R.C. Stein, et al., "Pleural Mesothelioma Resulting from Exposure to Amosite Asbestos in a Building," RESPIRATORY MEDICINE, 1989.

This is a case report of a mesothelioma as a result of exposure to amosite asbestos used in insulating material in the construction of an office. The patient was a 54 year old female office worker who was a non-smoker and was otherwise healthy. She had not lived in the proximity of an asbestos factory as a child nor did she live with other persons who worked with asbestos. Her only known exposure was to construction at the office. Exposure was to a sprayed-on insulation. Latency was 14 years. Asbestos fibers were found in her lungs.

Respiratory Medicine (1989) 83, 237–239

Pleural mesothelioma resulting from exposure to amosite asbestos in a building

R. C. STEIN, J. Y. KITAJEWSKA*, J. B. KIRKHAM*, N. TAIT†, G. SINHA AND R. M. RUDD

*The London Chest Hospital, Bonner Road, London E2 9JX, *Biological Electron Microscopy Unit, Queen Mary College, Mile End Road, London E1 4NS, †Society for the Prevention of Asbestosis and Industrial Disease, 38 Drapers Road, Enfield, Middlesex EN2 8LU, U.K.*

Introduction

Considerable concern has been expressed about the possible risks to health resulting from exposure to asbestos in buildings. We report a case of mesothelioma caused by exposure to amosite asbestos used as an insulating material in the construction of an office.

Case Report

A 54-year-old female office worker presented with a six month history of malaise, persistent cough and exertional dyspnoea. She was a lifelong non-smoker with no significant past medical history.

Examination revealed a left pleural effusion which was confirmed radiologically. No endobronchial lesion was observed at fibreoptic bronchoscopy. During aspiration of the effusion difficulty in traversing the pleura was noted. Pleural biopsy was unhelpful. Pleural fluid cytology was suggestive of mesothelioma. Her disease progressed and she died nine months after her presentation.

Post mortem examination confirmed the cause of death to be pleural mesothelioma. Histological examination showed a tubulo-papillary pattern (Plate 1). Histochemical stains showed a negative reaction for epithelial mucin (AB DiPAS) and a negative reaction for carcinoembryonic antigen (CEA). The histological diagnosis was made unequivocally and independently by two pathologists experienced in the diagnosis of mesothelioma. No asbestos bodies were observed on light microscopic examination of 30μ thick sections of lung.

Transmission electron microscopy of digested tissue showed 31 million fibres per gram of dry lung and 12 million fibres per gram of dry tumour. A count of

Received in revised form 11 July 1988

Correspondence to R. M. Rudd, The London Chest Hospital, Bonner Road, London E2 9JX, U.K.

