

National Cancer Program special communication

July 9, 1979

Reports have appeared in the public press suggesting that dust from fibrous glass may be responsible for causing a form of lung disease in workers similar to that produced by asbestos.

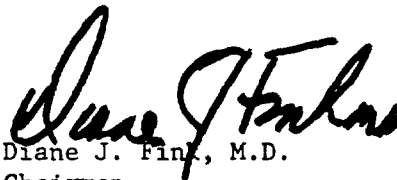
These reports are attributed to information about a case of pneumoconiosis presented at a meeting held under the auspices of the National Cancer Institute on November 15, 1978. They have occasioned widespread concern among those who make and use fibrous glass.

The purpose of this communication is to provide additional information which now shows that the case in question was almost certainly due to asbestos and not to exposure to fibrous glass.

Sincerely,



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PNEUMOCONIOSIS AND FIBROUS GLASS

Introduction

A paper entitled "A Case Study on Fiberglass Pneumoconiosis and Some Comments on the Principle of Pneumoconiosis Prevention" by Tatsuo Sano, Keizo Chiyotnni and Hisao Shida, was presented by Dr. Sano (Vice Director, Institute for Science of Labour, Environmental and Occupational Disease, Kawasaki, Japan) at a meeting of the Asbestos Education Task Force of the National Cancer Institute on November 15, 1978 in Bethesda, Maryland.

This report included preliminary findings in a case of pneumoconiosis that appeared to be associated with occupational exposure to fibrous glass dust. Although the authors noted that the results were not conclusive, news of the report published in the lay press occasioned widespread interest and concern because of the implication that exposure to fibrous glass was capable of producing lung disease similar to that resulting from exposure to asbestos.

Since that time Japanese investigators have completed previously planned studies on the case in question and have also examined other workers in the same factory. The findings and conclusions of these investigations are summarized below; they leave little doubt that prior exposure to asbestos dust was responsible for the effects observed.

Initial Findings

As reported by Dr. Sano on November 15, 1978, a woman, aged 54, who had worked from 1970 to 1973 in a factory making prefabricated houses, where she was cutting glass wool insulation, was found in 1975 to be suffering from irritation of the larynx and a dry cough, and to have developed calcified plaques on the diaphragm and parietal pleura.

Examination of the lungs by selective alveolobronchography showed changes in the small airways to those produced by inhalation of long-fibered asbestos dust. Ferruginous bodies were found in tissue obtained by transbronchial biopsy.

Tentative Conclusions

Discussing these results, Dr. Sano considered it possible that the type of pneumoconiosis seen in this case could have been caused by fibrous glass, but observed that such a conclusion could not be reached without additional evidence from electron microscopy. In particular, it was important to determine whether the ferruginous bodies were caused by fiberglass or asbestos. He did not believe that asbestos was involved because it was understood that the patient had neither lived nor worked where there was the likelihood of inhaling asbestos dust. Dr. Sano also noted that the lack of a thorough epidemiological study was an important deficiency.

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Analysis of Biopsy Specimen

The planned examination by electron microscopy of the tissue obtained by transbronchial biopsy has now been completed.

The results show that many small chrysotile asbestos fibers were present, but no glass fibers, nor any fibers of other materials. The chrysotile fibers were found in higher concentration than is normal for such tissue; they were between 0.05 and 0.07 um diameter, and 0.3-0.5 um long, some 1-2 um long. The fine structure of some of the chrysotile fibers was seen, by selected area electron diffraction, to be amorphous, which implies that some magnesium or silicon ions had been leached out of the fibers as a result of long residence in the tissue. Some non-fibrous mineral dust particles were identified as well as some particles of glass dust.

Chemical composition of the various particles detected was determined from x-ray energy spectra taken in the microscope.

Occupational History

At the time of the initial report, it was believed that the patient in this case had neither worked nor lived where exposure to asbestos dust was likely. A more thorough occupational history obtained subsequently indicates that she had held many different jobs, several of them in dusty conditions, and that there had been at least one opportunity for occupational exposure to asbestos as early as 35 years before symptoms of pneumoconiosis developed.

Other Workers

During discussion of the original report to the Asbestos Education Task Force, it was understood that six other cases of pulmonary disease had been found among workers at the same factory. The existence of these cases cannot be confirmed and is attributed to a linguistic error.

It is reported that all 20 workers in the factory have recently been examined at the Rosai Hospital for Silicosis. Only three of them had been exposed to fibrous glass. No disease was found that could be attributed to this exposure.

Comments

The information summarized above leads to the conclusion that the case of pneumoconiosis reported by Dr. Sano on November 15, 1978, was a result of exposure to asbestos dust, probably many years previously. This conclusion is entirely consistent with the large body of accumulated information on the natural history of asbestosis.

A Note on Sources and Acknowledgements

The report given by Dr. Sano to the Asbestos Education Task Force on November 15, 1978, had previously been presented at the 5th Conference on Pneumoconiosis,

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October 29 - November 3, 1978, Caracas, Venezuela.

