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Pleural Mesothelioma and Household Asbestos Exposure

Joachim Schneider, Kurt Straif and Hans-Joachim Woitowitz

Institut und Poliklinik für Arbeits– und Sozialmedizin der Justus–Liebig–Universität Giessen Aulweg 129/III 35385 Giessen, Deutschland

SUMMARY

This article discusses the development of asbestos-induced malignant mesotheliomas after non-occupational environmental exposure to asbestos through contact with occupationally exposed household members. In our policlinic, we have seen six fatal pleural mesothelioma cases (five wives and one son of asbestos-industry workers) with no history of occupational asbestos exposure. In five women, a causal relation was established between the fa[illegible] disease and inhalation of asbestos fibers while cleaning the contaminated work-clothes and shoes of their husbands at home. The son had also been exposed to asbestos throughout his childhood during daily visits with his father at the workplace.

INTRODUCTION

Asbestos-induced mesothelioma (No. 4105 of the German Ordinance of Occupational Diseases (BeKV) is one of the most frequently compensated occupational cancers /1/. Malignant mesothelioma is specifically regarded as a "signal tumor" of previous exposure to asbestos in the workplace /2/. To our knowledge, causes other than asbestos and the comparably bio-persistent mineral-fiber Erionite have not been found /3,4/. Mesothelioma is considered a marker for non-occupational asbestos exposure as well /5,6/.

Asbestos-induced mesotheliomas have been described in the vicinity of asbestos mines in South Africa /7,8/, Canada /9/, Australia /10,11/, and C[illegible]s /12/. Industrial asbestos manufacture also presents a significant health risk to the environment. In Saxony /13/, and especially in the Hamburg–Bergedorf area, the incidence of mesothelioma cases was significantly increased in areas that are close to industrial asbestos emitters /14-17/. Many reports have emerged from Turkey /18-20/, the northwestern part of Greece /21/, and Corsica /22/, /23/ about the endemic incidence of diffuse malignant mesotheliomas, caused by environmental exposure to asbestos or Erionite through soil erosion. In certain regions of central Anatolia, pleural-mesothelioma incidence figures of up to 216 per 1,000,000 inhabitants have been recorded, although the expected mesothelioma incidence in the general population was very low. The estimate of an annual mesothelioma mortality rate of 1 to 8 per one million inhabitants /24-26/ relates to about one mesothelioma death per 1000 to 10,000 deaths from all causes. An article by Grossgarten and Woitowitz /6/ presents an overview of asbestos-induced pleural mesotheliomas that are linked to indoor inhalation by household contacts.

PATIENTS AND EXPOSURE CRITERIA

In our policlinic, we have seen asbestos-induced tumors in members of households where at least one worker had been occupationally exposed to asbestos. Between 1986 and 1994, five women and one young man (one patient's son), with no occupational history of asbestos exposure, died of asbestos-induced mesothelioma. Their ages ranged from 42 to 68 years (Table 1). The diagnosis was always confirmed by histology on specimens obtained from clinical biopsy or autopsy. Detailed job histories revealed that neither the women nor the son had ever worked in an asbestos factory. Asbestos exposure was exclusively through residential inhalation of asbestos from contaminated work clothes or shoes that were brought home from the workplace by the husband. Throughout his childhood, the son had regularly delivered a hot

Table 1.
Asbestos-induced pleural mesothelioma in six wives and one son of asbestos-industry workers

Patient	Born	Died	Length of household exposure	Latency (years)	Occupation of household contact
G.J.	1923	1986	1950–59	35	Insulation-mat manufacturing
E.L.	1925	1993	1961–84	31	Turbine revision
H.B.	1926	1994	1954–71	38	Roofer (asbestos/cement)
M.L.	1931	1987	1969–76	17	Asbestos cardboards
G.K.	1933	1992	1964–74	26	Insulator
H.J.*	1946	1992	1950–59	42	Insulation mats manufacturing

*Son of G.J.

meal to his father at the workplace. From 1950 to 1984, the husbands were employed in different companies that manufactured asbestos mats, asbestos textiles, and asbestos cardboard, or in such occupations as insulators, roofers, or turbine-revision workers who process asbestos products. Thus, the asbestos exposure at such jobs is indisputable. The husbands of two patients, G.K. and G.J., had already developed pulmonary asbestosis. The asbestos-contaminated clothes were not removed in the factory for cleaning but rather were taken home, where the wives brushed them almost daily and hand-washed them once or twice a week. Residential fiber measurements are unavailable. The length of the household exposure was between 7 and 23 years (see Table 1). The latency period (time between onset of the residential asbestos exposure and the development of the disease) varied from 17 to 38 years.

