

EARLY MALIGNANT CHANGES IN PLEURAL PLAQUES DUE TO ASBESTOS EXPOSURE: A CASE REPORT

HILTON C. LEWINSOHN

TBA Industrial Products, Rochdale, England

PLEURAL lesions related to asbestos exposure are well documented (Meurman 1966; Mattson & Ringqvist 1970). The pathology has been described by Roberts (1971). Mattson and Ringqvist have studied 42 cases with radiological signs of pleural plaques at the Lung Clinic, Boras, diagnosed between 1961 and 1967. They define pleural plaques as 'bilateral, local, irregular thickenings of the parietal pleura which cannot be connected with haemorrhage, infection or injury'. These authors review the literature on the subject and point out Kiviluoto's (1960) conclusion that only amphibole asbestos can cause the development of pleural plaques. Two of Mattson and Ringqvist's patients have died of malignant mesothelioma during the 6-year period of observation. Fletcher and Edge (1970) have given an excellent description of the early radiological changes in pulmonary and pleural asbestosis. These authors prefer the latter term to pleural plaques in the title of their paper.

Case Report

A Caucasian male, born in 1907, died on 22 August 1970 after being absent from work for one week. He was born in the Channel Islands and worked as a farm labourer from 1921 to 1928. From 1928 to 1940 he worked in garages and was a truck and car driver. In 1940 he began his employment at a Rochdale asbestos textile factory, in an area now demolished. He worked on the disintegrators, crushing-machine, grit separators and was a member of the 'heavy gang' doing all sorts of dusty jobs which exposed him to chrysotile asbestos and occasionally also to crocidolite. In 1953 he changed his job to that of truck driver, delivering the bags of prepared asbestos fibre from the fiberizing plant to the factory for further processing. In 1960 he went to work in the company garage as a driver and garage-hand.

On engagement he was examined by the company Medical Officer but the records of this examination are no longer available. Medical records are available which show that he was regularly examined from 1950 onwards and that his chest was radiographed at approximately 2-yearly intervals until 1968 and annually thereafter. The medical records are brief. Rhonchi were first heard in his chest in July 1957. He was absent from work because of bronchitis during the winter of 1958-9 and thereafter was prone to annual winter

(Received for publication October 1973)

A02329

CTC008920

bronchitis. In 1964 he was regarded as suffering from chronic bronchitis. In 1967 he began to complain of dyspnoea on effort and he became orthopnoeic, had Grade 1-2 effort dyspnoea and complained of occasional chest tightness after the 1967-8 winter. His symptoms of orthopnoea and dyspnoea on effort gradually increased. He was last examined in March 1970. He had stopped smoking in 1960 and never resumed. On examination in March 1970 he was cyanosed, had mild hypertension (BP 150/90) and physical signs compatible with chronic bronchitis. No finger-clubbing was noted.

Special investigations carried out (at work) consisted of serial chest radiography, lung function tests and an ECG in March 1968 and March 1970. No comment on these films appears in the medical records until 1969 when some enlargement of the cardiac outline was thought to have occurred.

He applied to the Pneumoconiosis Medical Panel for Industrial Injuries (Disablement) Benefit in March 1970, but was not accepted as it was considered that he was suffering from chronic airways obstruction and not asbestosis. In view of his rather sudden death, and his history of exposure to asbestos, the Coroner was informed and requested an autopsy examination. The autopsy was performed by the Coroner's Pathologist (Dr D. S. Lyon) on 24 August 1970.

Autopsy findings

The relevant autopsy findings relate to the thoracic cage and its contents. All the main airways were patent and mucoid secretions were present in large quantities. The lungs were distended and obviously emphysematous. They were congested on sectioning them. There was no pulmonary fibrosis present on naked eye examination.

On the left side, the chest wall and visceral pleural surface was studded with tumour nodules varying in size from 1-2 mm to 10 mm or more in diameter (Fig. 1). Calcified plaques were present on the left dome of the diaphragm and hyaline pleural plaques were seen on the chest wall. Similar pleural plaques were present on the right but no tumour nodules were seen.

