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FURTHER OBSERVATIONS OF OCCUPATIONAL CANCER AMONG ASBESTOS MINERS

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ASBESTOS INFORMATION ASSOCIATION/

The knowledge available with regards to occupational cancer among asbestos miners is essentially due to the research work performed over the past 15 years in conjunction with the development of the asbestos industry. As we can see from the available literature, asbestosis was described in England and America before it became known in Germany. The majority of scientific works in England were published between the years 1927 and 1929 whereas asbestosis was not studied in Germany until the year 1931. One case of pulmonary asbestosis studied by Beger and Sroebe in Hannover is particularly responsible for the extensive progress made and the new interest aroused in the field of research with regards to this specific occupational disease.

Two of my own observations, as well as a few other works published in America and England on the relationship between pulmonary asbestosis and lung carcinoma, encouraged Nordmann to study this cancer problem in the year 1938. In his work, Nordmann was already able to compile six cases in which asbestosis was accompanied by a carcinoma of the lungs. Meanwhile, a few additional, as yet unpublished, cases were brought up at a meeting of the German committee on asbestosis in Dresden and Nordmann was therefore able to report on a total of 11 cancer cases in asbestosis patients at the 8th International Congress on Occupational Medicine and Diseases held in Frankfurt a. M. in the year 1938. Given the concurrence of the clinical and pathological-anatomical pattern of these cases, Nordmann came to the conclusion that the simultaneous presence of these two diseases was not coincidental but rather presented a picture of "occupational cancer among asbestos miners," confirming a causal correlation between asbestosis and lung cancer. Representatives of German trade associations also agreed

with this theory and recognized lung cancer among asbestos miners as an occupational disease.

Although earlier researchers in America and England were reluctant to agree with the alleged significance of asbestos dust for the development of primary lung carcinoma, these doubts no longer seem justified since Nordmann and Sorge have since been able to induce lung cancer in animals through the administration of asbestos dust. During the said experiments, it was possible to promote the development of a cornified multicentric squamous cell carcinoma in 20% of the mice receiving asbestos dust, as well as epithelial tumors in all stages up to atypical changes in 42 to 57% of the animals.

Since then, Linzbach and Wedler described one additional case of lung cancer in a German asbestos miner; we are not in a position to report on our own observation of the third and fourth cases of lung cancer due to asbestos dust--the latter was confirmed both by the autopsy and the histological analysis. This raises the number of primary lung carcinomas in asbestosis patients in both domestic and world-wide literature to 14 cases, including six which were observed in Germany itself.

We also consider the publication of these two cases to be particularly important since we were actually able to see the development of the carcinom on X-ray films.

Case 1. The first case involves H.B., a 43-year-old worker employed in an asbestos plant for a period of 11 years between the ages of 21 and 32, i.e. between 1920 and 1931. He was employed in a different plant from those previously reported. During that period of time, he worked for the

first four years in the dust cleansing department and the asbestos dust preparation department; after that he was employed mostly in the carpentry works and in the packing room where he was more exposed to the effects of asbestos dust. He was released in the year 1931 for lack of work.

The family history provided no evidence of pulmonary or congenital diseases. From his own history, we learned that in the year 1917 while in the military in Macedonia, he was confined to his bed for a period of six weeks due to severe pleuritis sicca. In the time that followed, he frequently suffered from bronchitis and even had repeated relapses of pleuritis. This case is all the more interesting since the course of the disease was studied thoroughly and over a long period of time. B. was originally a baker but had to abandon this occupation in 1920 due to repeated cases of bronchitis and constant coughing spells due to the flour dust. In 1925, i.e. after a 5-year employement in an asbestos plant, he was hospitalized and found to have lung tuberculosis. Tubercle bacilli were never found in the sputum, even during later examination by a specialist. During a medical evaluation performed in 1928, it was recommended that he be sent to a sanatorium for a period of three months. At that time, X-rays showed an enlarged hilus, particularly on the left, and the physical examination showed isolated areas of rattling crepitation during vesicular respiration. The medical examination also revealed a bilateral hilus tuberculosis and probably stationary dense tuberculosis in the area of both lower lobes with pleuritis on the right without formation of cavities. Since then he was repeatedly treated for lung disorders consisting primarily of irritation of the throat, minimal

expectoration and shortness of breath with stress. He was again hospitalized in a sanatorium in 1932. The constant worsening of his condition made it necessary for him to again be hospitalized in a sanatorium for a period of four months in the year 1934.

