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Case Report from the Thoracic Services Boston University Medical School

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Asbestos-Related Disease from Household Exposure[1]

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Key Words. Asbestosis · Asbestos effusion · Pleural plaques · Pleural calcification · Mesothelioma

Abstract. The importance of nonoccupational asbestos exposure has been emphasized recently. To illustrate this problem, we report 4 persons with asbestos-related disease from household exposure. There were 2 wives of asbestos workers, who cleaned their husbands' work clothes. One developed a mesothelioma and the other plaques, calcification, benign asbestos pleural effusion and subpleural parenchymal fibrosis. 2 men were exposed as children while playing in a cellar room which was also used for their father's muffler repair business. At ages 27 and 33, they had pleural and diaphragmatic calcifications.

The lungs and pleura react to asbestos in several ways. This fibrous silicate causes asbestosis, that is, a chronic interstitial pneumonia and it is associated with lung cancer. Pleural complications include plaque formation, calcification, diffuse thickening, effusion, and malignant mesothelioma. These disorders may result not only from 'inplant' direct or indirect exposure, but also from 'out-of-plant' neighborhood or household exposure. We describe 4 cases of asbestos-related disease in family members of asbestos workers.

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Case Reports

Case 1

A 60-year-old woman was evaluated in September, 1973, because of acute, severe back pain. She had anorexia and weight loss of 2 months' duration, there was no dyspnea, cough, or hemoptysis, and she had never smoked cigarettes. She took intermittent medication for hypertension. Although there was no pulmonary disease in her family, two sisters had breast carcinoma. Since 1933, she had worked in a shoe shop, a woolen mill, a clothing factory and an electronic firm.

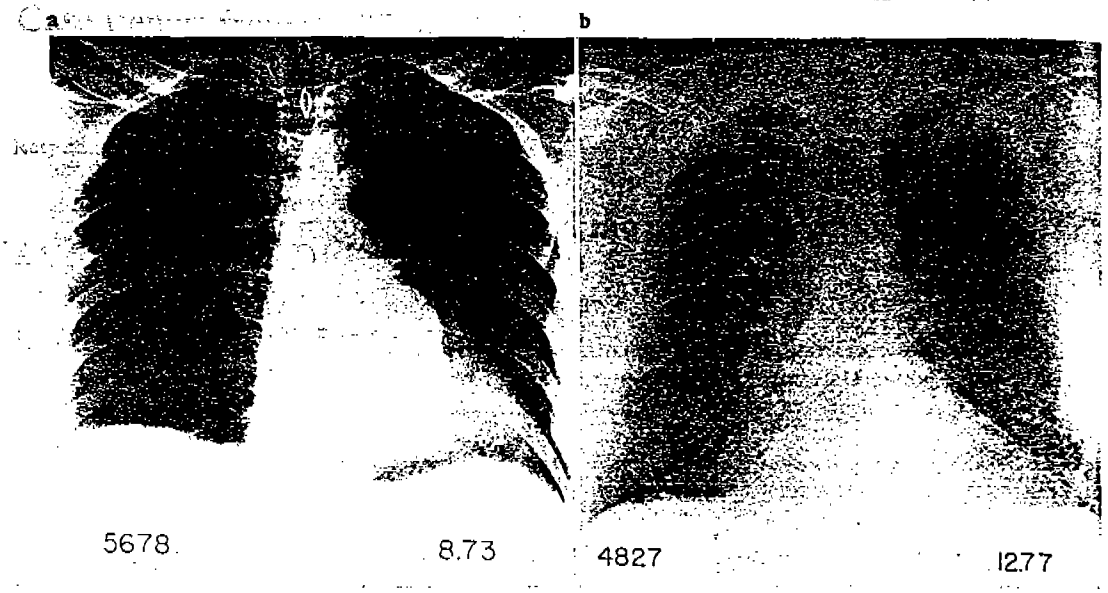

Fig. 1. a Overpenetrated chest roentgenogram of case 1, a 60-year-old woman. There is a 10-cm mass which, in the lateral view, proved to be behind the heart. Thoracotomy revealed an inoperable mesothelioma (fig. 2). b This roentgenogram is of her 55-year-old husband. It shows diffuse, linear, irregular opacities suggesting asbestosis.

