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CARCINOMA OF THE LUNG IN A DRYWALL TAPING WORKER REPORT OF A CASE

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SUMMARY

Squamous cell carcinoma, together with pulmonary asbestosis is reported in a drywall taping worker. In this trade vigorous sanding of dried taping compounds produces a dust which is known to contain chrysotile asbestos. Fibrils of chrysotile asbestos were seen on transmission electron microscopy of ashed lung tissue. The authors emphasize the need to institute adequate preventive measures in this trade.

INTRODUCTION

New industrial processes may considerably widen the range of occupational health hazards. Drywall construction was introduced in the United States on a large scale basis immediately after World War II. During the past two decades, drywall construction has achieved a dominant role in the United States construction industry. This trade has recently been described by us in detail, and has been shown to be associated with significant asbestos exposure [1,6]. The occurrence of a pulmonary carcinoma in a drywall taping worker with the finding of very large numbers of asbestos fibers in the lung raises the possibility that this trade may soon become the source of asbestos neoplastic disease, in addition to asbestosis.

Dust diseases of the lung may be diagnosed in different ways and with various degrees of accuracy. In order to achieve definitive diagnosis, the identification of the hazardous agent is required. The substance may be found in the material used in a work process, in air samples, or in human tissues, as a result of exposure.

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We report here a case of lung cancer in a drywall taping worker as well as the results of tissue analysis for asbestos fibers and other inorganic materials. The importance of utilizing adequate techniques for tissue analysis is stressed.

CASE REPORT

E.S., a 57-year-old white male, was admitted to another hospital in May 1971 because of weight loss, chest pain and sudden hemoptysis. Two weeks before, a chest radiograph had shown an infiltrative process in the left upper lung field. This persisted despite antibiotic therapy and the patient was admitted for further study.

Bronchoscopy was negative for malignancy, and cytological examination of bronchial washings showed only atypical cells. Scalene node biopsy was unremarkable. A left upper lobectomy was performed. A portion of the fourth rib was adherent to the pleural surface on the anterior lateral aspect. Section of the lung revealed an area of grayish consolidation beneath the rib adhesion. Macroscopically, the area was suggestive of a neoplastic tissue. There was no involvement of the lower lobe. The patient was followed for 1 year as an outpatient, developed symptoms suggestive of cerebral metastases, and died on May 23, 1972. No autopsy was performed. Past medical history had been unremarkable. The patient had been a heavy smoker and had been a drywall construction worker for 25 years, mixing, applying and sanding various spackling and taping compounds.

Histopathological examination

The tumor tissue in the surgical specimen from the peripheral part of the lung



Fig. 1. The peripheral part of the lung is shown with thickened neoplastic tissue and fibrotic alveoli. 270 ×

