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ASBESTOSIS

REPORT OF TWO CASES *

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Asbestos as such was known to Pliny. Charlemagne is said to have possessed a table cloth made of it, which was cleansed by passing through fire. The mineral deposits of asbestos are found in Canada, Italy, Africa and Rhodesia. It is a silicate occurring in minerals in combination with iron, copper, calcium or magnesium. It occurs in the form of fibers which can be woven. In addition, finer particles are used in the manufacture of sheeting, tiles, asbestos millboard and paper. The increasing need for brake linings for motor cars, fireproof curtains, insulations, mattresses and steam packings has expanded the spinning industry considerably. The use of the dust in cement and plaster for the erection of fireproof buildings has increased the danger of inhalation in the most hazardous form of the industry. Its expansion from 500 tons in 1880 to 330,000 tons in 1925 and the fact that methods of manufacture have been introduced for utilizing the fine dust for new purposes have contributed to the beginning appearance of a pneumokoniosis due to inhalation of asbestos dust,[1] known as pulmonary asbestosis.

In 1906, Montague Murray[2] presented evidence before the Departmental Committee on Compensation for Industrial Diseases, that he had had under his care in 1900 a worker employed in an asbestos factory who developed respiratory symptoms and died. At the autopsy, extensive pulmonary fibrosis was found, which Murray attributed to inhalation of asbestos dust.

In 1924, eighteen years later, Cooke[3] reported a similar case complicated by pulmonary tuberculosis. He was the first to describe the

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1. Merewether, E. R. A.: J. Indust. Hyg. **12**:198, 1930.

2. Murray, Montague: Departmental Committee on Compensation for Industrial Diseases, Minutes of Evidence, Appendices and Index, 1907, Cd. 3496, p. 127; Report 1907, Cd. 3495, p. 14.

3. Cooke, W. E.: Brit. M. J. **2**:147, 1924; **2**:1024, 1927.

"curious bodies" in the lung now known as "asbestosis bodies," while McDonald [4] worked out the histologic features. The latter believed that the bodies were portions of asbestos in process of alteration and absorption by hydrolysis, with the passing of silica into a colloidal state and later a gel. Stewart and Stewart and Haddow [5] showed that these bodies could be found in the juices expressed from affected lungs and could be found in the sputum by digesting it with equal quantities of antiformin. Lynch and Smith,[6] working without knowledge of this, demonstrated these bodies by digesting sputum with 10 per cent sodium hydroxide. Gloyne [7] proved by means of dark-ground illumination, that when the golden-yellow asbestosis body was dissolved in concentrated sulphuric acid, it had a central core consisting of a minute asbestos fiber. The asbestos fiber itself under dark-ground illumination has the appearance of a sharp piece of wire. Fibers measuring 200 microns [8] can pass the protective mechanism of the upper respiratory tract and enter the lung.

REPORT OF CASES

CASE 1.—J. M., a white man, entered the Jefferson Medical College Hospital on June 8, 1931, in the service of Dr. Edward Klopp. He complained of headache, abdominal distention and eructation of acid and gas. A diagnosis of chronic cholecystitis was made. A cholecystectomy was performed. Peritonitis developed, and the patient died. The history disclosed certain facts relative to asbestosis. The patient had worked as a spinner in an asbestos mill for nine years. Although the atmosphere was not very dusty, he wore silver dust protectors in his nostrils. For five years following such employment, he worked as a laborer. During the past five years, he had morning cough with moderate expectoration. There was no history of hemoptysis or pain in the chest. He had some dyspnea and cardiac palpitation. Physical examination was unimportant with the exception that he had an emphysematous type of chest. The important roentgen observations were slightly increased peribronchial markings throughout both lungs and some calcific deposits in both root areas. The cardiac shadow appeared to be normal. The sputum was not examined.

At the postmortem examination, the heart appeared normal. The left lung weighed 270 Gm. and the right 320 Gm. They were bluish gray, practically free from adhesions and crepitant throughout. On section, no gross pathologic lesion was demonstrable. Microscopically, there was a moderate grade of emphysema.