DISCUSSION

For almost three decades, the international scientific literature of environmental and occupational medicine has published papers describing asbestos-induced diseases in family members of workers who have been occupationally exposed to asbestos /5,6/. In the United States, the air concentration of chrysotile was measured in the houses of 13 asbestos miners who usually did not remove work clothing after working in the factory. About 75% of the air measurements in such households fell between 50 and >2000 ng/m^3 chrysotile /27/. For comparison, only 32 to 65 ng/m^3 chrysotile was measured in the homes of residents who were not employed in the mine. Measurements were also taken from the area where the work clothes were cleaned in an American asbestos textile factory. Between 10,000 and 80,000 (median 30,000) asbestos fibers of a length L >5 µm/m^3 air were found in the "cloth-cleaning area" /28/. Brushing asbestos contaminated work clothes results in asbestos-fiber concentrations of a few hundred crocidolite fibers per cm^3 /29/.

In 1989, Huncharek *et al.* /30/ reported that a shipyard machinist's wife, who had washed her husband's asbestos-contaminated work clothes at home, died of pleural mesothelioma. The post-mortem, lung tissue burden analysis yielded concentrations of chrysotile, amosite, and crocidolite, as well as tremolite, actinolite, and anthophyllite fibers per gram dry lung tissue of the same magnitude as that found in specimens from patients with occupational asbestos exposure.

Bianchi *et al.* /31/ found ferruginous bodies in the post-mortem lung tissue specimens of 73 mesothelioma patients, of whom 6 were asbestos workers' wives with only household exposure. Between 1,000 and 10,000 ferruginous bodies per gram dry lung tissue were measured in half the wives; as many as 10,000 to 100,000 were reported in one wife. Such concentrations are comparable to those found in 56 former shipyard-industry employees. Measurements showed between 1,000 and 10,000 ferruginous bodies per gram dry lung

issue in 20 shipyard workers, between 10,000 and 100,000 in 23 workers, and >100,000 in 10 workers. Low concentrations (<1,000 ferruginous bodies per gram dry lung tissue) were measured only in three shipyard workers and two of their wives.

The special risk for death from pleural mesothelioma in wives of asbestos-industry workers is well known from many international case reports /12,30-46/. The risk of mesothelioma death in household members has also been investigated in epidemiological case-control and cohort studies. In a case-control study in England, Newhouse and Thompson /47/ reported that 9 of 76 malignant mesothelioma patients did not have occupational asbestos exposure. Seven female patients had washed the work clothes of their occupationally exposed husbands and siblings. Two male patients were exposed at the respective ages of 9 and 13 years via their siblings who were working in an asbestos mill. The original publication did not distinguish between occupational and para-occupational exposure, however. A secondary analysis of household contacts gave a crude odds ratio OR = 16.75, with a 95% confidence interval of 1.36 to 136.78.

In a pairwise-matched, case-control study, Magnani and Polan /48/ described pleural and peritoneal mesotheliomas in 52 New York females. Of eight pairs, only the patients (seven wives and one daughter of occupationally exposed men) were exposed while washing asbestos-contaminated work clothes, whereas in one pair only the control was asbestos exposed. A secondary analysis excluding occupational mesotheliomas resulted in an odds ratio OR = 8.0, with a 95% confidence interval of 1.0 to 64.0. The authors presented the occupation of the male family members as the main risk factor for the disease.

A case-control study of McDonald /5/ (n=557 mesothelioma cases) revealed that in eight pairs, only the patients (five during childhood) had been exposed to asbestos via household contacts. By contrast, in two pairs, only the controls had a history of domestic exposure to asbestos. The calculated $\chi^2 = 3.03$ was not statistically significant ($p = 0.08$).

A cohort study of Anderson /49/ revealed that from 1941 to 1945, only three mesotheliomas were diagnosed among 2218 household members of amosite workers. The published data did not specify, however, the expected mesothelioma mortality rate, which should have been close to zero. Further, occupational asbestos exposure after 1945 cannot be ruled out from the information provided in this study.

In a cohort study by Magnani *et al.* /50/, 1740 female household members were monitored from 1965 to 1988. Overall, 210 deaths occurred versus 229.1 expected deaths. Four patients died of pleural tumors (three were confirmed mesotheliomas), which is significantly more than the expected rate of 0.5 (SMR=600). The 95% confidence interval of the SMR (215.9 to 2028.8) is statistically significant. The studies reviewed here confirm that despite certain methodological limitations, household members—predominantly wives—of asbestos workers, have an increased risk of dying from mesothelioma.

Other reports show that not only wives but also sons and daughters of asbestos workers have a high risk of dying from mesothelioma. In the Anderson cohort study on amosite workers /49/, for example, the three adult patients (a 40-year-old son and two daughters, aged 41 and 51 years /46/) who died from mesothelioma had been exposed to asbestos at home during their childhood.

