Case Reports

Pleural Mesothelioma and Neighborhood Asbestos Exposure

Findings From Microchemical Analysis of Lung Tissue

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IN 1978, we reported a case of malignant pleural mesothelioma with unusually long survival. In that report we detailed histopathologic and immunologic observations that may have been related to the long-term survival.[1]

See also pp 68, 91, and 96.

In this article, we summarize the clinical course and relate the probable etiology of the disease to an unusual type of asbestos exposure.

Report of a Case

A male patient, born in 1924, was well until the beginning of 1970, when he noted a gradual increase in shortness of breath and sharp pain in the right side of the chest. He experienced repeated pleural effusions and was found to have malignant pleural mesothelioma. He was not given chemotherapy or any specific treatment for the disease, which showed a very slowly progressing course. In December 1979, there was a rapid deterioration in the patient's clinical status, with marked progress of the chest roentgenographic abnormalities (Fig 1). The patient had respiratory failure and died at the beginning of January 1980.

The occupational history did not reveal any direct occupational exposure to asbestos, but the patient had been employed in a factory adjacent to a major navy yard in New York City from 1957 to 1966. During that period, the activity of ship building and ship repair was high at this navy yard.

A section of lung parenchyma was digested in a solution of sodium hypochlorite, and the residue, including inorganic mineral particles, was prepared for analysis by polarized light microscopy (PLM) and transmission electron microscopy (TEM).[2] The residue observed by PLM contained both coated fibers (ferruginous bodies) (Fig 2) and uncoated crystalline fibers (Fig 3), possibly amphibole fibers. They were 2 to 3 μm in length and 1 μm in diameter and could not, therefore, be positively identified by PLM.

The residue was, therefore, also analyzed by TEM. A large number of amphibole asbestos fibers were observed that were both uncoated and coated with ferritin granules (asbestos bodies). The amphibole identity of fibers was further confirmed by selected-area electron diffraction. Microchemical analyses were obtained on both fibers and asbestos bodies. The uncoated amphibole fibers were identified as amosite, as were the cores of the asbestos bodies. Microchemical analysis and morphological characterization also confirmed the presence of lesser amounts of chrysotile asbestos.

Comment

Asbestos-related disease is emerging as a major public health problem in many countries throughout the world. Widespread use and occupational exposure to asbestos in US shipyards, particularly during World War II, is one reason for the currently high incidence of asbestos-related diseases, including lung cancer and mesothelioma.[3] There is typically a long latency period between asbestos exposure and resulting disease. The disease-causing potential of airborne asbestos has been proved to reach beyond factory gates, and cases of mesothelioma have been reported in persons residing in the vicinity of asbestos-using industries.[4,5]

This case report lends additional credence to our earlier suggestion that exposure to asbestos in the neighborhood of the shipyard may be related to the development of malignant mesothelioma in this particular

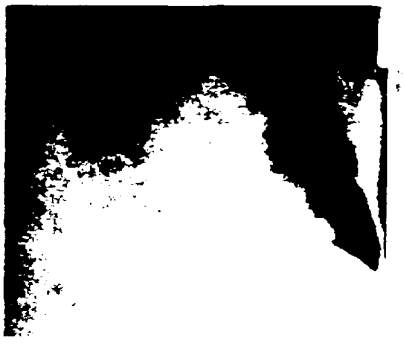

Fig 1.—Chest roentgenogram showing advanced stage of mesothelioma. Right lung is almost completely encased by pleural tumor. Note "scalloping" of pleura of left side—typical roentgenographic feature of mesothelioma.

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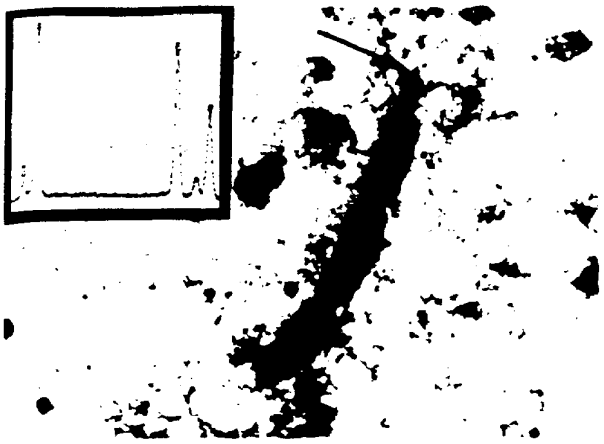

Fig 2.—Electron photomicrograph of asbestos body in lung tissue, entirely coated with ferritin except upper tip (arrow) permitting the core fiber to be identified as amosite by microchemical analysis (inset).

Fig 3.—Electron photomicrograph of amosite fiber found in lung tissue. Fiber about 6 μm in length is identified by its microchemical analysis (inset) and crystal structure analysis.

patient. The identification of amosite asbestos fibers in the lung tissue of our patient provides plausible evidence for this etiologic connection. Amosite asbestos is not found in the lungs of persons from the general population, and its occurrence, therefore, indicates either an occupational exposure or an exposure to a specific environmental source. Although only a very small portion of the total amount of asbestos used consists of amosite, this asbestos type is commonly used in shipbuilding and repair and was used a great deal in the shipyard adjacent to which our patient worked. Of added interest is a previous but unpublished observation of ours of a patient who resided a few blocks from the same shipyard and who experienced the development of malignant mesothelioma at the age of 29 years. In that case, there was potential for neighborhood asbestos exposure since childhood, which explains the young age of that patient; amosite asbestos fibers were also found in the lung parenchyma.

References

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Hypotension and Cardiopulmonary Arrest Associated With Concurrent Haloperidol and Propranolol Therapy

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HALOPERIDOL exerts its antipsychotic effect presumably through potent blockade of central dopamine receptors[1] and has caused hypotension in some patients.[2] Propranolol hydrochloride is a β-adrenergic receptor blocking drug that is widely used in treating hypertension. A review of the literature does not disclose adverse effects in patients from a combination of haloperidol and propranolol. We report herein a patient who had three episodes of hypotension and two episodes of cardiopulmonary arrest after taking concurrent oral doses of haloperidol and propranolol.

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Report of a Case

A 48-year-old white woman with a 27-year history of schizophrenia was first admitted to our hospital in 1980. At that time, the patient was treated with