneal tumours, the time from the first exposure to the onset of symptoms was always long, from 20 to 46 years. All cases presented with abdominal pain, discomfort or ascites. Once diagnosed, the illness ran a course of less than six months. Our patient presented six years after diagnosis and died within two months of the diagnosis of peritoneal mesothelioma.

There is no definitive treatment for mesothelioma and asbestosis. The only practical way to avoid asbestos-related diseases is to enforce strict occupational health regulation and supervision. In Singapore, the threshold limit values for occupational exposure over an eight-hour period is 2 chrysotile fibres more than 5μ long/ml of air, 0.5 amosite fibre/ml and 0.2 crocidolite fibre/ml. Some non-asbestos fibrous substitutes have been used but they are not entirely free from significant health hazards (6). If asbestos-related diseases are diagnosed, the question of compensation should be considered. Legislation on compensation varies in different countries. In Singapore, workers are entitled to compensation if asbestos-related diseases are notified not later than 36 months after ceasing to be employed (7).

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