

Prevalence of Pleural Calcification in Persons Exposed to Asbestos Dust, and in the General Population in the Same District

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It is of interest whether pleural calcification is primarily the result of long years of exposure to asbestos dust or whether there are factors other than exposure to dust. We have investigated persons working for a long period in a plant processing asbestos products (chrysotile), persons without occupational exposure to dust but living in the vicinity of the plant, and consanguineous relations of patients with pleural calcifications. We have also studied a large population above the age of 40, in the district in which the plant is situated. Comparison of the groups disclosed that prevalence of pleural calcifications was closely related to opportunity for exposure to asbestos dust either occupationally or by family or neighborhood contact, as contrasted with the unexposed population. The prevalence in the group with direct or indirect asbestos exposure was 5.3, 5.8, 3.5%; whereas in the unexposed population it was 0.34%. These results indicate that asbestos is primarily responsible for pleural findings, but that some pleural disease may be the result of the other factors, still not known. The identification of other causes is hampered by the long period which need elapse from the onset of the process to the radiological appearance of the pleural change.

Soon after the demonstration of the fibrogenic properties of asbestos clinical evidence appeared demonstrating pleural changes in persons with long work exposure in asbestos mines or factories. The unusual finding of pleural calcification was attributed, in the fifties, solely to the effect of asbestos [Cartier (4), Lynch and Cannon (12)]. Later, when unexplained findings of the same character were made in the populations not known to be exposed to asbestos, the etiology seemed less certain [Jacob and Bohlig (9); Anspach (1); Anspach, Roitzsch, and Clauswitzer (2); Bohlig (3)].

Epidemiological studies of populations designed to investigate possible exposures to asbestos dust led in the sixties and later to sharp differentiation of both points of view. In the first instance, are Finnish, Canadian, American and other investigators who believe that occupational or nonoccupational exposure to asbestos provide the background for pleural findings [Kiviluoto (10, 11); Raunio (16); Cartier (5); Selikoff (19); Zolov, Bourilkov, and Babaljev (21); Rubino, Concina, and Scansetti (18)]. In the second group are authors who describe regional concentrations of pleural calcification with no evident contact with asbestos and who, for this reason, reject an asbestos etiology. Prominent in this second group are Czechoslovak workers [Hromek (8); Marsová (13);

Rous and Studený (17)]. Graubner (7) arrived at similar conclusions as a result of studies in Halle in East Germany. Graubner's results, however, were recently reviewed by Strubel (20) who found in some of the cases, a positive history of prior exposure to asbestos dust (insulation workers) or to talc or mica exposure.

We have previously found, in a 16 year study of employees of plant A (in which primarily Soviet and Canadian chrysotile has been used) that pleural calcifications are closely associated with long exposure to asbestos (14). When we extended these studies in 1969 to a population not occupationally exposed, we found rather surprising results (15). We now report our epidemiological results to 1971. Pathological-anatomical, functional and biochemical findings will be reported elsewhere.

MATERIALS AND METHODS

We include in the group of pleural calcifications, persons with X-ray evidence of linear, flat or irregularly opacities, mostly bilateral and often on the diaphragms (with often well-maintained costophrenic angles) with no known inflammatory pleural disease, haemothorax or pneumothorax.

We have examined a large portion of the population in district N, in which factory A is situated. We classified individuals studied in three groups.

1. Persons with long occupational exposure to asbestos dust.
2. Persons with less intimate contact with asbestos.
3. Persons of district N with no known exposure to asbestos.

X-Ray studies were carried out by a mobile unit in plant A and by a stationary unit at District Hospital N. Persons with suspicious findings were asked to appear at our Clinical Unit for further detailed examinations. Persons residing in distant regions were examined by physicians of the respective Departments of Occupational Health, who sent us their complete findings for assessment.