On examination the blood pressure was 180/110. No rales were heard, a soft systolic murmur was present, and left periumbilical tenderness was detected. Her chest roentgenogram revealed a mediastinal mass (fig. 1a). A thoracic aneurysm was suspected and an exploratory thoracotomy was performed immediately. An inoperable tumor was found. She was readmitted 2 months later with left-sided weakness and died. Autopsy revealed a mesothelioma involving the left pleural surface, left lung, liver, abdominal cavity and brain. The tumor consisted of large cells which were frequently elongated or spindle-shaped and sometimes more rounded so as to suggest a biphasic pattern (fig. 2). The nuclei were quite pleomorphic and had coarse chromatin and some contained one or occasionally two very prominent eosinophilic nucleoli. The uninvolved pleura had hyaline plaques (fig. 3a). In the lung there were scattered asbestos bodies, but no diffuse interstitial pneumonia or fibrosis (fig. 3b, c). Because of these findings, a detailed exposure history was obtained from her husband. He was a machine operator in an asbestos product factory for 23 years and we had seen him annually as part of a respiratory survey. His examination revealed physiologic and roentgenographic evidence of minimal asbestosis with progression in 1977 (fig. 1b). The family lived on a farm 23 km from the factory, and the wife had never been in the plant. However, she had brushed white dust from her husband's work clothes twice weekly for many years.

Case 2

A 56-year-old woman saw her family physician in September, 1971, because of dyspnea while climbing stairs or performing housework during the previous 6 months. There was no chest pain, hemoptysis, cough or sputum. In 1957 a tuberculin test had been positive, but the chest roentgenogram was normal. She had smoked one pack of cigarettes daily since age 18, had no chronic medical problems, and did not take medication.

From 1942 to 1949, she packed munitions for an arms company and then worked in a woolen

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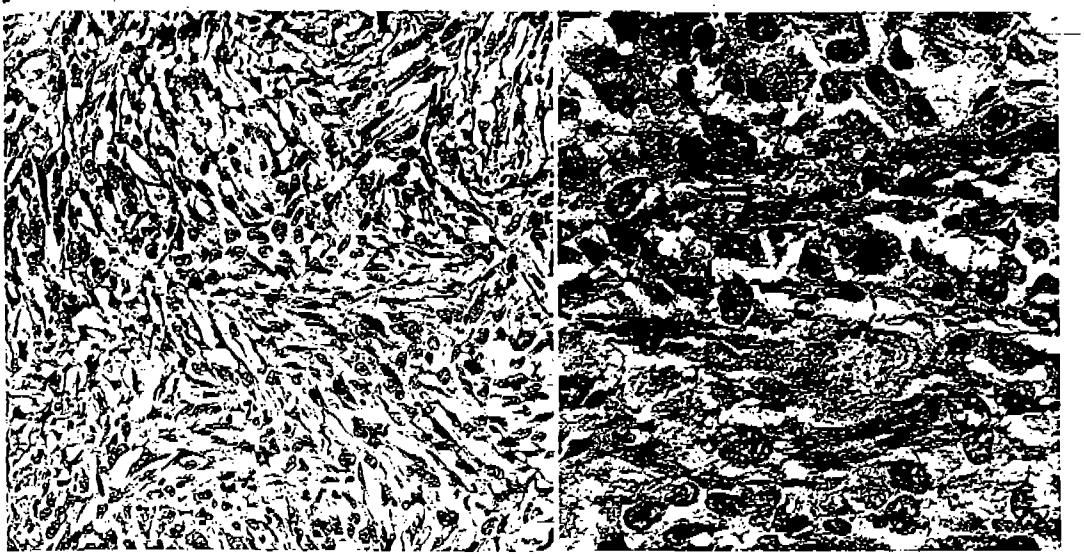

Fig. 2. Microphotographs of the mesothelioma of case 1. a Low-power view showing elongated or spindle-shaped cells arranged in interwoven bundles. b Higher magnification. The tumor cells are irregularly polygonal in shape, have abundant cytoplasm, and simulate an undifferentiated carcinoma. This pattern, and that seen in a, were intimately intermingled with clear transitional forms as well, thus suggesting a biphasic pattern. The nuclei have coarsely granular chromatin and prominent nucleoli. A mitotic figure is seen near the center of the right field. HE.

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mill for 2 years. From 1951 to 1956, she worked for a rubber shoe company. Talc was used in the manufacturing process; but, as an inspector, she worked in a separate area at the end of the line. Since then she had been employed in a hospital food service department.