During his last stay in the sanatorium, the treating physician came to the conclusion that this was not a case of lung or hilus tuberculosis but rather very probably a severe case of asbestosis as indicated by the patient's occupational history. The steps taken during that year to have this lung disease recognized as an occupational disease were dropped since pulmonary asbestosis had not as yet been recognized as an occupational disease in the second legislation on accident insurance coverage applying to occupational diseases. It was only after the third legislation of 12-16-36 had finally recognized asbestosis diseases as occupational diseases subject to compensation that a new application was submitted and the diagnosis of hilus and lung tuberculosis was corrected to indicate asbestosis of the lungs.

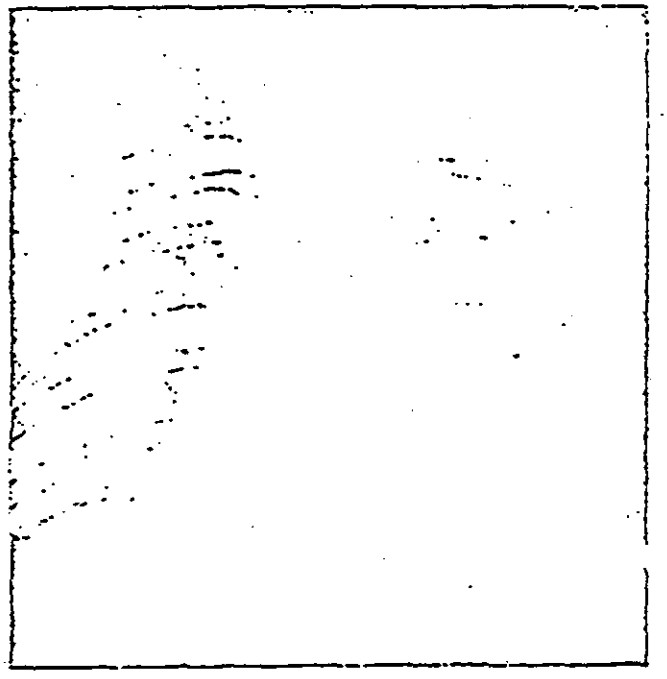


FIGURE 1. Case 1. X-ray taken in 1928. Definite enlargement of the hilus with fine and minute thickenings in both lower lobes.

During an examination performed by Dr. Feuerhake of Hannover in May of 1938, B. complained of a shooting pain in the left side of the chest, extreme shortness of breath even with minimal strain and irritation of the throat with some tenacious expectoration. Clinically his condition was extremely weakened with asthenic habits. Weight: 49 kg; height: 154 cm. Marked dyspnea at rest with 34 respirations per minute, symmetrical respiratory excursion of the thorax, insufficient extension with respiration (77.5-79.5 cm). Minimal displacement of lower lung limits. Physical findings minimal, including sharper respiratory sounds and fine crepitation, particularly over the lower part of both lungs and more so on the left than on the right. No tubercle bacilli. Heart normal, pulse regular. Blood pressure 65/115 mm Hg, blood sedimentation considerably accelerated.

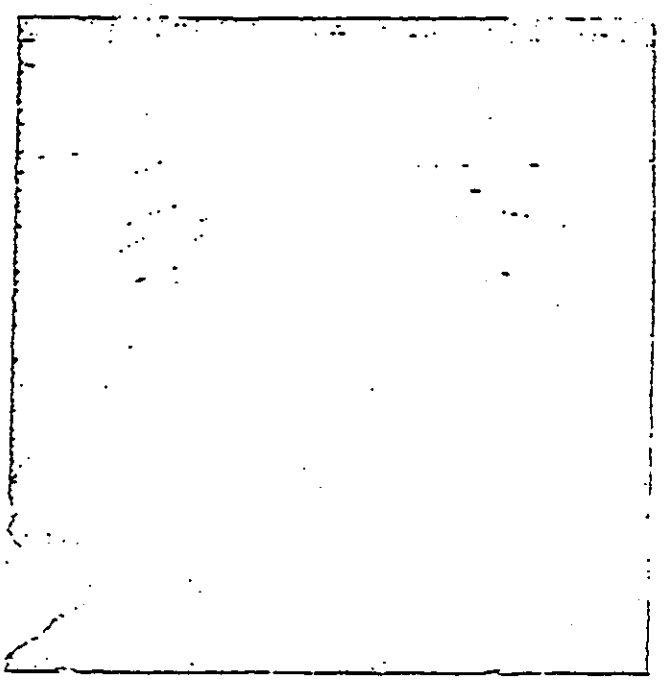


FIGURE 2. Case 1. X-ray taken in 1938. Extensive thickening of both lower lobes with occasional fine and minute converging spots.

