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Case Reports

Floor Tile Installation as a Source of Asbestos Exposure[1-3]

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SUMMARY

Asphalt or vinyl-asbestos floor tile contains fifteen to twenty-five per cent asbestos, but the mineral fibers are firmly embedded in the binding material. Installation of these tiles would, therefore, seem to be an unlikely source of a hazardous dust exposure. Case reports of two installers of floor tile, one with biopsy-proved mesothelioma and another with extensive pleural calcifications are presented. Both workers had frequently sanded asphalt and vinyl tile floors prior to installation of new floor covering. An investigation of the work process revealed that under simulated conditions of work, asbestos dust concentrations as large as 1.3 fibers per ml were found in air samples passed through membrane filters worn by a person engaged in sanding vinyl asbestos. Characteristic asbestos fibers were seen in electron photomicrographs of these samples. These findings suggest that before the tile sanding procedure is performed adequate respiratory protection should be provided or alternate, available installation methods should be used.

Introduction

Asphalt or vinyl-asbestos floor tile contains 15 to 25 per cent asbestos; however, the mineral fibers are firmly embedded in the binding material (1). Installation of these tiles would, therefore, seem to be an unlikely source of hazardous dust exposure. Recently the writers studied two installers of floor tile, one with biopsy-proved mesothelioma and the other with extensive pleural calcifications who had no other known exposure to asbestos. These workers had frequently sanded asphalt and vinyl tile floors before installation of new floor covering. In this report, these cases and the results of an investigation of exposure to asbestos during tile sanding are presented.

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Case Reports

Case 1: A 44-year-old man was admitted because of chest pain of one week's duration. He had worked from 1948 to 1967 as a floor tile installer and routinely sanded old tile before resurfacing it. There was no history of other occupational dust exposure. He had worked in a nondusty area of a shipyard from 1945 to 1947 repairing gyroscopes. From 17 to 30 years of age, he had smoked one package of cigarettes per day.

Four months before his present admission an illness developed with left anterior chest pain and hemoptysis. On admission to another hospital, a left pleural effusion was noted. He was treated with penicillin and was discharged improved. One week before his current admission, hemoptysis and severe, pleuritic, left chest pain again developed.

There were dullness and decreased breath sounds over the left lower chest. No friction rub was heard. A chest roentgenogram revealed a left pleural effusion without calcifications.

A thoracentesis yielded 2,000 ml of serosanguineous fluid that contained 3,400 leukocytes per mm³, predominately mononuclear cells, 154,000 erythrocytes per mm³, and no polymorphonuclear leukocytes. The glucose concentration was 25 g per 100 ml; the concurrent blood glucose was 80 mg per 100 ml. Total protein concentration of the fluid was 4.8 g per 100 ml. No asbestos bodies were seen, and cytologic examination of the cells in the fluid suggested malignancy. Histologic examination of a needle biopsy of the pleura showed malignant papillary mesothelioma.

Case 2: A 61-year-old floor tile installer was referred to the pulmonary clinic because of an abnormal chest roentgenogram taken during a routine periodic medical evaluation.

The patient had worked for the past 30 years installing asphalt and vinyl tile. There was no history of other occupational dust exposure, chest trauma, pneumonia, or hemoptysis. He complained of mild dyspnea on climbing two flights of stairs, but denied orthopnea, paroxysmal nocturnal dyspnea, and ankle edema. He had smoked one package of cigarettes per day for the past 15 years.

Physical examination revealed a well developed, plethoric white man. The anteroposterior diameter of the chest was increased, and fine, scattered, crepitant inspiratory rales were noted at the bases bilaterally. Marked clubbing of the fingers was present.



Fig. 1. Posteroanterior chest roentgenogram revealing extensive pleural calcification, pleural thickening, and pulmonary fibrosis.

dense plaque of calcification in the left pleural space and sheetlike calcifications in the pleura on the right. Dense calcifications were seen on both diaphragmatic pleural surfaces. The diaphragms were flattened.

The vital capacity was 64 per cent of predicted with a one-second forced expiratory volume of 65 per cent of the total. Arterial oxygen tension at rest was 71 mm Hg. The single breath pulmonary diffusing capacity was 60 per cent of predicted.

Dust Exposure

To simulate normal work practice, samples of vinyl tile were laid on a plywood sheet using tile cement. This sheet was placed inside a room approximately 10 feet wide, 12 feet long, and 7 feet high that was exhausted at a rate of four air changes per hour. An operator wearing a respirator sanded the tile for approximately 20 minutes using a conventional belt sander with a coarse grit.

During the work period, air sampling was carried out to determine the exposure to asbestos fibers. Two personal samplers worn by the operator collected air samples on membrane filters at an average sampling rate of 3.6 liter per min. Fibers longer than 5 μ with an aspect ratio greater than 3 were counted under phase contrast microscopy by the technique of Edwards and Lynch (2). Dust concentrations for these two parallel samples were 1.2 fibers and 1.3 fibers per ml of air, respectively. These concentrations are



Fig. 2. Electron photomicrograph of a replicated membrane filter showing asbestos fibers.

of 5 fibers per ml (3) but do represent significant exposures. Under other work conditions, the threshold limit value could be exceeded.

The fields of these membrane filters examined under phase microscopy were consistent with those seen in samples collected in other asbestos operations such as marine insulation installation and tearout. Discrete fibers were evident and were easily counted. Characteristic asbestos fibers are shown in the electron photomicrograph of a replicated membrane filter sample (figure 2).