Her chest roentgenogram revealed linear, irregular opacities in the lung bases and minimal bilateral pleural effusions. Therefore, she was admitted to a community hospital. The physical examination was recorded as normal. Evaluations for connective tissue diseases and tuberculosis were negative. A left thoracentesis yielded 3 ml of blood-tinged fluid with a total protein of 3 g/100 ml, a sugar of 42 ml/100 ml, and a white blood cell count of 1,300, mostly polymorphonuclear leukocytes.

A thoracotomy was performed to establish a definite diagnosis. The lung was adherent to the chest wall, and the pleura was 4–5 cm thick in some places. The biopsy showed alveolar wall hypercellularity, subpleural interstitial fibrosis and scattered asbestos bodies (fig. 4).

We first saw her in July, 1974, for disability evaluation. Dyspnea had persisted and a dry cough had developed. Physical examination revealed bilateral, fine, crackling rales. The chest film now showed linear irregular opacities that were graded 1/1 by the ILO U/C classification [1] (fig. 5a). Additionally, both costophrenic angles were blunted, and she had a long mediastinal calcification parallel to the right heart border. Physiologic studies showed a forced vital capacity (FVC) of 2.07 l or 73% of the predicted value. The forced expired volume in 1 sec (FEV_1) was 1.63 l, thus there was no evidence of airflow obstruction with an FEV_1/FVC of 79%. The expiratory reserve volume of 0.74 l and residual volume of 1.24 l were normal. Gas exchange was abnormal: the single breath diffusing capacity was low, 14.5 ml/min/mm Hg or 68% predicted, and steady state studies showed an elevated alveolar-arterial oxygen pressure difference $P(A-a)O_2$ of 49 mm Hg at rest and 45 mm Hg during exercise. Normally the steady state diffusing capacity (Dss) is 15 ml/min/mm Hg at rest and doubles to 30