The last examination and observation by a specialist took place in October of 1941. The patient seemed very ill and weakened. The thoracic pain had increased and was localized primarily in the left part of the chest. He complained of severe throat irritation with minimal expectoration, exhaustion, and loss of appetite. Shortness of breath was even worse than before; dyspnea at rest with 42 breaths per minute. No tubercle bacilli but numerous asbestos particles to be detected in the sputum. Chest circumference was 78.5/79 cm; thorax hardly dilatable. The left half of the thorax was somewhat sunken in as compared to the right. There were marked signs of circulatory decompensation with cardiac hyperplasia on the right, increased pulse frequency and extremely low blood pressure. A half-spherical broad-based undisplacable tumor was present on the back over the left lower part of the thorax; the latter was firm and tender to the touch. The growth spread all the way to the lower rib cage and, above, up to three finger widths below the lower edge of the shoulder blades. With the exception of moist weakened respiratory cells and the lack of tactile fremitus in the lower posterior half of the thorax, there were no clinical

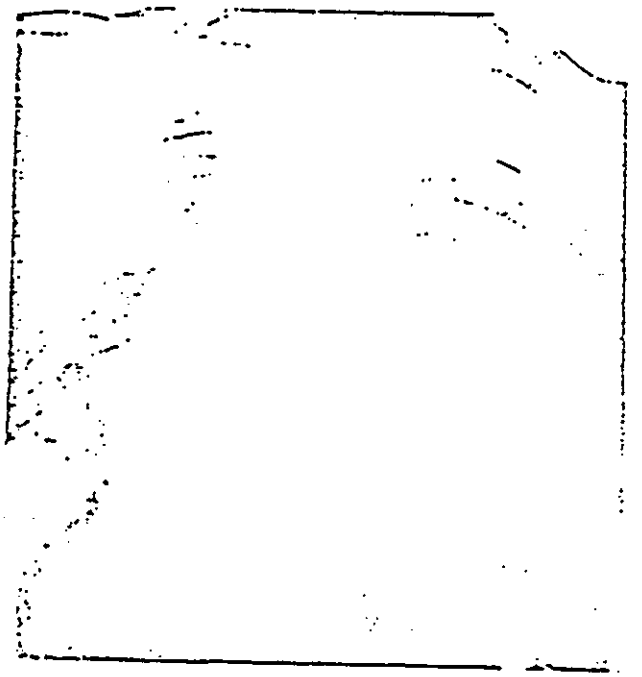


FIGURE 3. Case 1. X-ray taken in 1941. Advanced fibrosis. Homogenous shadow over the left lower lobe.

changes to be observed on the left side of the lungs as compared to the previous examination. The X-ray showed that fibrosis of the lungs had progressed also. Both fields were covered with dense spots, mostly about the size of a needle head, mostly badly defined and covered with regular veiled areas.

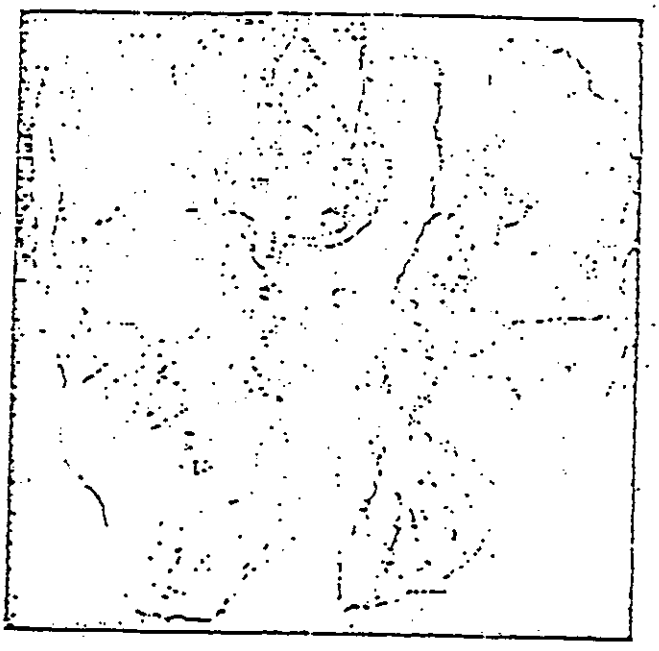


FIGURE 4. Case 1. Squamous cell carcinoma in left lower lobe with diffuse fibrosis of the lungs.