During the sanding operation, a point-to-plane electrostatic precipitator was used to collect a series of samples on grids for electron microscopy. These fibers met the criteria proposed for asbestos by Gross and associates (4) (figure 3). The ends of the fibers had profiles characterized by steplike interruptions, and fibril bundles were easily seen. Fibers were clumped together within the matrix of the tile material.

Discussion

Both of the tile installers had diseases known to be associated with exposure to asbestos.

The first patient had typical clinical, roentgenographic and pathologic findings of pleural mesothelioma. Epidemiologic investigations of cases of mesothelioma have shown asbestos exposure in more than 40 per cent (5, 6). The incidence of mesothelioma in the general population is from 1 in 1,000 to 1 in 10,000 deaths (7). Because the present studies revealed that air-borne asbestos particles are generated during sanding of



Fig. 3. Air-borne asbestos fibers collected by electrostatic precipitator.

tile, asbestos must be strongly considered as a cause of the patient's illness. Because his employment in gyroscope repair did not involve work in engineering spaces, it is unlikely that he was exposed to asbestos dust. Nonoccupational exposure to asbestos or mesothelioma unrelated to asbestos cannot be excluded.

Significant asbestos exposure was also implicated in the second patient who had extensive bilateral pleural calcifications. Selikoff found 150 cases of pleural calcification in 1,117 installation workers and concluded that in the absence of a history of chest infection or trauma, bilateral pleural calcification can usually be considered to be due to asbestos exposure (8). The causal relationship seems clear because the patient had no history of pulmonary infection or trauma and had a known exposure to asbestos. It was more difficult, however, to assess the extent of underlying pulmonary asbestosis in this worker. The commonly described clinical manifestations of this disease include dyspnea, basilar rales, clubbing of the fingers, decreased vital capacity, abnormal diffusing capacity, decreased compliance, hypoxemia, and characteristic radiographic changes in the lungs (9). The patient exhibited most of these findings and had no evidence of heart disease, other systemic disorder, or occupational exposure. It is likely, therefore, that he had at least minimal pulmonary asbestosis as well as pleural disease.

Asbestos tile has been installed on countless floors during the last 30 years. Because this industry is the second largest consumer of this mineral in the United States, the potential hazard is significant if the current method of sanding these floors continues (1). The clinical findings in these subjects and the results of air sampling in a simulated work environment suggest that before the tile sanding procedure is performed ade-

quate respiratory protection or an alternate installation method should be used. The association between installation of tile and asbestos-related disease suggests that epidemiologic, environmental, and clinical investigation of tile installers be conducted. In addition, practicing physicians should be alerted to the possibility of asbestos-related illness in this occupation.

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RESUMEN

Instalación de baldosas para el piso como una fuente de exposición a asbestos

El asfalto o la baldosa para el piso de vinilo-asbestos contiene de 15 a 25 por ciento de asbestos, pero las fibras numerales están firmamente incrustadas en el material de ligamiento. La instalación de estas baldosas, por consiguiente, no parecería ser una fuente peligrosa de exposición al polvo. Se presentan los casos de 2 instaladores de baldosas para el piso, uno con un mesotelioma comprobado por biopsia y el otro con calcificaciones pleurales extensas. Estos trabajadores habían lijado frecuentemente baldosas el piso de asfalto y de vinilo antes de instalar los nuevos pisos. La ocurrencia de estos casos llevó a una investigación del proceso de trabajo. Bajo condiciones de trabajo simuladas, se encontraron concentraciones de polvo de asbestos tan altas como 1.3 fibras por ml en muestras de aire en filtros de membrana que llevaba puesta una persona envuelta en lijar los asbestos de vinilo. Fibras de asbestos características de estas muestras fueron vistas en fotomicrógrafos electrónicos. Estos hallazgos sugieren que antes del proceso de lijar se lleve a cabo, se debería usar una protección respiratoria adecuada o usar otros metodos de instalación al alcance.

RESUME

L'installation des carrelages: source d'exposition à l'amiante

Les carreaux d'asphalte ou d'une combinaison de vinyl et d'asbeste contiennent de 15 à 25 pourcent d'amiante, mais les fibres sont intimement liées à l'agent agglutinant. L'installation de ces carreaux parait donc une source improbable de troubles pneumoconiotiques. Les rapports médicaux de deux ouvriers installeurs de carrelage, l'un souffrant de mésothelioma confirmé à la biopsie, et l'autre étant porteur de calcification pleurales sont presenté ici. Ces deux ouvriers avaient souvent sablé des carrelages d'asphalte ou de vinyl-asbeste au papier de verre, avant d'installer des nouveaux carreaux. L'incidence de ces cas a incité à une investigation du procédé. En recréant les conditions de travail, des concentrations de poussière d'amiante d'un niveau aussi elevé que 1.3 fibres par ml ont pu etre mesurée dans l'air ambiant. Les mesures furent determinée en faisant passer des échantillons d'air sur un papier filtré fixé sur un travailleur en train de sabler des tuiles de vinyl-asbeste. Des photomicrographies de ces prelevement ont montré qu'il s'agissait de fibres amiante caractéristiques. En consequence, nous devons appliquer une protection respiratoire suffisante lors du sablage de tuiles ou choisir un autre procédé d'installation.

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