TABLE 1. VENTILATORY CAPACITY AT VARIOUS AGES

	February 1968 (age 60 years)	April 1968 (age 60 years)	March 1969 (age 61 years)	March 1970 (age 62 years)
FEV ₁ (litres)	1.23	2.26 (4.06)	1.87 (3.67)	1.84 (3.68)
FVC (litres)	0.88	1.00 (3.08)	0.17 (2.67)	0.86 (2.65)
FEV ₁ /FVC %	49 (70-80)	44 (70-80)	46 (70-80)	46 (70-80)
Gas transfer factor (ml/min/mm Hg)	—	26.5 (22-5)	19 (24)	17 (24)
Paco ₂ rebreathing	—	44.5 (36-40)	46 (36-40)	47 (36-40)

Figures in parenthesis indicate predicted normal values.

These results are compatible with restrictive and obstructive lung disease.

The mediastinal surface of the pericardium was studded with tumour nodules as was the outer wall of the oesophagus. The heart was enlarged due

CTC008921

AU2630

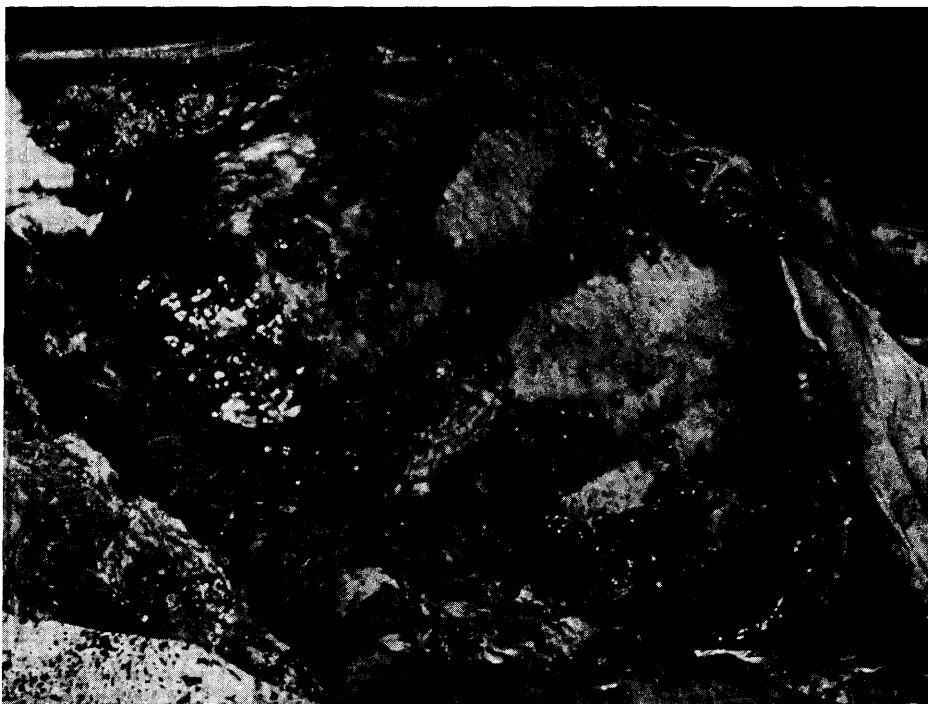


FIG. 1. Pleural plaques on the left chest wall with multiple polypoid nodules of tumour arising from them at post mortem

to left and right ventricular hypertrophy and dilatation. Patchy coronary atheroma was present and patchy myocardial fibrosis was found. The liver and spleen showed chronic venous congestion.

The diagnosis in this case was recorded by the Coroner's Pathologist, as 'mesothelioma due to exposure to asbestos'. Histological sections were also examined by the members of the U.I.C.C. panel of pathologists. The tumour was classified as a tubulopapillary type of malignant mesothelioma, with some areas showing sheets of undifferentiated cells but no sarcomatous elements. Asbestos bodies were found in the lungs and in places slight asbestosis as well.

Discussion

From the pathological point of view this case has several features of interest. Firstly, it shows discrete nodules of mesothelioma arising from both visceral and parietal pleural surfaces (Figs 2-5). While these may be the result of seedling deposition, it could also be argued that the diffuse mesothelioma is the result of confluence of a number of multifocal tumours, arising in both visceral and parietal pleural layers. The mesothelial cells lying adjacent to the tumour in Fig. 4 appear abnormally hyperplastic and suggest an unstable, pre-malignant state.