RESULTS

The first group consisted of approximately 800 workmen of factory A employed for more than 10 years. We carry out roentgenographic screening in the factory each year, and all persons with findings suspicious of asbestosis, pleural calcifications or other lung changes are admitted to our Clinic for further detailed examination. Interpretation of the films is undertaken by two readers (Dr. Veselsky of the Department of Occupational Health, Prague, and Dr. Navrátil). Each year a number of the older employees are pensioned and younger employees are newly recorded as they reach 10 years of exposure. For this reason the number changes from time to time but it usually approximates 800. We continue observation of those retired on pension, with examinations each year, and follow-up to possible death.

Appendix No. 1

For example in the abreographic survey 1971 the age distribution was as follows:

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Age	No. of persons	%
31-40	190	25.2
41-50	245	32.5
51-60	182	24.0
61-70	126	16.7
71 and above	12	1.6
Total	755	100.0

In this employed group we found 42 persons with pleural calcifications (5.3%); 23 of them had simultaneous X-ray signs of asbestosis, 19 had only isolated findings of pleural calcification.

Appendix No. 2

The age distribution of persons with pleural calcifications ascertained within the entire period of studies, i.e., during 16 years, was as follows:

Age	No. of persons	%
31-40	1	2.4
41-50	4	9.6
51-60	19	45.0
61-70	16	38.1
71 and above	2	4.9
Total	42	100.0

The stated age limits relate to the time when we ascertained signs of pleural calcifications for the first time and thus these age limits do not mean the present age of the patients. Till the present day already 6 of the recorded persons have been recorded as deceased. It is important that only one of the patients in which we ascertained pleural calcification for the first time in 1959 was below the age of 40 (36 years). The absolute majority of persons with pleural findings ranges within the age groups of 51-60 years and 61-70 respectively.

The second group, with less intimate exposure, may be divided into two subgroups:

a. *The population living in the neighborhood of the factory* (those over the age of 20). This group includes individuals who are not and never were employed at the factory.

b. *Blood relations of factory employees, older than 20 years.* Asbestos air pollution in the vicinity of the factory may have been heavier 20, 30 and more years ago. At present, environmental dust concentrations and chrysotile content in the airborne dust in the vicinity of the factory, are lower because of improved industrial hygiene practice in the last 15 years (Šimečewk, Internal Hygienic Report 1969).

Blood relations may be assumed to have some increased exposure by virtue of their daily contact with employees, who may have come home with contaminated work clothes.

There were 155 persons in the neighborhood group. Pleural calcifications were present in 9 (5.8%). Five were also blood relatives of employees.

It was quite difficult to investigate blood relatives of exposed factory employees. In some cases, we found it necessary to ask physicians from other

regions, to which these relatives had moved, for cooperation. Still, we were able to trace more than 90% of persons in this group, examining 114; 91 relatives lived in district N and 23 had moved to other regions. Pleural calcifications were present in 4 (3.5%) two living in the same district, two other having moved to distant regions, more than 15 years before.

The third group was the most numerous and included persons living in district N but not in the neighborhood of the factory. During area X-ray surveys in 1970 and 1971, we selected persons above the age of 40, not working in plant A.

Appendix No. 3

The population below the age of 40 was also examined at the Department of Lung Diseases in N within the abreographic survey ordered by the State Health Administration for prevention of tuberculosis. No findings on the pleura complying with our criteria were reported however. For this reason we directed our attention towards the population above the age of 40 already because the age distribution of patients with pleural calcifications at plant A determined the age limits which the survey was supposed to respect.

8133 persons were so examined. In 36 (0.44%) there was a finding of pleural calcification. In this population survey the films were read by Dr. Trippé, Chief of Department of Lung Diseases in N, and by Dr. Navrátil. Detailed examination of persons with such findings demonstrated, to our surprise, that 8 of them had worked in factory A before World War II. However, 28 persons had no history of direct or indirect asbestos exposure. They were employed in agriculture, on the railroad or in different service occupations. Many were retired. These 28 cases of pleural calcifications gave an incidence of 0.34%, among the remaining 8097 examined.

