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PLEURAL PLAQUES AND ASBESTOS: FURTHER OBSERVATIONS ON ENDEMIC AND OTHER NONOCCUPATIONAL ASBESTOSIS

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Association between exposure to asbestos and neoplasia, increasing world production of asbestos and the possibility of environmental and other nonoccupational exposure to asbestos, have caused an increased interest of medicine in this mineral. In this connection, asbestos may gain considerable fame as a new discovery. However, it had that thousands of years ago. About 4500 years ago, asbestos was generally used in Finland as a cementing agent in the preparation of clay pottery. Asbestos ceramics, according to archeologic finds a Finnish invention, was used during a period of 3000 years. Approximately 500 A.D. the use of asbestos ceramics in Finland and its neighboring countries slowly ceased to be reused first about 1,000 years later.[1] Thousands of years ago, asbestos was in everyday use by families and consequently the Stone Age men "geologically" well knew where asbestos could be obtained.

Anthophyllite-asbestos is a fairly common mineral in Finland and tremolite-asbestos is especially characteristic of the Karelian formations in Northern Karelia. Anthophyllite has been mined since 1918, the annual output being around 15,000 metric tons.

Near two open asbestos mines several hundred cases of pleural calcification were found in people not employed in the mining industry.[2] Corresponding observations have been reported in East Germany.[3,4] Pleural plaques, as described in these reports, were similar to those characteristically seen in asbestos workers. Regardless of the type or degree of pulmonary reaction or fibrosis, these cases may be called cases of endemic asbestosis, until a better specification is found (pleural asbestosis?).

Pulmonary Fibrosis in Endemic Asbestosis

The majority of endemic cases of pleural calcification showed no subjective symptoms of a respiratory distress. Slight pulmonary fibrosis was roentgenologically detectable in about 14 per cent of 150 cases. The evaluation *in vivo* of the degree of fibrotic changes and their possible connection to asbestos dust is very complex and difficult. As a result of pathologic lung changes, the heart also may be affected. To study this, the following comparison was drawn: A roentgenologic volume measurement of the heart was made in 130 pleural plaque cases by using three right-angled heart diameters, two horizontal and one sagittal. Control material was gathered by taking the cases nearest to the plaque cases in the roentgen file. Thus,

the age distribution in both groups automatically was similar, since the files are kept according to the birth number (year, month, day) and the control material was purely incidental. Heart volumina of the plaque group were 18 per cent greater than those of the controls.

In a group of 38 plaque cases and 23 control cases, pulmonary function tests were performed. Here only the vital and diffusing capacities are taken into consideration. The diffusing capacity measured by CO-method using rebreathing technique, was pathologically decreased in nine cases of the material and two cases of the control. Statistically the difference is insignificant. The mean vital capacity was 3600 in the material and 4000 in the control series. The reduction in vital capacity was statistically significant at 95 per cent level (analysis of variancy, $F = 4.76$).

Because reduction in vital capacity may occur in many diseases and regarding to the statistical insignificancy of the difference in diffusing capacity, positive conclusions are impossible. In endemic asbestosis, the possibility of pulmonary fibrosis must nevertheless still be taken into account.

Pulmonary and Pleural Neoplasia and Asbestos in Northern Karelia

Pulmonary and pleural neoplasia occur in a high percentage among asbestos workers, as is already well known. A close relationship has even been stated between environmental exposure to asbestos and pleural mesotheliomas.

Two hundred and sixteen cases of histologically proven lung carcinoma, found during 1958-61 in the Central Hospital of Northern Karelia, were used as a material for studying the asbestos-neoplasia problem. The areal distribution of all 216 cases did not show a higher incidence round the two asbestos mines, but corresponded to the areal distribution of pulmonary tuberculosis and cardiac insufficiency. Coincidence of endemic asbestosis and pulmonary carcinoma was observed in 21 cases. A comparison between the latter mentioned and carcinomata without asbestosis, as to the site of the tumor (upper or lower lobe), was drawn. In 67 advanced cases locating the tumor was not possible because of diagnostical difficulties. The tumor site in coincidental cases was on right 7 upper 6 lower lobe and on left 2 upper/3 lower lobe, and in other cases 48 upper/21 lower lobe on right and 43 upper/19 lower lobe on left. Four cases occurred with a tumor penetrating the thoracic wall. These showed signs of endemic asbestosis.

During 1959-62, the Central Hospital paid special attention to the possible occurrence of pleural mesothelioma. However, only one case was recorded. Preliminary clinical diagnosis was cholecystitis and chest roentgenogram showed pleurisy on right. After antibiotic therapy without success and increase of right chest pain, pleural tumor was suspected.

Thoracotomy revealed tumor infiltration in the basal pleura and histology proved a pleural mesothelioma. Lifelong environmental asbestos exposure occurred in this case.