Secondly, this case illustrates that foci of mesothelioma can arise from the

A02331

CTC008922



FIG. 2. Mesothelioma arising in the cells overlying a hyaline parietal pleural plaque. The tumour arises from a narrow stalk and proliferates in a polypoid form to cover the adjacent pleura. $\times 48$



FIG. 3. Mesothelioma arising in the cells overlying a hyaline parietal pleural plaque. $\times 140$

CTC008923

A02332

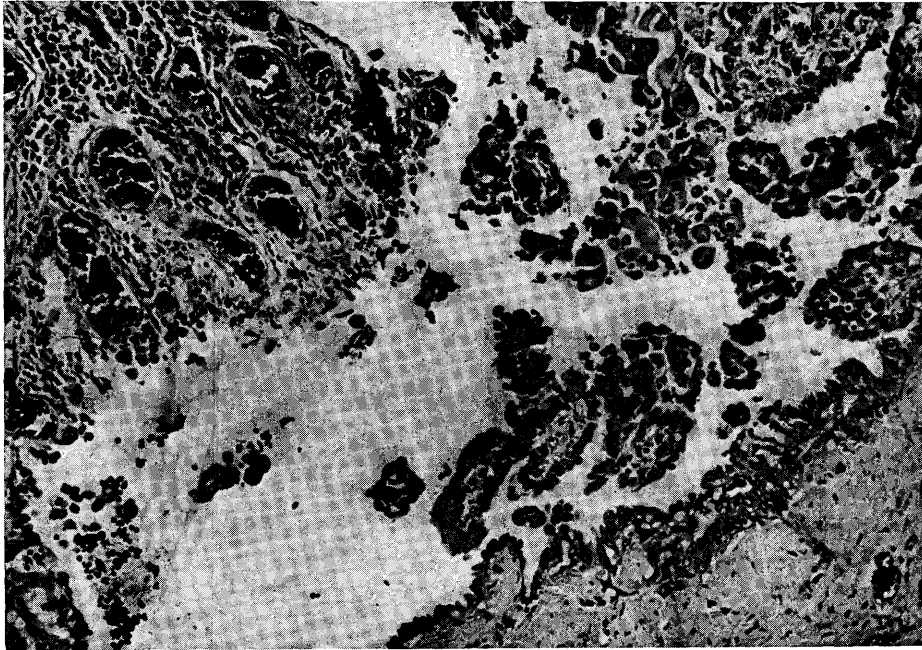


FIG. 4. Unstable mesothelial cells lining the parietal pleura adjacent to a focus of mesothelioma. The appearance suggests a diffuse origin of the tumour of multifocal distribution rather than multiple seedling deposits. $\times 200$

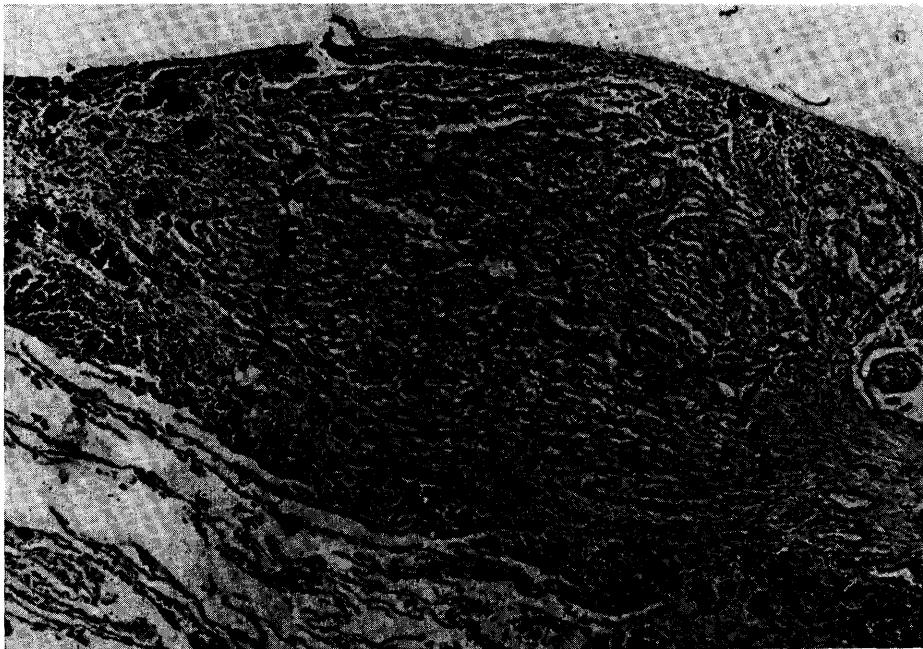


FIG. 5. A focus of mesothelioma in the visceral pleura of the lung. This could have arisen per se in the visceral pleural cells, but seedling deposition or lymphatic spread can not be excluded. $\times 48$

pleural cells overlying a hyaline plaque (Fig. 3). This finding does not answer the question 'Are pleural plaques pre-malignant?' but it does demonstrate that the mesothelioma cells on the surface of a pleural plaque are able to give rise to a tumour.