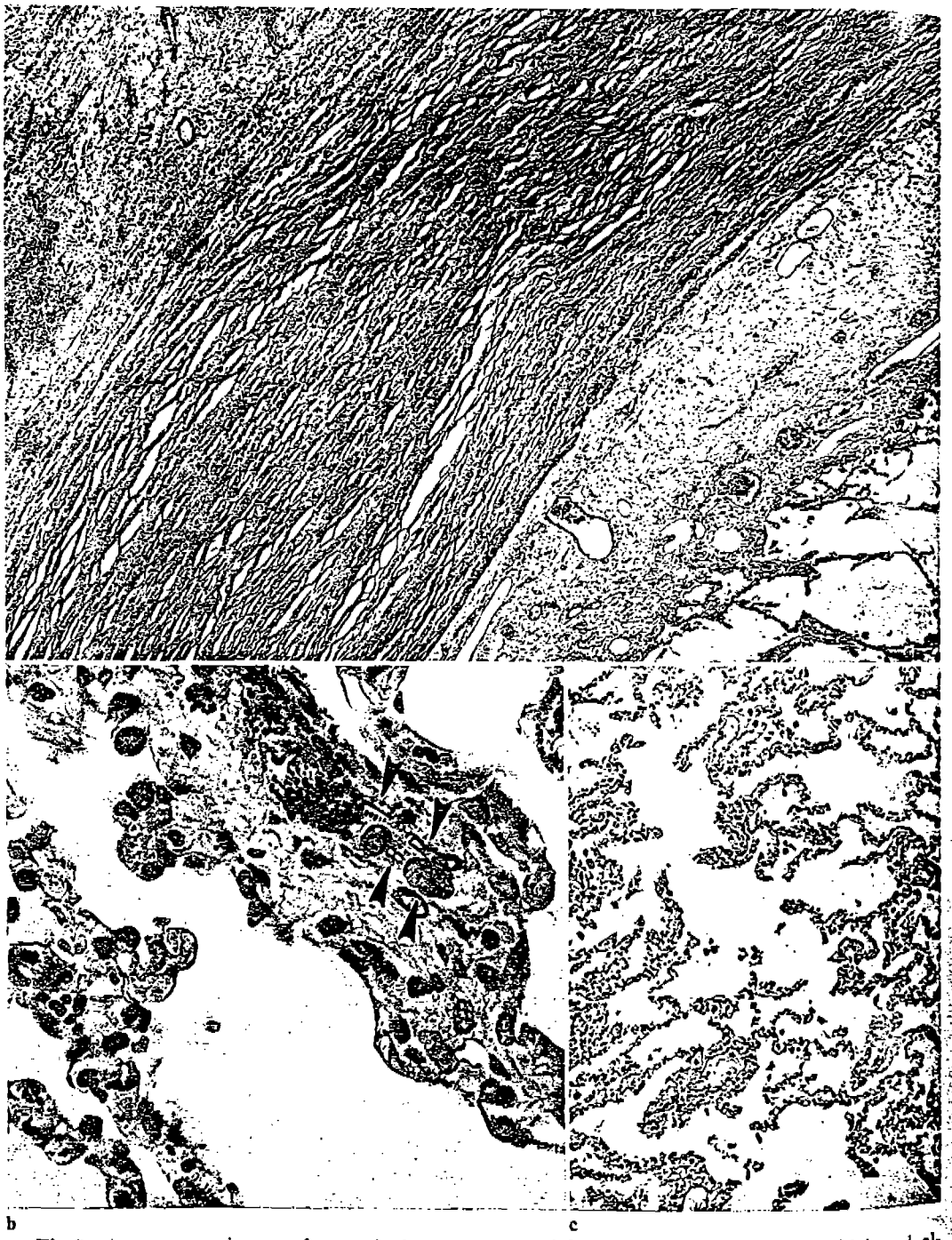

Fig. 3. Autopsy specimens of case 1 show a hyaline plaque with diffuse pleural thickening (a), normal lung tissue (c) and slightly thickened alveolar walls with asbestos bodies (arrows) (b). HE

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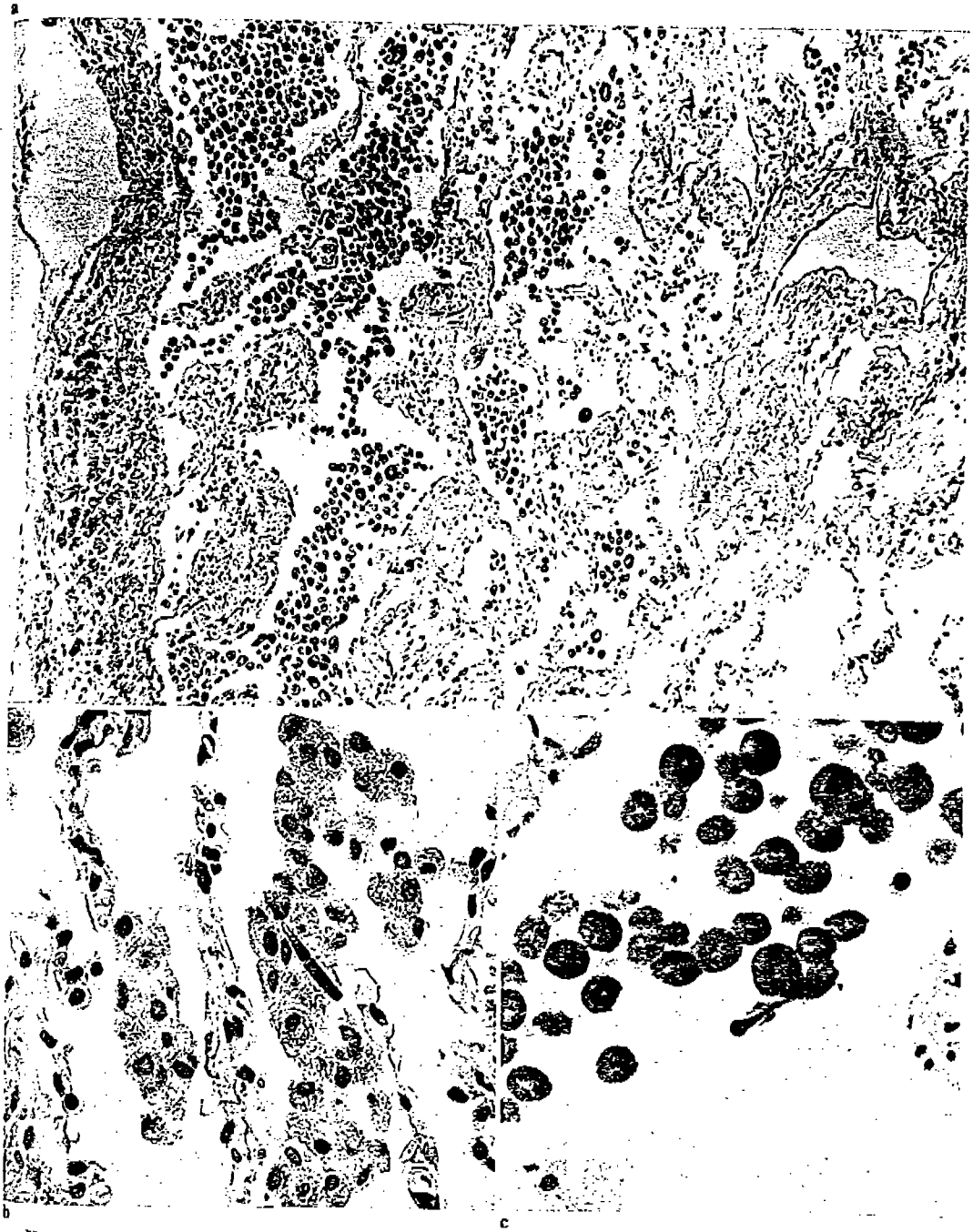

