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Pleural Mesothelioma After a Short Interval From First Exposure in the Wine Filter Industry

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Pleural mesotheliomas are usually reported after a long interval has passed since first exposure to asbestos. We, however, describe a case observed after a latent period of only 7.5 years in a worker exposed in a factory manufacturing auxiliary products for wine treatment, including chrysotile asbestos filters. The exposure to asbestos lasted 3–4 months per year, during which airborne fiber concentrations ranged from 1 to 4 ff/cc in the patient's workplace. Due to the characteristics of the manufacturing process, the asbestos fibers were very thin in diameter. The patient also suffered from nasal breathing impairment. An examination of the literature showed that asbestos-related mesotheliomas have been reported, albeit rarely, after less than 10 years from onset of exposure. Therefore, it is believed that this case should be related to past exposure to asbestos.

Key words: pleural mesothelioma, latent period, chrysotile asbestos, wine filtering, occupational exposure

INTRODUCTION

In a recent paper, Langer and McCaughey [1982] report a case of mesothelioma in a brake repair worker 27 years after the first exposure to chrysotile. Here we describe a case of pleural mesothelioma observed in a worker exposed to chrysotile asbestos used for wine filtering, with a latent period that is much shorter—7.5 years from first exposure to onset of cancer. This type of exposure is uncommon; the case is worth describing because of the nature of the exposure and because of the short interval between first exposure and tumor manifestation.

Occupational exposure to chrysotile is believed to have caused far fewer cases of mesothelioma than that of crocidolite and amosite or to mixed exposure including amphiboles [Anonymous, 1981]. This belief is mainly based on the results of epidemiologic studies on chrysotile miners and millers: Among 332 deaths at the Balangero Mine, Northern Italy, there was only one suspected case of pleural mesothelioma after a long follow-up period [Rubino et al. 1979]; 10 cases were definitely attributable to chrysotile among 4,463 deaths in a Quebec study [McDonald et al. 1980]. In addition, recent studies on friction material [Newhouse et al. 1982] and asbestos textile industries [McDonald et al. in press] where amphiboles were never used, show comparably low mesothelioma rates.

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However, animal experiments carried out by intrapleural injection of suitably sized mineral fibers showed that the carcinogenic potential of chrysotile, amphiboles, and other mineral fibers, whether naturally occurring or man-made, were all similar. These experiments have clearly demonstrated the importance of the size of the fibers, those ranging from 0.5 to 2 μm in diameter and from 5 to 50 μm in length being the most carcinogenic. The experiments, however, did not take into account differences in lung penetration, which, depending also on fiber shape, is less for curvilinear chrysotile than for amphiboles [Stanton and Wrench, 1972; Pott, 1980].

Crocidolite is still used in the asbestos cement industry and up to the 1960s was used in textile and friction material manufacture, often situated in the same factory. Therefore, since exposure to chrysotile alone was until recently quite rare in asbestos manufacture, it is difficult to provide definite epidemiologic evidence that chrysotile fibers are less hazardous, as far as mesothelioma is concerned.

CASE DESCRIPTION

The patient was born March 11, 1933. He smoked six or seven cigarettes per day up to 3 years before the tumor became manifest. He had worked as an agrarian expert in several farms and sugar refineries. Questioning of the patient failed to demonstrate any known previous asbestos exposure in these jobs. There was also no history of family contact exposure.

Beginning in 1971, the patient was employed in a factory manufacturing auxiliary products for wine treatment, where chrysotile asbestos was used for filtering. There was no relevant past medical history, prior to the appearance of early symptoms of the tumor, except malaria at age 9, allergic rhinitis, and two operations for nasal polyps.

Chest X-rays were carried out in October 1975, April 1977, and December 1977 and showed no abnormalities. In addition, spirometry, performed in December 1977, was normal.

In February 1979, he developed acute left chest pain, dyspnea, and fever. Chest X-rays then revealed left pleural effusion, and the patient was admitted to the hospital. Erythrocyte sedimentation rate was 47. Tine test was negative, and ECG was normal.