It is evident that although the general population of District N with no known relation to asbestos exposure was by far the largest group (Table 1), pleural

TABLE 1
PREVALENCE OF PLEURAL CALCIFICATIONS IN OBSERVED GROUPS

	No.			
	Examined persons	Pleural calc. findings	Expected no.	Significance (χ^2)
Inhabitants of the district N main group (general population)	8133	28 (0.34%)		
Asbestos workmen in asbestos factory A	~800	42 (5.3%)	2.75	225.4*
Inhabitants of the neighbourhood about factory A	155	9 (5.8%)	0.53	102.2*
Blood relatives of asbestos workmen	114	4 (3.5%)	0.39	29.2*

* Prevalence of pleural calcifications in the general population group is significantly lower compared with other three groups ($p < 0.01$).

calcifications in the groups with direct or indirect exposure to asbestos dust were so numerous as to strongly affect the prevalence of pleural findings in the population as a whole. Among the factory employees, there were 42 cases of pleural calcifications against an "expected" 2.75 (based on the unexposed general population); in the neighborhood group there were 9 cases against an expected 0.53, and in the family group there were 4 instances as compared with expected 0.39.

These results lead to the conclusion that the direct and indirect exposure to asbestos dust is associated with an increased incidence of pleural calcifications. It is also of interest that the relationship of prevalence of pleural calcifications between the general population group and the other three is statistically significant at the 1% level.

DISCUSSION

This report, reviewing the experiences of several different population groups, supplements our previous observations limited to exposed persons (14). They also tend to confirm results of English, Canadian and American authors, concerning effects in occupationally exposed populations (5, 6, 19).

It should be noted that in our group of the general population, without any known asbestos exposure, 0.34% showed quite typical findings. According to Finnish and other workers (10, 11, 16, 21) one could expect even here minimal but nevertheless definite asbestos exposure in some cases, as by airborne asbestos. However, from the point of quantitative effect, such asbestos exposure probably represents a very low hazard in terms of pleural changes in the general population living and working far from possible asbestos sources. Parenthetically, serpentine and amphibole minerals are not found in the soil of the region studied, with almost only sedimentary deposits found.

Although most cases of pleural calcification in this investigation were related to direct or indirect asbestos exposure, some had no such history. The question might properly be asked whether the causes in such cases were the same as in other series in which asbestos exposure could not be identified (8, 13, 17). We are not able to give a clear answer. Comparison of the results in the several studies shows considerable variation (Table 2). It is difficult to say whether

TABLE 2
PREVALENCE OF PLEURAL CALCIFICATIONS IN GENERAL POPULATION*

Author	No. of persons examined	Pleural calcifications	%
Rous and Studený (17)	9760	644	6.59
Hromek (8)	9871	273	2.79
Maršová (13)	44549	241	0.54
This study	8133	28	0.34
Zolov, Bourilkov, and Babaljev (21) ^b	3325	155	4.66
Kiviluoto (11) ^b	35000	77	0.23

* Review of published investigations.

^b Certain environmental asbestos exposure is presumed.

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such variation is the result of variations in "nonasbestos" calcification factors or secondary to differences in conditions of study in the reported investigations (as, X-ray criteria of calcification).

Our findings clearly demonstrate that asbestos (chrysotile) exposure is associated with the occurrence of pleural changes and that its influence is far greater than any other supposed "unknown factor."

No signs of tuberculosis were found in any of the persons examined, irrespective of the group concerned. The observations by others studying populations without asbestos exposure, provided no evidence that there might be a tuberculous background to the changes. Considerations of calciphylaxis (Selye) may be justified, and we shall discuss this in our report on biochemical changes.

SUMMARY

Epidemiological studies demonstrate that pleural calcification in persons with direct and indirect exposure to asbestos, are most probably caused by inhaled particles of asbestos. The incidence increases with the elapsed period from the start of exposure and duration of exposure. Fibrosis proceeds even when dust exposure has ceased, for many years. Our clinical material provides clear evidence for this slow but progressive development of the pleural process.

Finally, some cases of pleural calcification may occur without known asbestos exposure. Their cause has not yet been clarified.

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