Fig. 4. Lung biopsy of case 2 at low magnification (a) shows minimal pleural thickening, alveolar wall hypercellularity and some fibrosis resembling organizing chronic pneumonia. Many air spaces contain large macrophages. At higher magnification (b, c), isolated asbestos bodies are scattered throughout the lung. HE.

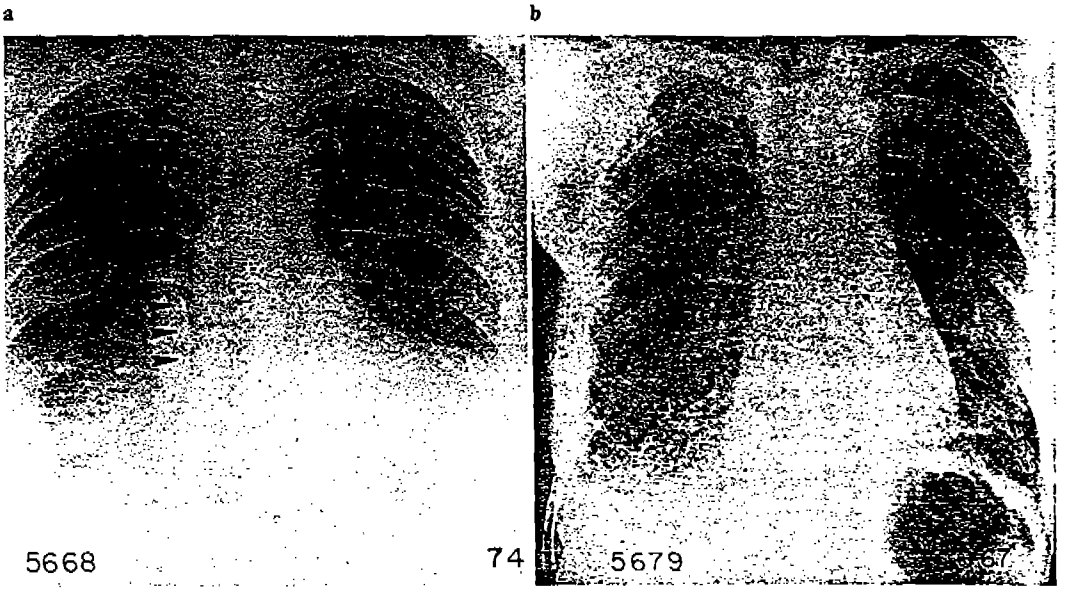

Fig. 5. a Chest roentgenogram of case 2, a 56-year-old woman. There are diffuse linear opacities at both lung bases with blunting of the costophrenic angles as well as plaques and diffuse pleural thickening. A mediastinal calcification is indicated by arrows. b This roentgenogram is of her 58-year-old husband. There are diffuse linear opacities and honeycombing indicative of advanced asbestosis. A blunting of the right costophrenic angle and diffuse pleural thickening were the first indications of a mesothelioma of which he died 6 months later. He had worked in an asbestos fireproofing factory since 1939.

with exercice, her Dss was 14.6 at rest but only increased to 18.9 with exercise.

Review of her environmental history revealed that her husband had worked in an asbestos product factory beginning in 1939. He developed severe asbestosis (fig. 5b) and died of a pleural mesothelioma 28 years later in 1967. The wife's only contact with asbestos occurred while cleaning her husband's work clothes. Periodically these were so laden with white powder that dusting had to be performed outside the house.