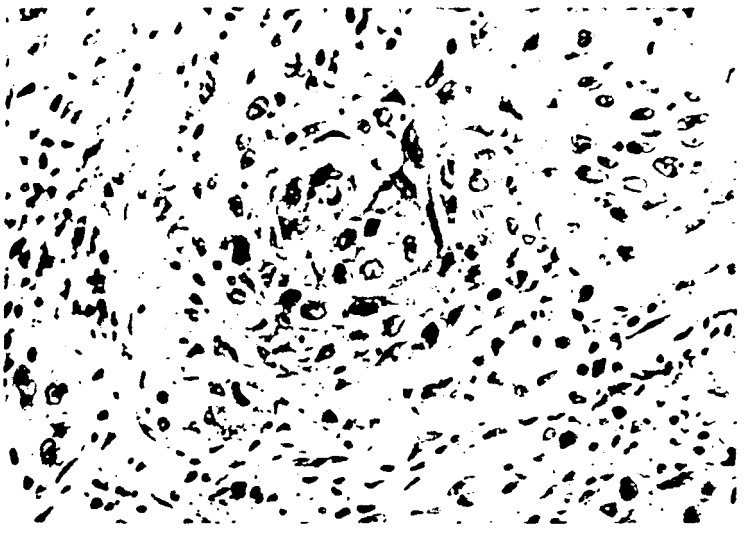


Fig. 2. Tumor tissue with pearl formation. 1700 x

was a well-differentiated squamous cell carcinoma (Fig. 1). The neoplastic cells were irregular in shape and formed nests of various sizes. Occasionally pearl formation was observed (Fig. 2). Polymorphism, atypical features and abnormal mitosis was remarkable. There was invasion of the tumor into the pleural region with much fibrosis.

In addition to the neoplastic changes described above, the pulmonary parenchyma showed congestion, edema and broncho-pneumonia, which was partially organized. Alveolar macrophages had accumulated in alveolar spaces. Hemosiderin and anthracotic pigments were frequently seen in the cytoplasm of the macrophages.

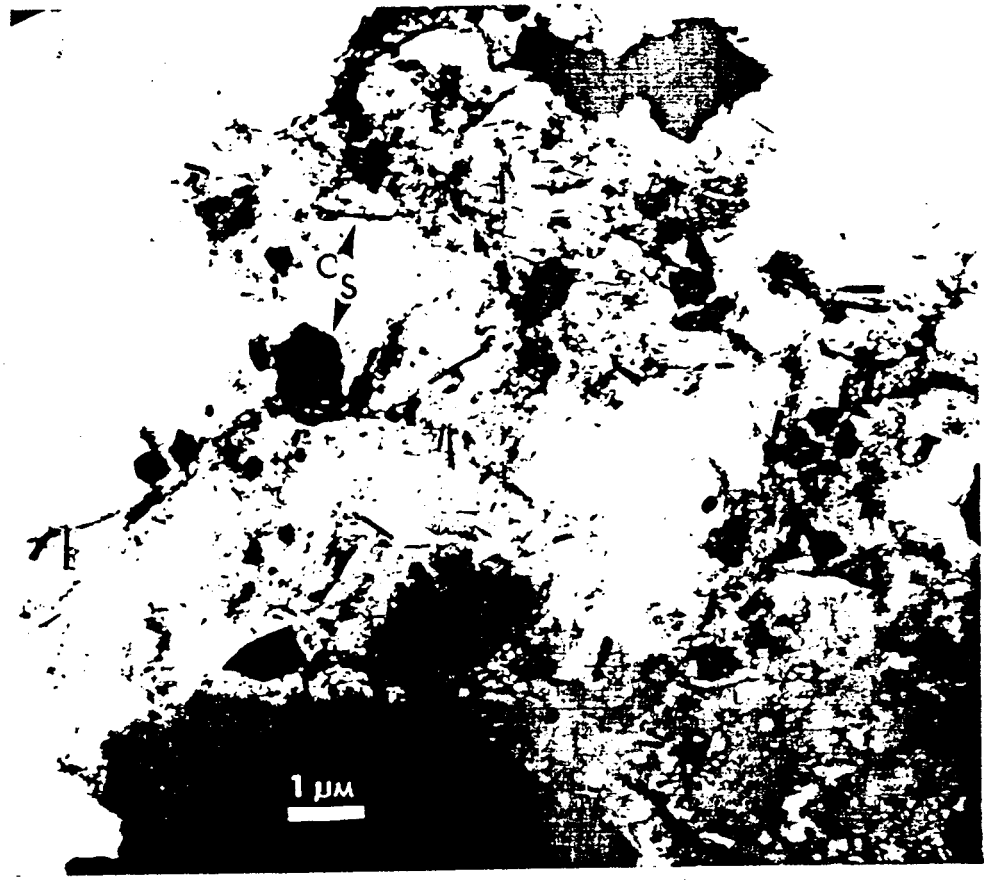
Of interest was the presence of pulmonary fibrosis, in the form of alveolar septal (Fig. 3) and peri-bronchiolar fibrosis. This fibrosis, consistent with pulmonary asbestosis, was not accompanied by the finding of asbestos bodies either in the alveolar space or in the fibrotic tissue itself.