Glage /51/ published one of the first reports from Germany on malignant mesothelioma, with a demonstration of asbestos fibers and ferruginous bodies, in the lung of an adult whose prolonged household exposure to asbestos had taken place during infancy. The patient was between three and seven years old when she was exposed to the asbestos-contaminated work clothes of her father, who cleaned carding machines. An occupational exposure of the daughter to asbestos was ruled out.

Kane *et al.* /52/ reported five adults under the age of 40 with pleural mesotheliomas. Residential asbestos exposure happened during childhood, exclusively through their fathers, most of whom were working in the shipyard industry.

Household exposure can begin at a relatively early age /53/. At least 5 of 668 mesotheliomas diagnosed in Canada between 1960 and 1975 and in the United States in 1972 can be linked to a household contact during infancy /9/. Konetzke *et al.* /54/ reported that of seven eight cases of

asbestos-induced diseases ensuing from asbestos exposure during childhood were attributed exclusively to non-occupational exposures. The published data, however, were not sufficient to determine whether the asbestos-induced diseases were mesotheliomas or merely radiologically diagnosed pleural plaques.

The contaminated work clothes of siblings also poses a risk. Newhouse *et al.* /47/ mentioned three mesothelioma patients, who at three years of age had been exposed to the clothing of sisters who were working in an asbestos mill. Between 1965 and 1992, many reports of mesothelioma cases that had been linked to contact during infancy with the work clothes of either fathers or siblings or both, were published in the United States (15), Canada (1), England (5), Germany (2), Sweden (2), Italy (2), and Australia (1) /9,32,36-37,42,44-47,51-52,55-59/; see /60,61/ for overview/. Occupations of the fathers/siblings comprised working in an asbestos-mill /47, an asbestos-factory /57/, an asbestos-cement-factory /56/, and with asbestos-products /46/; insulators /32,37,38/; carding-machine cleaner /51/, pipe lagger /55/, foundry worker /58/, steam fitter /40/; glass /52/ and shipyard industry /45,52/ workers; and a railroad worker /44/. Twenty-six patients had been exposed to fathers and eight to siblings.

Several reports have described a family clustering of asbestos-induced malignant mesothelioma in patients with household exposure /62,63/. A review by Dawson *et al.* /64/ covers 27 families comprising 64 (40 male and 24 female) mesothelioma patients; only 30 patients had a history of occupational asbestos exposure. The exposure of 11 patients was exclusively through household contact, 6 others lived near an asbestos factory, and 17 had no known asbestos exposure. Otte *et al.* /65/ described three mesothelioma patients in a single family, where family members had helped with packing an asbestos-cement product that was manufactured in a very dusty atmosphere in their home. In another report /58/, a pair of siblings who died of mesothelioma had been exposed to asbestos solely through the workclothes of the father, who worked in a foundry where asbestos textiles were used for heat protection.

Other articles describe mesotheliomas in multiple family members, mainly wives and children, with no occupational history of asbestos exposure. Krousel *et al.* /40/ and Li *et al.* /37, 42/ reported that wives and daughters of insulators died from mesothelioma. Like others, the exposure was due to inhaling asbestos while washing the work-clothes of the husband. During infancy, one daughter had reportedly worn diapers that were made from cotton sacks that had been previously used for storing asbestos.

We can infer from the evidence presented in this case series not only that mesotheliomas caused exclusively by household asbestos exposure are a problem in Germany now but also that such tumors will continue to appear in the future. During the period of reconstruction after World War II, the annual consumption of asbestos in the Federal Republic of Germany increased until the mid-1970s. In 1975 for example, 164,000 tons of raw asbestos were turned into about 1.5 million tons of asbestos-containing products in various industries /66/. Accurate estimates of how many workers were constantly or occasionally exposed to asbestos during the manufacture and use of asbestos-containing products are not available, but figures of >800,000 such individuals have been claimed /66/. About 45,000 workers were employed in industries processing raw asbestos, which is a plausible number. A relatively high fluctuation in dusty jobs can be assumed /4/. A valid estimate of household members at risk cannot be given.

In our policlinic, the latency period for the malignancy ranged from 17 to 36 years, which is typical for occupational pleural mesotheliomas /67,68/. The duration of the household exposures—7 to 23 years—agrees with the reported range of 2 to 41 years /47/. In England, shorter exposure periods preceding mesothelioma have been reported, but it should be noted that in such cases, the first year of exposure fell between 1912 and 1941, when occupational hygiene standards were rare.

The combination of short exposure periods and the long latency of asbestos-induced diseases underscores the significance of taking a detailed exposure history of mesothelioma patients who claim to have had no occupational asbestos exposure. Such patients should always trigger a suspicion of secondary asbestos exposure through household contact.

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