0954-6111/89/030237+03 $03.00/0

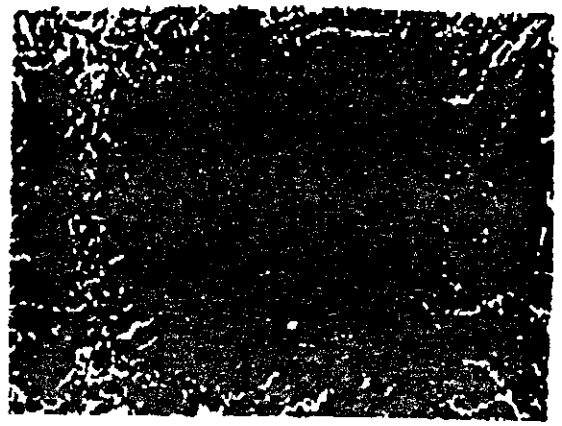
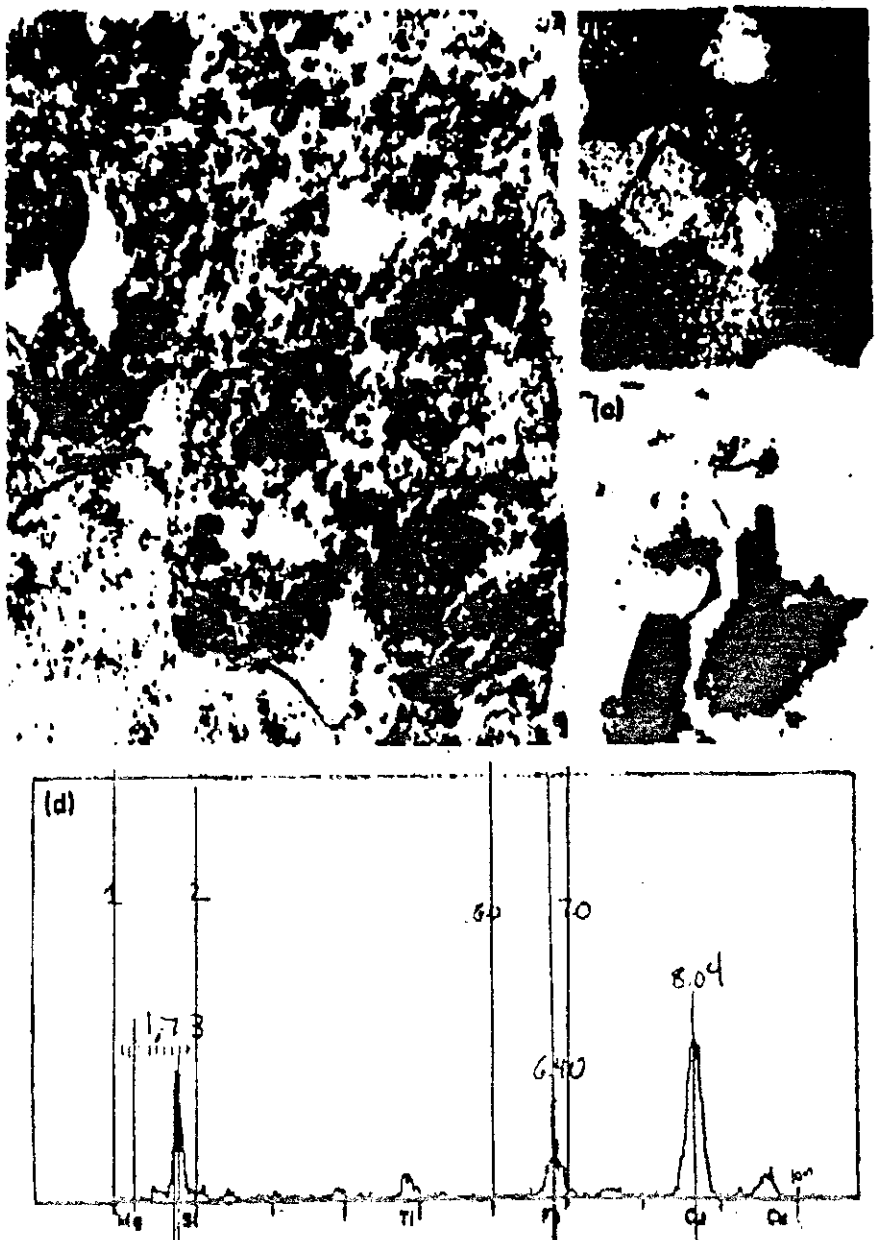


Plate 1 (a) A section of the mesothelioma stained with haematoxylin and eosin and shown at low power (microscope magnification × 150). This demonstrates the tubulo-papillary pattern. (b) A section of the mesothelioma stained with haematoxylin and eosin and shown at higher power (microscope magnification × 300). This shows an area of the tumour with a sarcomatous pattern.

500 fibres in the lung digest demonstrated 90% to be morphologically compatible with amosite asbestos. The rest had the appearance of glass or other non-asbestos fibres. Energy dispersive X-ray microanalysis (1) of a random selection of 40 fibres thought on morphological grounds to be amosite showed in each case an elemental composition compatible with amosite (Plate 2). In addition some bundles of fibres were seen and X-ray micro-analysis of these demonstrated the presence of titanium, a common constituent of paint.