The least affected areas were the upper lung parts; the right complementary space was almost free of any changes. The left lower lung field showed, starting with the fourth rib, a homogenous shadow with a clear area in the lower lung field over the first diagonal diameter, between the cardiac wall and this peculiar shadow. Around the aforementioned tumor, the tenth rib showed extensive degeneration of the outer part whereas the pleural corticalis remained unaffected and well-kept. Based on the roentgenological findings, it was assumed that there was also a periosteal rib sarcoma beginning from the tenth rib.

A few weeks following this examination, characterized by a rapid loss of strength, the patient died on 11-3-41 under conditions of increasing circulatory loss and respiratory insufficiency.

During the autopsy (Obd. #736/41), a highly contracted area was found between the lungs, particularly around the left lower lobe. The latter was shell-shaped and had grown together with the pleura. The cross sections of the lungs had a grayish-black scaly appearance with firm whitish connective tissue processes and a reticular pattern. This dense, thick pattern was particularly evident in the lower lobes and around the hilus. In the upper parts, the connective tissue induration was less marked and the peripheral areas were emphysematous. In the left lower lobe, there was a firm grayish-white badly-defined tumor about the size of a chicken egg with a hand palm-sized involvement of the Pleura costalis around the 7th, 8th and 9th ribs, as well as circumscribed degeneration of these ribs. The externally palpable tumor on the left side of the back, on the other hand, was extrapleural and was totally unrelated to the lung tumor; nevertheless, this had caused extensive degeneration of the 10th and 11th ribs. The hilar lymph nodes as well as the bifurcation lymph nodes were enlarged and grayish-black in color but without connective tissue induration. The axillary lymph nodes on the left, however, showed diffuse metastases. There was considerable dilatation of both cardiac ventricles as well as marked hyperplasia on the right. There were otherwise no noticeable pathological or anatomical changes to be observed in the organs.

Histologically, we found diffuse fibrosis consisting of a connective tissue enlargement of the alveolar septa in the upper lung parts. There was increased lung fibrosis in the middle sections and more particularly in the lower lobes up to the broad callosity area. The pulmonary alveoli

are only present in the form of small deformed hollow spaces. There are occasional lymphocytes and infiltrations of plasmatic processes. All lung sections are found to contain numerous asbestos particles of all shapes as well as asbestos needles sometimes grouped together in large bundles. These can be found in the alveolar spaces as well as in the connective tissue section without their being any evident regularity in the distribution of asbestos particles and needles; these are mostly surrounded by giant cells and macrophages. The carcinoma observed in the left lower lobes has a structure similar to that of squamous cell carcinoma but with more extensive cornification and infiltrative growth in the connective tissue and in the alveoles. However, the bronchi are also filled with carcinomatous infiltrations. In the cancer-free lung sections, especially in the area of the lower lobes, we can see numerous bronchial epithelial growths partly present in the form of squamous cell metaplasia. The morphological image shows the degenerative rib tumors and the enlarged axillary lymph nodes to be typical metastases of lung carcinoma.

Case 2. Our next observation concerns an asbestos worker employed as mixer in an asbestos plant for a period of 21 years. During that time, he developed a severe case of asbestosis and died of lung cancer localized in the asbestosis seat 30 years after his initial employment.

The autopsy was requested by the patient's treating physician and family since it was known that the man had been employed for several years as mixer in an asbestos plant and had developed asbestosis but the cause of death and its possible relation to an occupational disease had not been determined.

During a mass examination of the employees of the asbestos plant performed three years prior to the man's death, the occupational physician in Hannover had recommended that this man who was suffering from asbestosis II-III retire from the plant and find a dust-free occupation; however, since then he had lost track of him.

When questioned, his 56-year-old wife gave the following information in regards to the man's history: He was a trained turner but had lost his left eye even during the training period so that he was receiving a 25% pension from a trade association.

Between 1912 and 1931 he had continuously worked in the same department of an asbestos plant, i.e. in the asbestos carding department, and had left this position only because the plant had shut down due to lack of business. During the period from 1931 to 1937 he was then either unemploy or did part-time work in various plants. The asbestos plant then reopened in 1937 and he was able to resume his occupation until 1939 at which time he was advised by the above-mentioned physician to retire from this occupation. He apparently was very surprised by the result of that examination since he had had no complaints whatsoever. Even after that, his wife stated that he had not suffered from any abnormal cough. He then went to work in a chemical plant where he had previously worked (prior to 1912) and was listed as disabled on 3-26-42. His wife reports that during the course of the last cold winters he had not appeared as healthy as before and that this was not due to any improper nourishment or disease. He always came home totally exhausted and finally seeked the advice of a physician. The physician related his problem to the previously diagnosed

asbestosis. During the eight weeks that followed, he continued to lose his strength. Finally, both his family and physician recommended that he see an internist and the latter, in turn, referred him to Friederikenstift Hannover a few days prior to his death. Once hospitalized, his condition was found to be so severe that it was impossible to determine an accurate diagnosis. A palpable tumor was found in the right upper abdomen, probably over the liver, and the X-rays showed a large shadow on the right side of the right lung lower lobe. The patient was considerably obstructed with mucous and died of exhaustion.