Compared to the high incidence of benign pleural lesions or plaques among population environmentally exposed to asbestos, pulmonary and pleural neoplasia are rare. Especially the vague areal distribution of pulmonary carcinoma in Northern Karelia counteracts a positive conclusion concerning causal relationship between endemic and pulmonary carcinoma. It should also be noted, that in the series of 216 cases the distribution by sex was 216 male, 0 female. No explanation for this was detectable.

Cases of Pleural Plaques Found in Central Finland

Among 35,000 routine chest roentgen examinations during 18 months in 1963-64, 77 cases of pleural plaques were found. Roentgenologically these were similar to those seen in endemic asbestosis. Of the 77 cases, 68 were questioned concerning a possible occupational or other exposure to asbestos and mixed dusts. The results are presented in TABLE 1, also showing the occurrence of roentgenologically detectable pulmonary fibrosis in these cases. In cases without any detectable exposure to dusts, pulmonary changes were slight or absent, and the patients showed no subjective symptoms of respiratory distress, whereas e.g. the bricklayers had various degrees of dyspnea and showed roentgenologically definite pulmonary fibrosis associated with local emphysematous areas. In most cases with pulmonary fibrosis, visceral pleural adhesions, directed from the plaque to the lung, were discernible especially using fluoroscopically aimed various oblique projections. This phenomenon was not found in plaque cases without pulmonary fibrosis.

The discovery of an asbestos dust exposure in case history is often very difficult. A 50-year old woman had been treated over a period of several years for an unspecific dyspnea. The clinical diagnoses were asthma or cardiac insufficiency. The patient showed typical bilateral pleural calcification. However, she denied any possibility of asbestos exposure or even having ever visited the mining area. Two years later the patient came upon the following information. As a five-year old child she had spent three months in the immediate vicinity of the asbestos mill. As many others, she had used bunches of asbestos fibers as a playing material.

With the basis of previous observations on endemic asbestosis in Eastern Finland and further examinations concerning the severity and duration of exposure, it can be noted, that the plaques develop very slowly. It seems apparent that even very slight exposure over a short period may cause pleural changes many years or even decades later. The problem presented by the time factor, very different to that seen in occupational constant expo-

TABLE 1
PARTICULARS CONCERNING 77 CASES OF PLEURAL PLAQUES FOUND IN 35,000 ROUTINE CHEST ROENTGEN EXAMINATIONS AT TAMPERE CENTRAL HOSPITAL DURING 18 MONTHS IN 1963-64

Occupation	Number of cases	Roentgenological evidence of pulmonary fibrosis	Normal or very slight fibrosis	Exposure
Bricklayers	8	6	2	Intermittent exposure to mixed dusts containing asbestos.
Building workmen	7	2	5	
Factory workers, etc.	20	6	14	Very slight exposure to dusts without known asbestos
At random	25	4	21	Not detectable
—	2	0	2	Born in Kuusjärvi, Eastern Finland. Environmental exposure to asbestos.
—	1	0	1	Husband exposed to asbestos 20-28 years previously.
—	4	1	3	Father exposed to dusts containing asbestos.
	77			

sure, highly complicates the chances to prove whether or not there has been a dust exposure and/or any relationship to asbestos.

The four (last) cases in TABLE 1 deserve special attention. The first was found in routine chest-photofluorograph. The case history revealed that the patient's father had 50 years ago been occupationally exposed to asbestos dusts. This was the only possible connection to asbestos. For further information the husband of the patient and her three sisters were examined. The husband showed no pathological changes in the lungs or pleura, but all three sisters had bilateral pleural changes, two had very typical pleural plaques. One of them also had visceral pleural adhesions and pulmonary fibrosis. All but the last mentioned were subjectively symptomless. Histology of biopsy material taken by thoracotomy, revealed pulmonary

pleural fibrosis and in four micron thick paraffin sections no asbestos fibers were visible.

Comments

In advanced cases of pleural plaques, roentgen diagnosis is easy. Misinterpretations as lung or pleural tumors are possible, especially if relationship to asbestos or dusts is not known. In the literature, benign local parietal pleural changes "of obscure origin" consisting of hyaline sclerotic fibrin, at first misinterpreted as lung or pleural tumors, have also been reported.[7] To judge from the description of those pleural changes, they seem very similar to those seen in asbestosis.

The correct roentgenological observation and interpretation of parietal pleural plaques enables discovery of pneumoconiosis even in cases which show no other clinical evidence of an inhalation damage. Even though not pathognomonic to asbestosis, pleural plaques are of significance for industrial hygiene and may be very helpful in epidemiologic studies.

The detection of exposure to asbestos or mixed dusts is often very difficult in case histories, mainly because the interval between exposure and manifestation may range from several years to decades. Also, it should be noted that asbestos is found with great difficulty in paraffin sections of ordinary thickness.[8] In cases of pleural plaques asbestos may be found more often if they are specially searched for and if special methods are used.

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