Case 3

A 33-year-old shipyard worker was seen by us in 1978 during an industrial respiratory survey. Review of the chest roentgenograms revealed calcifications over both diaphragms and well-defined rounded plaques on both lateral chest walls (fig. 6a). He had come to the shipyard 13 years earlier in 1965 as a pipefitter, but remained only 4 months. He then worked in a machine shop elsewhere without contact with asbestos for 5 years, and then for 6 years in several jewelry shops. In 1976, he returned to the shipyard as a lagger, when he was exposed only to fiberglass. His physical examination and pulmonary function studies were normal.

Case 4

The 27-year-old brother of case 3 was also seen during the survey in 1978. His chest roentgenogram showed a 3-mm calcification on the left diaphragm, a longer hairline calcification on the right diaphragm and a 4-cm calcification on the left chest wall (fig. 6b). Review of his preemployment chest roentgenogram revealed the same lesions. He began working at the shipyard in 1975 as a lagger using only fiberglass. His physical examination and pulmonary function studies were normal.

We discussed the radiographic findings with the two brothers pointing out that, in case 4 the preemployment film at age 24 had shown calcifi-

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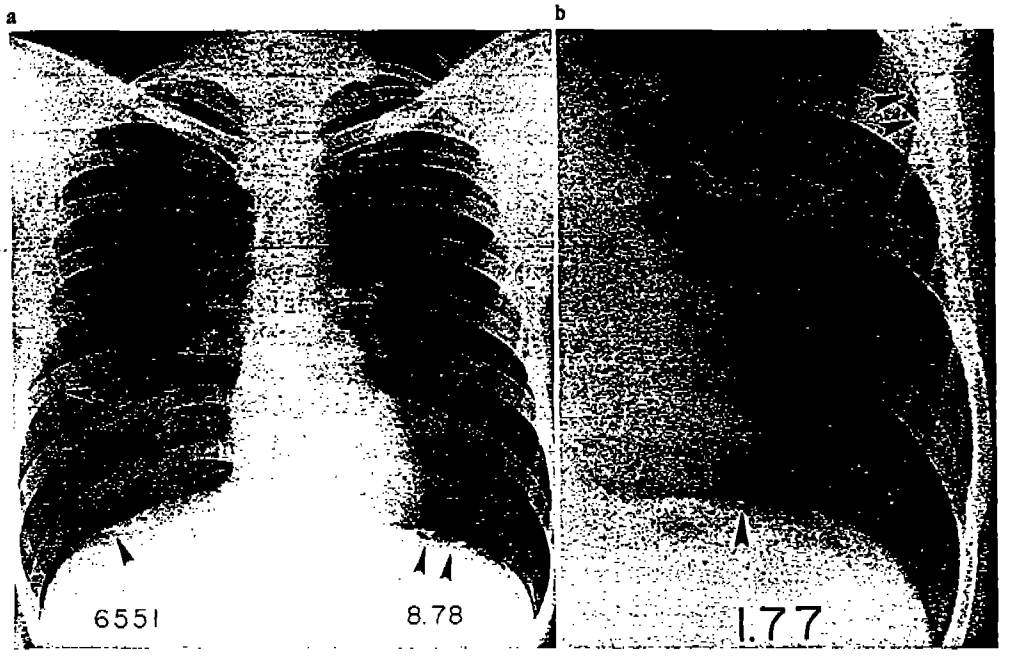

Fig. 6. a Roentgenograms of case 3, a 33-year-old man. Calcifications over both diaphragmatic leaves and rounded plaques on the right are indicated by arrows. b An enlarged section of the chest roentgenogram of case 4, his 27-year-old brother. A tiny diaphragmatic calcification and a thin 4 cm long calcification on the left chest wall are indicated by arrows.

cations, and in case 3, at age 33 there were plaques and calcifications with possibly minimal 4 months' exposure only 13 years ago. We suggested that these changes could not be the result of occupation. The older brother then explained that they had acquired asbestos disease from their father who had been employed at the shipyard as a lagger for 20 years and, for 22 years before that, had been a lagger and asbestos worker at a chemical plant. He had brought home asbestos sheets and 'fluff' from the chemical plant and he established a small business at home for repairing burned-out mufflers with asbestos sheets. For this purpose, the cellar room where the children played was filled with sheets and blocks. Furthermore, he had constructed a tree house from these asbestos materials. The children played there for many years. The father had no respiratory symptoms and screening physiologic studies were normal. However, his chest roentgenogram showed bilateral pleural plaques.

Discussion

The dangers of indirect exposure to hazardous dust within the workplace are well known. The possibility of exposure outside of a plant, so-called neighborhood or household exposure, probably was first recognized in connection with beryllium [2]. More recently the importance of neighborhood exposure to asbestos has been emphasized.