The autopsy (Obd. #304/42) revealed a rather thick indurative growth in the lungs and a moderate brownish induration of the lung tissue with a more or less even distribution of coals in the nodules of plates to trefoil-shaped sections. A cancerous area about the size of a fist was found in the right lower lobe, which had grown through the right diaphragm so that a large tumor had formed in the right hepatic lobe. Other deposits were found in the lymph nodes of the mediastinum, the hepatic roots and the vertebrae.

The inner organs showed moderate chronic congestion. The right cardiac ventricle was highly enlarged and distended with fine callosities on the posterior wall of the left ventricle. Secondary findings also included a few gallstones.

As could be expected based on the macroscopic image, the microscopic examination of all lung parts revealed the presence of coal as well as induration of the lung tissue in the form of strands and nodes evidenced by an irregular fibrous enlargement of the septa. Accordingly, the alveolar

spaces in this area are the narrowest. In addition to the phagocytes filled with coal dust, these contain compressed alveolar cells and large quantities of asbestos needles and asbestos particles. Between these, the alveolar tissue is obviously inflated and each view shows a large quantity of asbestos particles. There are several atypical epithelial growths in smaller cell fields of the right lower lobe; the cells are small, atypical and uncharacteristic. The tumor stroma is exceptionally wide and consists mainly of collagenic fibers. The cancer in several areas is necrotic so that the collapsed cells can easily be identified as induration. The lymph nodes of the lung roots contain large amounts of coal dust as well as hemosiderin and short isolated asbestos particles or larger spherical hemosiderin lumps.

X-rays taken in 1939 and 1942 (Figs. 5 and 6) show a similar development of carcinoma during the patient's last few years. Although in the first case it was still impossible three years prior to the patient's death to recognize the future seat of cancer in the left lower lobe on the X-rays, the second case three years prior to his death already showed a strand-like shadow formation in the right lower lobe which was considerably thicker than that of the left. The usual symmetry of lung fibrosis is missing here and this is actually where the fatal carcinoma develops. This is totally in agreement with observations previously made on anatomical preparations, i.e. that the atypical epithelium develops in the proliferated connective tissue of bronchial growth (Nordmann) or in the immediate vicinity of the latter (Nordmann and Sorge).

The clinical course as well as the pathological-anatomical pattern of our observations indicates a series of characteristics of this occupational disease of asbestos workers. According to previously published cases of asbestos cancer, we can say that,

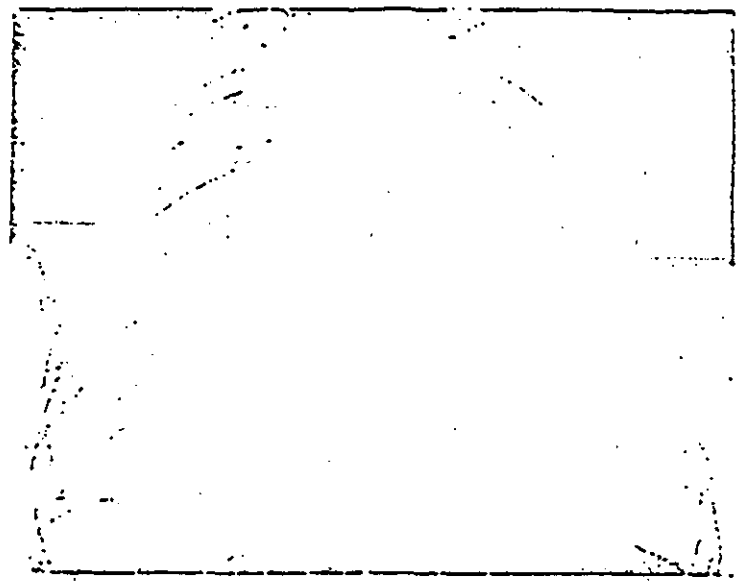


FIGURE 5. Case 2. X-ray taken in 1939. Definite enlargement of the hilar shadows bilaterally with strand-shaped thickening of both center fields.