After hearing the report in November 1978, the Division of Cancer Control and Rehabilitation of the National Cancer Institute wrote to Dr. Sano requesting information on any new findings in connection with the workers who had been exposed to a fibrous glass.

At the end of February 1979, a personal communication from Dr. I.J. Solikoff, Mt. Sinai School of Medicine, indicated that electron microscopical analysis of the biopsy material by N. Kohyama, H. Kyono, and K. Kawai, National Institute of Industrial Health, Kawasaki, Japan, showed the presence of chrysotile fibers but no glass fibers. Confirmation, and a copy of Dr. Kohyama's report were received in March with a report by Dr. Jon L. Konzen, Owens Corning Fiberglas, who, with Dr. Paul Kotin, Johns Manville Corporation, met with Japanese occupational health authorities in February 1979. Further details of the occupational history and the health of other workers were included in the latter report. So far as is now known, none of the reports mentioned above have been published in the scientific literature.

THE NATIONAL CANCER PROGRAM is a joint effort by the Federal Government, nonprofit organizations, private and public institutions to coordinate the discovery and implementation of knowledge that will advance the attack on cancer. The commitment of the National Cancer Program to the American people is that the most promising research leads will be pursued with diligence and that every research advance that can benefit patients will be applied without delay.

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To Asbestos
File

- 1008/79 Asbestos and Smoking. I. J. Selikoff and E. C. Hammond.
J. Am. Med. Assn. 242: 458-459, August 3, 1979. 4 refs.

New data are cited on the relationship between asbestos exposure and lung cancer. A follow-up of 12,051 asbestos insulation workers from January 1, 1967 through December 31, 1976 showed death rates for lung cancer per 100,000 man-years, standardized for age as follows: 11.3 for men who neither worked with asbestos nor smoked cigarettes, 58.4 for men who worked with asbestos but did not smoke, 122.6 for cigarette smokers who had not worked with asbestos, and 601.6 for those unfortunate enough to have had both exposures--cigarettes and asbestos. By itself does asbestos increase the risk of lung cancer? The answer is yes. But even four or five times the risk, when the base risk is low (as it is for nonsmokers in general), does not result in many cases. (Clearly, any excess is undesirable.) On the other hand, smoking itself causes a major increase, and when that high risk is then multiplied manifold, an immense increase is found; among asbestos workers, unhappily, one in every five deaths is due to lung cancer. The newest data carry the asbestos-smoking interaction a step further--to increased risk of death of asbestosis. Again, asbestos exposure by itself carries the risk of fatal progressive pulmonary fibrosis. In this series, five men who never smoked cigarettes died of asbestosis. But the asbestosis mortality for men who smoked a pack or more a day was 2.8 times as high as the asbestosis mortality for men who never smoked regularly. Smoking, with its own bronchitis, emphysema, and fibrosis, adds an undesirable and sometimes unsupportable burden to the asbestos-induced pneumoconiosis.

--Condensed from text

- 1009/79 Automatic Detection and Counting of Asbestos Fibers.
Vickers Instruments, Inc. Am. Ind. Hyg. Assn. J. 40: A-50--A-53, July 1979.

Two new techniques developed in Britain will enable hazards due to airborne asbestos fibers to be assessed more quickly, more reproducibly and more accurately than by current manual counting methods. One is suitable for the immediate analysis of samples collected from working environments; the other is intended to aid compliance with health and safety legislation as well as research into improved safety standards. Both techniques can be extended to evaluations of other fibrous materials.

--Condensed from text

- 1010/79 Mesothelioma and Exposure to Mixtures of Chrysotile and Amphibole Asbestos.
E. D. Acheson and M. J. Gardner. Arch. Environ. Health 34: 240-242, July/August 1979. 16 refs.

This paper provides a new analysis of previously published work and draws attention to the possibility that mixtures of amphiboles and chrysotile appear more commonly in the lungs of mesothelioma patients compared to controls than do either of the main types of fiber alone. The possibility that these results may indicate a synergistic interaction between chrysotile and the amphiboles is discussed in the light of the epidemiological data.

--Authors' abstract

- 1011/79 Numbers of Asbestos Bodies in Urban Patients with Lung Cancer and Gastrointestinal Cancer and in Matched Controls. A. M. Churg and M. L. Warnock.
Chest 76: 143-149, August 1979. 25 refs.

The authors compared the numbers of asbestos bodies extracted from the lungs of 103 patients with lung cancer and 50 patients with gastrointestinal malignant neoplasms to the numbers of bodies extracted from lungs of control patients matched for age, sex, smoking habits, and, in some cases, occupation. All patients were urban dwellers over the age of 40 years, and none was a primary asbestos worker. No differences in the counts of asbestos bodies were observed between the tested and control populations. The numbers of asbestos bodies did correlate well with occupation; the highest counts were found in male manual laborers. The authors conclude that in the urban population studied herein, the numbers of asbestos bodies alone do not correlate with the presence of pulmonary or gastrointestinal carcinoma; however, uncoated asbestos fibers are also known to be present in the lung, and the possibility that such tumors may be related to the numbers of these fibers in lungs remains to be explored.

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