Mesothelioma was related to asbestos exposure by *Wagner et al.* [3] in 1960. This study contained one patient with household exposure, the daughter of an asbestos miner, and 14 neighborhood cases, that is, persons who lived near the mine or who, as

Table I. Neighborhood- and Household-exposed cases of mesothelioma

Authors	Country	Year	Total cases	Exposures				
				none or unknown	occupational	neighborhood	household	asbestos worker in household
Wagner et al. [3]	South Africa	1960	33	1	17	14	1	father
Newhouse and Thompson [4]	England	1965	83	32	31	11	9	husband: 5 sister: 3 daughter: 1
Lieben and Pistawka [5]	USA	1967	42	21	10	8	3	father: 2 sons: 1
Milne [6]	Australia	1969	15	4	10	0	1	father
Champion [7]	Canada	1971	2	0	1	0	1	father
Rubino et al. [8]	Italy	1972	54	43	5	3	3	wife: 1 relatives: 2
McDonald and McDonald [9]	Canada	1973	71	40	26	0	5	father: 3 husband: 2
Lillington [10]	USA	1974	2	0	1	0	1	husband
Greenberg and Lloyd Davies [11]	England	1974	246	65	167	13	1	brother
Anderson et al. [12]	USA	1976	4	0	0	0	4	father: 3 brother-in-law: 1
Whitwell et al. [13]	England	1977	100	11	88	0	1	father
Vianna and Polan [14]	USA	1978	52	30	6	6	10	husband: 9 father: 1
Edge and Choudhury [15]	England	1978	50	1	48	0	1	husband
Li et al. [16]	USA	1978	2	0	0	0	2	father

children, had played on the mine dumps. Since then, several cases of mesothelioma in spouses, siblings and children of asbestos workers have been documented throughout the world (table I) [3–16]. There are some indications that both the occurrence of mesothelioma and the latent period, that is, the duration between first exposure and diagnosis, are dose-related [4, 13]. The interval between first exposure and death was shortest, averaging 29 years, for a group of heavily-exposed factory workers and longest, 49 years, for presumably minimally exposed persons living in factory neighborhoods [4]. The fact that mesothelioma can result from neighborhood exposures suggests that the threshold is low. It is possible that, rather than a real threshold, the very lowest doses simply have so long a latent period that it exceeds man's normal lifespan. Although the latent period almost invariably is very long, the duration of exposure may have been as brief as 3 weeks, but averaged 20 years [4, 6, 11].

The clinical features of mesothelioma are now well described [3–16]. Ther appears to be no correlation with cigarette smoking. Because effusion occurs in over 90% and may be massive, rapid onset of shortness of breath is common. The effusion

is hemorrhagic in only 15%. Roentgenographic markers of asbestos exposure, such as plaques or calcification, are seen in 60%. The diagnosis sometimes can be established by fluid cytology or pleural needle biopsy; more often, exploratory thoracotomy is performed. This sometimes may spread the tumor, causing increased pain and more rapid progression [15]. Grossly, there is permeation of visceral and parietal surfaces by a continuous layer of tumor, but this appearance also may be caused by primary bronchogenic or metastatic tumor. Histologic cell types are classified as epithelial, sarcomatous or, as in our case 1, mixed. Differential diagnosis is not a problem with the mixed cell types, but may be difficult in the other two. For example, a UICC panel, established to verify the diagnoses, reviewed 195 registry cases and considered only 134, or 69% as 'definite' [11]. Prognosis is poor: one-half of patients die within 1 year and few survive more than 3 years. Although radical surgery such as pleuro-pneumonectomy has been encouraged [17], comparison with untreated series suggests that it does not increase survival. Chemotherapy and radiation also have not altered mortality.

Benign asbestos-related effusion, which occurred in our case 2, was first described in 1 case in 1962 [18]. Since we reported a series of such patients in 1971 [19], this manifestations of asbestos exposure has been confirmed in many cases throughout the world [20, 21]. We defined 'asbestos effusion' by four criteria: definite exposure, confirmation by thoracentesis of serial chest films, no evidence of any disease associated with effusion, and no malignant tumor within 3 years. In longitudinal studies of 931 workers, we found a prevalence of 3–5% [22]. Asbestos effusion, like other pleural manifestations, may develop after minimal exposure. However, in contrast to these others, it occurs early, that is, within 10 years of first exposure. Effusions are bilateral or recurrent in 50%, and sanguinous in one-third. Most are transient and require no therapy. With severe residual thickening, there may be physiologic abnormalities and then decortication may relieve the restriction if there is no underlying parenchymal disease.