contrary to the usual cases of lung carcinoma, approximately half of the cancer cases were relatively young; two cases were 35 years old, another was 41 and, in our cases, the cancer carriers were aged 43 and 56, respectively. The latency period between the beginning of employment in an asbestos dust environment and the time of death due to lung cancer also shows similarities; this was found to be 15-21 years on the average. This was found to be the case in our observation where this was found to be 21 years; in the second case it was even 30 years. According to statistics published by Bridge, the average latency period for uncomplicated cases of asbestosis is 15.1 years. According to the same authors, this period of time would be 42.5 years for silicosis and silicosis patients would reach the cancer-hazardous age much more frequently than asbestos workers although carcinoma rarely appears in normal

silicosis cases. This partly confirms the probability of lung fibrosis in asbestosis being a promoting factor for the development of lung cancer.

According to Nordmann, if we calculate the percentage of all autopsy-confirmed cases of asbestosis in Germany, we get approximately 20% taking into consideration the relatively small number of cases. Out of 50 cases of asbestosis confirmed during the autopsy, Gloyne found six cases of carcinoma or a percentage of 12% which should be much closer to reality. These clinical figures more or less agree with those of Nordmann obtained through experimental studies in which he was able to induce lung carcinoma in 20% of the mice treated with asbestos dust.

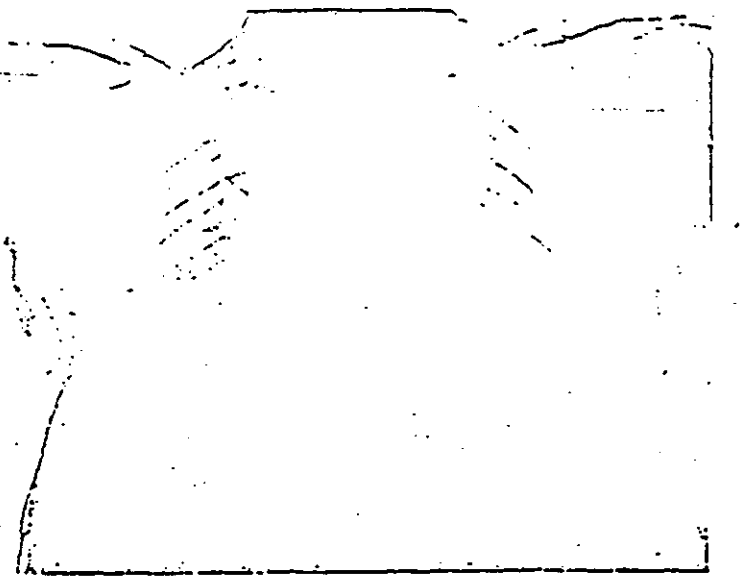


FIGURE 6. Case 2. X-ray taken in 1941. Thickening of all strand shadows. Wider homogenous shadows in lower right central field; carcinoma seat located in right lower lobe as determined during the autopsy.

The duration of the employment in the asbestos dust environment was generally between 7 and 8 years with the exception of one case reported by Nordmann where a patient was suffering from asbestosis after as little as one and one-half years. It has repeatedly been reported, for instance in Linzbach and Wedler's case, that the patient had only been exposed to asbestos dust during the first three years of his occupation. In our first case, the dust inhalation only lasted four years while the patient

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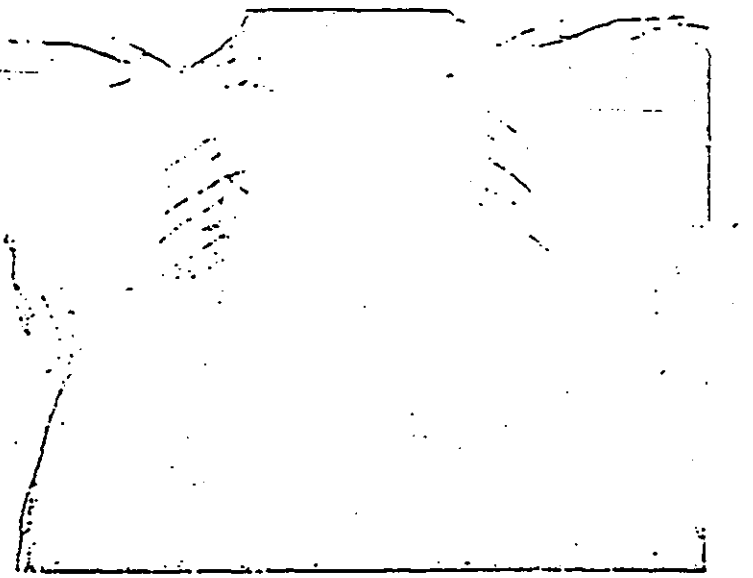