Fletcher (1972), in a mortality study of 408 male shipyard workers with pleural plaques, has found 3 cases of mesothelioma and 16 bronchial carcinomas. He finds the occurrence of these 3 cases to be very significant considering the rarity of the disease in the general population.

The case report presented in this paper illustrates the possible origin of a mesothelioma in non-calcified pleural plaques. Eight chest radiographs are available dating back to June 1951—11 years after first exposure. Pleural thickening of the 'flange' type as described by Fletcher and Edge (1970) is first apparent on the right side of the radiographs dated January 1964. Subsequent films show definite 'hardening' of the pleural lesion. Fig. 6 illustrates the appearances noted on the last available film in March 1970. There is no evidence of underlying pulmonary fibrosis.

Dalquen et al. (1970) regard plaques as an epidemiological fossil, indicating an endemic asbestos exposure in certain populations. These authors believe that all observed cases of pleural plaques should be officially registered as they could provide the basis for further prospective studies on the aetiology of pleural mesothelioma. The case described tends to support this conclusion.

Sheers and Templeton (1968) have found that in workers exposed to asbestos



FIG. 6. A chest radiograph showing a pleural lesion in the right thorax (March 1970)

A92334

CTC008925

in the Royal Dockyard at Devonport, the prevalence of pleural fibrosis ranges from 28% in continuously exposed workers to 1.9% in those with least exposure. Mackenzie and Harries (1970) point out that in shipyards, as in the asbestos industry, there has been a changing pattern of disease due to better control of dust. This has resulted in a longer maturation period and hence they point out that not so much is seen of the florid, rapidly progressive interstitial pulmonary fibrosis which was so common 30 years ago. It is thus easier to see changes in the pleura where there is only very minor fibrotic change present. The uncertainty of the relationship of pleural reactions to the possible development of malignant disease is stressed, especially as they are encountering more extensive pleural reactions, often with effusion, in younger men than before but after long exposure of 20-25 years. These young patients have longer for the disease to progress and for possible malignant changes to develop.

Summary

Pleural plaques are generally accepted as an index to asbestos exposure. The occurrence of pleural plaques and other pleural lesions in workers exposed to asbestos must be carefully recorded to determine the precise prognostic value of this radiological sign in the study of the natural history of malignant mesothelioma of the pleura.

ACKNOWLEDGEMENTS

I should like to thank Dr D. S. Lyon for preparing the histological sections and allowing me to use them to illustrate this case. I should like to acknowledge the useful comments by Dr J. S. P. Jones on the histology in this case and to thank him for his help in producing Figs 2-5.

REFERENCES

- DALQUEN, R., HINZ, I. & DABBERT, A. F. (1970) Pleuraplaques, asbestose und Asbestexposition, eine epidemiologische Studie aus dem Hamburger Raum. *Pneumologie*, 143, 23.
- FLETCHER, D. E. (1972) A mortality study of shipyard workers with pleural plaques. *Br. J. industr. Med.*, 29, 142.
- FLETCHER, D. E. & EDGE, J. R. (1970) The early radiological changes in pulmonary and pleural asbestosis. *Clin. Radiol.*, 21, 355.
- KIVILUOTO, R. (1960) Pleural calcification as a roentgenologic sign of non-occupational endemic anthophyllite-asbestosis. *Acta radiol.*, Suppl. 194, 1.
- MACKENZIE, F. A. F. & HARRIES, P. G. (1970) Changing attitudes to the diagnosis of asbestos disease. *J. R. naval med. Serv.*, 56, 116.
- MATTSON, S. B. & RINGQVIST, T. (1970) Pleural plaques and exposure to asbestos. *Scand. J. resp. Dis.*, Suppl. 75.
- MEURMAN, L. (1966) Asbestos bodies and pleural plaques in a Finnish series of autopsy cases. *Acta path. microbiol. scand.*, Suppl. 181.
- ROBERTS, G. H. (1971) The pathology of parietal pleural plaques. *J. clin. Path.*, 24, 348.
- SHEERS, G. & TEMPLETON, A. R. (1968) Effects of asbestos in dockyard workers. *Br. med. J.*, iii, 574.

A02335