Pleural plaques and calcifications are the most common manifestations of asbestos exposure. They may be seen after slight or very brief contact, but always with a latent period of at least 15 and more commonly 30–40 years [23–25]. Eventually, they may be seen in one-half or more of all roentgenograms of asbestos-exposed workers, while in unexposed populations they are exceedingly rare, about 0.03% among 10,000 routine radiographs [26]. These lesions also can occur from 'neighborhood' exposure in persons living near asbestos mines or factories [24–27], in agricultural workers tilling soil containing asbestoform minerals [28] and in those unknowingly using asbestos-containing paints [29]. 'Household' or family exposure as a cause of plaques is illustrated by all 4 of our cases and has been reported by others [12, 26, 30, 31]. It appears to be more important than 'neighborhood' exposure because of the higher frequency among family members compared with those living near plants; exposure from workers' clothes may be greater than that from the atmosphere [26]. For example, in a radiographic and clinical survey in Hamburg, among 92 persons with plaques or calcifications, the cause could be traced to occupational exposure in 34, to domestic contacts in 22 and to environmental exposure in only 21; and 5 had no exposure history [26]. In another large study of 326 house-

hold members of asbestos workers, 7 had calcifications and 42 had pleural thickening [12].

Calcifications are readily recognized, most often on the diaphragm, while uncalcified plaques are difficult to see, especially if they are *en face* in the posteroanterior view. Mediastinal calcifications, as in case 2, are rare, while involvement of the pericardium is not uncommon. Plaques are not precancerous lesions nor do they cause functional impairment, since they are situated on the chest wall and diaphragm, do not involve the lungs, and the pleural space remains free. For these reasons plaques and calcifications should not be regarded as a compensable disease [26]. However, they are important epidemiologic markers. No treatment is indicated; however, because of inadequate occupationals and environmental history or because of failure to review older roentgenograms, some patients are unnecessarily operated upon.

Asbestosis is defined as a pneumoconiosis, that is, a chronic interstitial pneumonia and fibrosis due to dust inhalation. Like all asbestos-related disease it is dose-related, but the dose required for recognizable manifestations certainly is larger than that required to produce pleural-based disorders. Therefore, asbestosis resulting from 'out-of-plant' neighborhood or household exposure is unlikely. For example, in patients with mesothelioma related to neighborhood or household exposure, lung tissue, when available, did not show asbestosis (table I), though often it contained asbestos bodies (fig. 3). However, in a radiographic study of household contacts, 2 or 0.6% were said to have definite signs of asbestosis [12]. Clinical, radiographic or physiologic manifestations of asbestosis in our experience are never seen less than 10 years after onset of exposure. Dyspnea is the most common symptom, and fine, end-inspiratory crackles (rales) are heard in over 65% of patients [32]. Physiologic abnormalities include decreased vital capacity and abnormal gas exchange documented by low diffusing capacity and elevated $P(A-a)O_2$ during exercise. Significant obstruction is no more common than in control populations matched for age, sex and smoking habits [33]. The usual radiographic abnormalities consist of linear or irregular opacities that are most marked at the lung bases and often obscure the cardiac and diaphragmatic outline. Histologically, asbestosis is characterized by a usual interstitial pneumonia and fibrosis that may proceed to honeycombing. After a latency period of 10–15 years, both the severity of the lesions and their rate of progression are dose-related. Usually, there is progression even if the workers are removed from further exposure. Death often has been attributed to cardiorespiratory failure. However, in recent years it has become apparent that 20–50% of those asbestos workers who smoke die of bronchogenic carcinoma [13, 34].

A diagnosis of an occupationally or environmentally related disease generally is based on adequate history taking. This task is especially difficult in relation to asbestos for several reasons. First, there are over 1,000 asbestos-containing products, that, until recently were not labeled as to content so that workers in secondary industries often were not aware of exposure. Second, because of the long latency period, exposure often occurred in the distant past and has been forgotten. Third, asbestos-related manifestations may result from brief or minimal exposure. Finally, job descriptions such as 'plasterer', 'fireman' or 'engineer' are often misleading, because official titles

tend to remain the same while hazardous exposure may change dramatically with the development of new technologies. From the foregoing it is apparent that eliciting exposure histories from persons peripherally exposed, for example, from those living in neighborhoods or from family members, is even more difficult.

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