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was employed in a dust-filled environment. This confirms the fact that asbestosis can develop with minimal inhalation of asbestos dust and that the development of lung fibrosis is not impeded by leaving the dust-filled atmosphere. On the contrary, there seems to be a definite tendency for it to continuously develop even several years after inhaling the dust. On the other hand, long periods of employment have also been observed. In our second case, the patient worked for 21 years with asbestos in a high-risk area. This seems to be a very good example of a highly resistant male who was able to cope with a severe case of asbestosis until he too became a casualty of occupational cancer.

During the clinical course, at least during the initial stages, it is difficult to differentiate the disease from tuberculosis. For that reason, tuberculosis was almost always diagnosed before pulmonary asbestosis became known; this is in fact what happened in our first case. The X-rays taken during the early stages of asbestosis also show few characteristic signs. This disease almost always begins with a swelling and enlargement of the lung roots which gradually spread to the lower lobes and slowly to the upper lobes. This X-ray evidence in the sense of hilus tuberculosis, however, must be doubted since isolated hilus tuberculosis is rarely observed among adults. In the more advanced stages, the X-rays allow a fairly definite differentiation of asbestosis from lung tuberculosis. The fine and minute occasionally-grouped spots in the lung field which always increase downward, without any coarse bundle shadows, are highly indicative of asbestosis. This finding would only be indicative of tuberculosis if accompanied by a miliary spreading, but the clinical

pattern would again indicate the inaccuracy of such an assumption. Regardless of the fact that with one form of asbestosis, in spite of the extensive lung changes, no tubercle bacilli were to be found but rather mostly asbestos particles, we must remember that the physical findings in lung asbestosis are mostly minimal and do not show any definite signs of disease. However, it is not only the X-ray image and the minimal physical symptoms that do not indicate tuberculosis. On the contrary, the suspicion of pulmonary asbestosis in the more advanced stages of the disease should be confirmed by the general bad condition of the patient, marked respiratory insufficiency with dyspnea at rest, hyperplasia of the cardiac musculature with occasional cardiac arrest on the right, as well as highly accelerated blood sedimentation and evident low pressure which is often observed. With the exception of this, however, lung asbestosis though not as frequently as silicosis can sometimes be accompanied by tuberculosis. According to a summary published in England by Wood, 30% out of 100 cases of asbestosis observed were accompanied by tuberculosis.

Even on a pathological and anatomical level, previously observed cases of asbestos cancer agree with our own observations. In the majority of cases the carcinoma has its seat in the lower lobe and five out of six cases are actually cases of cornified squamous cell carcinoma. This means yet another characteristic since lung carcinoma as we know usually develops from the large bronchi and is actually bronchial carcinoma whose histological structure is usually that of a circumscribed epithelial growth of non-specific cellular character, i.e. the carcinoma simplex as it is

called. In rare cases, this can be small-cell immature carcinoma or adenocarcinoma of alveolar epithelial origin. Squamous cell carcinoma is at least as rare as the two aforementioned types of cancer. During the histological studies of the previously published cases, Nordmann and Wedler were able to find frequent growth and metaplasias of the bronchial epithelium in non-carcinomatous areas of the lungs. This was again observed in our cases since epithelial proliferations and a metaplastic bronchial epithelium could also be observed in areas unaffected by the carcinoma. We are therefore justified in considering this degenerative process of the bronchial epithelium as the point of origin of asbestos cancer and the isolated adenocarcinoma must be considered as exceptional in cases of asbestosis. With regards to this a comparison should be made to Schneeberg lung cancer observed among Schneeberg miners since squamous cell carcinoma of the lungs was often observed there too.