The patient had not lived in proximity to an asbestos factory and she had not lived in a household including an asbestos worker. She had always been an



Mg=1.25

Plate 2 Electron microscopy and energy dispersive X-ray analysis. (a) Lung tissue section showing fibres (×16 000) (b) Fibres in a digest of lung tissue (×32 000) (c) Fibres in a digest of tumour tissue (×32 000) (d) X-ray analysis showing elemental composition of silicon, iron, and magnesium compatible with amosite. The titanium peak may be derived from inhaled paint fragments. Copper calibration peaks are also evident.

office worker and she had never engaged in any employment recognized to be associated with asbestos exposure. The only history of asbestos exposure related to the office block in which the patient worked from the time of its construction 15 years before her death until she became ill 14 years later. The ceilings of the building were sprayed with an inch thick layer of soft asbestos covered by a layer of paint. This evidently deteriorated rapidly, since as a result of complaints about dust in the offices, the ceilings were sealed, repaired and repainted about eight years after the building was constructed. The patient's husband, a painter and decorator, was engaged to spray the ceiling. Once a week he took his overalls home and his wife put them in a bag for the laundry. She did not launder them herself and she is unlikely to have obtained any significant exposure to asbestos dust from this activity which occurred only seven years before her death. Close questioning of her husband did not reveal any previous history of having worked with asbestos and he confirmed that his wife had never laundered his overalls herself.

Some years later material from the ceiling of the building became dislodged during electrical maintenance works. Analysis of a sample of the material by dispersion staining and polarising light microscopy showed it to consist of 70% amosite asbestos.

Discussion

We are not aware of previous reports of mesothelioma resulting from exposure to asbestos in buildings. The 14 year interval between the onset of exposure and presentation with symptoms of mesothelioma is towards the lower end of the observed range but certainly within it (2). No previous asbestos exposure could be demonstrated.

No data are available on the airborne fibre counts in the building concerned but the history suggests that environmental contamination may have been considerable. The electron microscopic asbestos fibre count in the lung tissue was within the range found in persons with occupational exposure to asbestos (3). The identification of the asbestos in the lung and tumour tissue as of the same type as that found in the sample of material from the office block supports causation of the tumour by exposure to asbestos in the building in which the patient worked.

The propensity for fibres to become coated to form asbestos bodies varies with the dimensions of the fibres and between individuals (4). In this case light microscopy failed to identify asbestos bodies in tissue sections but electron microscopy demonstrated substantial numbers of uncoated fibres, illustrating the value of the technique in establishing the relation between asbestos exposure and subsequent disease.

The existence of a health hazard resulting from asbestos in buildings has been assumed by extrapolation from data on industrial exposure. Doll and Peto (5) suggested in a recent review that the number of deaths in the UK resulting from the use of asbestos in buildings is likely to be negligible. They emphasised that their predictions were based on assumptions that exposure would be confined to chrysotile, which is probably a less potent cause of mesothelioma than amphiboles, and that fibre counts would be very low when asbestos coatings are well maintained. This case illustrates that proximity to poorly maintained asbestos in buildings can result in exposure of a degree sufficient to cause serious disease.

Acknowledgements

JYK was funded by the Society for the Prevention of Asbestos and Industrial Diseases, 38 Drapers Road, Enfield, Middlesex.

References

1. Pooley FD. The identification of asbestos dust with an electron microscope microprobe analyser. *Ann Occup Hyg* 1975; 18: 181–186.
2. Greenberg M, Lloyd Davies TA. Mesothelioma register 1967–8. *Br J Ind Med* 1974; 31: 91–104.
3. Wagner JC, Pooley FD, Berry G *et al.* A pathological and mineralogical study of asbestos related deaths in the United Kingdom in 1977. *Ann Occup Hyg* 1982; 26: 423–431.
4. Morgan A, Holmes A. Concentrations and dimensions of coated and uncoated asbestos fibres in the human lung. *Br J Ind Med* 1980; 37: 25–32.
5. Doll R, Peto J. Effects on Health of Exposure to Asbestos. Health and Safety Commission. London: Her Majesty's Stationery Office, 1985: pp. 53.