Mineralogical analysis of lung tissue

A 5-μm thick unstained, uncovered, tissue section was prepared for electron microscopy by the carbon extraction technique [4]. The prepared specimen was examined by transmission electron microscopy utilizing an RCA EMU-3G. Scan magnification ranged from approx. 2000—31 000 ×. At magnification greater than 15 000 ×, particles too small to be seen by light microscopic methods became visible. The appearance of numerous particles by electron microscopy, invisible by all other microscopic techniques, has been described [5]. Everywhere in the relic tissue numerous particles were observed (Fig. 4). Sheet silicates, diatom fragments and chrysotile asbestos were observed in



Fig. 3. Alveolar septal fibrosis. 1600 x



nearly all fields of view. Chrysotile fibers tended to be present as fibrils, with fibers rarely observed. Identification of the asbestos was based upon morphological characteristics as described previously [2,3]. A number of other particles, crystalline in nature, were present in the lung tissue. Selected area electron diffraction patterns obtained of these particles were not symmetrical and therefore no attempt was made to identify them. The average particle size of these particles was less than 1 μm in greatest dimension. The number of these particles in the tissue specimen indicated an occupational exposure to dust.

The mineralogy of spackling, taping and patching compounds has been reported [6]. Sheet silicates (e.g., talc or clay) and chrysotile asbestos are common components. The occupational history of the patient was consistent with the very large number of mineral particles in the lung tissue.

DISCUSSION

The identification of asbestos in drywall spackling and taping compounds and the considerable prevalence of asbestosis among those who use these materials has added a new occupational perspective to industrial drywall construction. This trade is of growing importance in the United States construction industry. At the present time, some 75 000 individuals are directly working in this trade.

The case reported here illustrates the importance of tissue analysis in the evaluation of dust disease of the lung. Histological study was adequate to define the presence and nature of the neoplasm but failed to demonstrate the concomitant dust burden of asbestos and other minerals. The particles were too small to be seen by optical microscopy. They were readily demonstrated by electron microscopy.

The very small size of the chrysotile fibrils (300 Å × 0.5 μm) found by electron microscopy and the virtual absence of large fibers is consistent with important pathogenicity of such small fibers. The fibrils found in our case may be derived from various sources. The asbestos content of taping compounds primarily consists of fibers in the size range of 0.25 μm to 8.0 μm [6]. The work performed by the patient, however, included vigorous sanding of dried taping compounds. The reduction in fiber size may thus be a result of fragmentation which occurs during this procedure.

The biological fragmentation of inhaled larger fibers in vivo may also be a source for the small fibers. The diagnosis of asbestosis (alveolar-septal or peribronchiolar fibrosis) was of importance, and gives evidence for the association of drywall taping and asbestosis.



Fig. 4. Transmission electron micrograph of ashed lung tissue. The particle population, associated with relict ashed tissue remnants, consists primarily of chrysotile fibrils (C) and sheet silicates (clays and micas, S). Particle identification is based on morphological characteristics and selected area electron diffraction patterns. The amount of inorganic debris present is high and consistent with an occupational exposure to dust; average particle size of the dust is less than 1 μm, in greatest dimension. All chrysotile fibers and fibrils, some fifty in this area, are less than 1 μm in length. Scale as marked.

The carcinogenic potential of asbestos has been well-documented during the past three decades. Lung cancer is one of the most important asbestos-associated neoplasms. It has also been well-established however, that asbestos-associated lung cancer does not depend on asbestos alone, but on the combined effect of cigarette smoking and asbestos exposure [7]. The concordance of those two exposures may possibly also explain the occurrence of pulmonary carcinoma in this asbestos-exposed individual.

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