However, this formal genetic explanation of asbestos cancer does not explain the causal genesis, i.e. the promoting factor the development of cancer formations. As far as Schneeberg cancer is concerned, it is assumed that the chemical structure of the ore is the main cancer-promoting factor. It is therefore only normal that we should consider the possibility of asbestos also having a chemically carcinogenic effect. The thorough studies performed by Beger have shown that, in addition to manganese, iron and calcium, asbestos needles also contain silicious earth which is released with the needles and is responsible for the tissue injuries suffered by persons suffering from asbestosis. Contrary to this, however, Sundius and Bygden during their studies felt that the effect of asbestos was primarily a mechanical one. Di Biasi shares this opinion which is

generally accepted nowadays. However, I feel that we should neither deny the chemical effect of silicic acid nor assume that asbestos dust has an exclusively mechanical influence. The experimental studies performed by Siegmund and Koppenhofer using colloidal silicic acid also confirmed the noxious effect of silicic acid consisting primarily of connective tissue proliferation. On a clinical level, it should also be noted that the observation of a few cases of lung fibrosis due to silicious earth also constitutes further evidence of the mode of action of silicic acid. None of the observations, however, indicate that the direct cancer-promoting effect of asbestos lies in the silicic acid; this is particularly emphasized by the fact that lung carcinoma is rarely observed with silicosis. We must, however, not exclude the possibility of other unknown carcinogenic substances being present in asbestos.

On this point, Linzbach and Wedler also mention the presence of skin warts observed in asbestos workers and which could also be due to the effects of asbestos. However, the development of such warts has never been found to be carcinomatous.

Thereupon we attempted to find in the morphological changes an explanation for the high incidence of cancer in asbestosis patients. Nordmann as well as Linzbach and Wedler particularly discussed this question and determined the characteristic nature of lung asbestosis. Although with usual cases of silicosis we find circumscribed connective tissue indurations in addition to fibrous concentrically layered nodules and nodes, we found in cases of asbestosis proliferation and regeneration processes over the entire lungs consisting of a regular

diffuse fibrosis. In chronic indurative tuberculosis we find no diffuse induration but rather a circumscribed induration of the lung tissue next to the normal lung section, as was the case with silicosis. Also to be noted in the morphological picture of asbestosis is the lack of extensive tissue necrosis and the relatively minimal inflammatory process of the lung tissue. In the foreground, we have the diffuse connective tissue proliferation and an abundant formation of giant foreign body cells in the immediate vicinity of the asbestos needles and asbestos particles. In the same diffuse fashion, the proliferative changes can be seen to take place in the alveolar and bronchial epithelia. Contrary to silicotic induration which consists only of firm collagenic connective tissue, in asbestosis we find normal epithelial parts of the lungs in the middle of reticular scar tissue in addition to more or less severe inflammatory epithelial proliferations up to regular squamous cell metaplasia, whereby the normal tissue coordination should be distorted. In addition to the fibrous regenerative processes, the long lasting irritation of the lungs due to asbestos dust also results here and there to multiple excessive regeneration processes which can constitute a preliminary condition for the development of a carcinomatous growth as determined according to other scientific, clinical and experimental experience. We can therefore expect that lung carcinoma in asbestosis would be diffuse and develop exclusively in the area where the morphological changes are most severe; experience has shown that this would be in the lower lobes. The multiple origin of asbestos cancer as determined by Nordmann based on the results of his experiments is again clinically confirmed by our own observations. As

previously said, the extent of the cancer-promoting role of other chemically carcinogenic substances present in asbestos cannot be determined at this time.

To summarize, we can say that there can be no doubt as to the causal relationship between asbestosis and lung cancer, even if the causal genesis of this special type of cancer cannot be explained at this time. From a morphological point of view, the tissue shows predisposing characteristics for the development of cancer thus providing a formal genetic explanation for these tumors. Given the rarity of asbestosis, the 14 previously observed cases of lung cancer with this type of coniosis represent a relatively frequent complication of this occupational disease producing a percentage rate ranging between 12 and 17%. Medical follow-ups and mass examinations should help to determine which employees should be kept away from asbestos plants due to respiratory disease predisposition and excessive sensitivity of the mucous membranes. Of course asbestosis can develop even without such predispositions to chronic bronchitis. In our first case, it was certainly not the patient's first occupation as a baker which could have resulted in chronic irritation of the bronchi due to the flour powder environment but rather his second occupation in an asbestos plant. Only four years working and inhaling relatively small amounts of dust were sufficient to determine this man's destiny. The second case was actually detected during such a mass examination by a work physician at which time he was removed from the site without the occupational cancer being in any way impeded. The clinical symptoms alone therefore are not sufficient to allow an accurate prognosis of asbestosis to be made.

By publishing these cases with a typical clinical as well as pathological and anatomical pattern, we wish to again arouse your interest in this specific occupational disease. We would also hope that in view of the constant expansion of the asbestos industry, newly developed proper preventive measures will help reduce the number of casualities due to this occupational disease to a minimum.

SUMMARY

Two new cases of occupational cancer among asbestos workers are reported and the development of carcinoma in an asbestosis seat is demonstrated by a series of X-rays.

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