After 1 week of hospitalization, approximately 1 liter of hemorrhagic fluid was removed. Thereafter, thoracentesis was needed once a week. Tomography revealed lobulated shadows on the left chest wall. Cytological examinations of pleural fluid on three aspirations showed atypical mesothelial cells, which appeared isolated or grouped in papillary or pseudotubular clumps.

No changes were found on bronchoscopy. Spirometry was still normal. Therefore, he underwent decortication and the diagnosis of mesothelioma was histologically confirmed. Indeed, at microscopic examination the tumor showed typical tubulopapillary structures (Fig. 1) with some sarcomatoid areas. The epithelial-like cells presented characteristic hobnail features.

The patient died in September 1979.

WORKROOM DESCRIPTION

Asbestos processing began in 1971. The operation consisted of opening the fibers in order to increase their surface for filtering purposes and was carried out for

Fig. 1. The pleural tumor shows tubulopapillary structures typical of mesotheliomas (H.&E., original magnification, ×65).

3–4 months a year, during which some 100 tons of filtering material were produced. Asbestos fibers were mostly delivered in bags of 1–25 kg to wine factories, where the filters themselves were prepared. A small part was mixed with sulfur in the factory to obtain, by moulding, small disks to be burned in the vats into which sulfur dioxide is then released. Three workers usually carried out this process in a workroom which is about 65 m long and 13 m wide.

One worker was assigned to a two-level fiber-opening machine. Manual bag opening and machine loading of raw fibers were performed at the higher level, whereas filling and weighing of bags containing the opened fibers were carried out at the lower level. The other two workers (one was the patient described here) were then assigned to recording and checking the raw material at entry and the final products to be delivered.

Airborne dust measurements conducted by one of the authors in 1977 [Scansetti and Botta, 1980] showed the following fiber concentrations: in excess of 22 ff/cc at bag opening (upper level); 12–15 ff/cc on both sides of the scale (lower level); 4 ff/cc at 1 m from the scale itself, and 1 ff/cc in the surrounding environment.

At the time these measurements were made, an individual protection mask was supplied to only one worker, the one assigned to the machine. This man is at present suffering from asbestosis and has obtained compensation through the National Insurance Institute.

Processes involving asbestos handling were discontinued in 1978.

DISCUSSION

The exposure to asbestos fibers in the patient's workplace cannot be considered a heavy one and, as noted before, the fiber opening process lasted for 3–4 months per year. There are no available data on dust concentration in the remaining months, but residual dusts tend to linger. This is, therefore, a case of intermittent exposure to asbestos.

On the other hand, the patient was exposed to very thin fibers, which may more easily migrate to the pleura. He also had suffered from allergic rhinitis and nasal polyps requiring surgical treatment. This is likely to have increased the dust lung deposition, since it is known [Chan and Lippman, 1980; Lippmann et al., 1980] that nasal filtering is more efficient in normal subjects (without rhinitis or polyps) especially when air flows are low, as in the case of jobs not requiring effort.

In this patient, the interval between first exposure to asbestos and symptoms was only 7.5 years, whereas pleural mesothelioma generally is known to occur after a long latent period, often after 35 or more years. Although a lower limit for such a latent period cannot be established, it is usually reported to range from 15 to 20 years; with few exceptions a latent period of under 10 years has been reported when heavy exposure is present [Bohlig and Otto, 1975].

The British Mesothelioma Registry [Greenberg and Lloyd Davies, 1974] included 167 cases of mesothelioma with a history of asbestos exposure, among which only one case had an interval between first exposure and death shorter than 10 years (namely, 3.5 years). Cochrane and Webster [1978] reported 69 cases related to crocidolite out of 70 examined. In only one case was the interval to onset of symptoms 9 years.

Similarly, the French Registry [Bignon et al, 1979] includes only one case out of 157 exposed to asbestos in which the exposure started 10 years before death. The National Cancer Registry of the German Democratic Republic identified 915 cases [Konetzke et al, 1981]; among these 332, ie, 36.7%, had had a professional exposure to asbestos; in 6 cases, ie, 1.8%, the latent period between first exposure and tumor manifestation was shorter than 10 years.

In conclusion, even though there have been cases reported in the literature, albeit rarely, with a short latent period between first exposure and appearance of cancer, the present case, in our opinion, should be related to past asbestos exposure.

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