4. McDonald, S.: Brit. M. J. **2**:1025, 1927.

5. Stewart, M. J.: Brit. M. J. **2**:509, 1928; **2**:581, 1929. Stewart, M. J., and Haddow, A. C.: J. Path. & Bact. **31**:172, 1929.

6. Lynch, K. M., and Smith, W. A.: J. A. M. A. **95**:659, 1930.

7. Gloyne, S. R.: Tubercle **10**:404, 1929.

8. Wood, W. B., and Gloyne, S. R.: Lancet **1**:445, 1930.

Some of the alveolar walls were slightly thickened. A few typical asbestosis bodies were seen in the alveolar walls. There were scattered small collections of brown and blackish dust, free and within phagocytic cells, in fibrosed areas. Much of this dust and all of the asbestosis bodies stained positively for iron. The liver and the spleen were not pigmented.

The question of the relation of pulmonary asbestosis to tuberculosis is an interesting one. The fact that they are frequently associated is settled. Whether tuberculosis becomes implanted on pulmonary asbestosis or whether the occupational disease lights up a quiescent lesion is a mooted question.[1] It is true that when tuberculosis occurs, it is liable to be of an anomalous type. Gardner and Cummings [9] exposed series of guinea-pigs to attenuated tubercle bacilli and to an atmosphere laden with asbestos dust and attenuated tubercle bacilli. They found, in the first instance, that normal guinea-pigs receiving the strain of attenuated tubercle bacilli showed tubercles in the lung and in the tracheobronchial lymph nodes comparable to the primary complex in man. The lesions caseated and healed by resolution. Spread of the infection with macroscopic disease in other viscera was rare. On exposing guinea-pigs to attenuated tubercle bacilli and to asbestos dust, 32.2 per cent of one group of animals showed some evidence of spreading tuberculosis. New disease began as a local extension from primary tubercles and metastasized to areas where dust reaction had occurred. The tendency to healing by fibrosis was marked. Macroscopic disease in the spleen was common and occurred occasionally in the liver. In another group receiving asbestos and attenuated tubercle bacilli, the localization of tubercles was atypical. Many bacilli were seen trapped in foci of dust reaction. Some produced local tubercles; others immediately entered dilated lymph vessels and were carried to the tracheobronchial lymph nodes. Tuberculosis of these nodes sometimes occurred without involvement of the lung. Early disease of the spleen and of hepatic lymph nodes occurred in the majority of cases. The combined action of tubercle bacilli and asbestosis in the lung produced more fibrosis than did either agent acting independently.

Case 2.—J. J., aged 52, entered a hospital in December, 1928. In 1921, he worked in an asbestos factory for a period of about nine months. In this factory, no precautions were taken against inhalation of asbestos dust. Exactly what his duties were was impossible to determine. On admission, he complained of fatigue, loss of weight and a slightly productive cough. Tubercle bacilli were demonstrable in his sputum at that time. On roentgen examination, a cavity in the right upper portion of the chest and a fine stippling in the lower portions of the lower lung

9. Gardner, L. W., and Cummings, D. E.: J. Indust. Hyg. 13:65, 1931.

fields suggesting pneumokoniosis were found. There was improvement; the patient was discharged and continued under observation as an ambulatory patient. In September, 1930, he was admitted to the Jefferson Hospital for Diseases of the Chest, in the service of Dr. B. Gordon. He complained of the same symptoms as before. Physical examinations revealed the following salient facts: He was markedly emaciated. The chest was emphysematous in type. Expansion was much limited, particularly in the right upper portion, where there was lagging. Over the apex of the right lung, physical signs indicative of a cavity were present. Over the remainder of both lungs, expansion was limited; tactile fremitus was increased; breath sounds were roughened and somewhat exaggerated, and there were numerous scattered râles. The area of cardiac dulness was within normal limits; the heart sounds were distant; there was a mitral murmur at the apex. The abdomen appeared to be normal. There were clubbing of the fingers and curving of the nails; the skin was slightly cyanotic. The patient left the hospital in March, 1931, against medical advice, somewhat improved, but he returned a month later with exacerbation of all his symptoms and an acute respiratory infection. His course was progressively downward; bronchopneumonia developed, and he died, April 2, 1931. At no time during his stay in Jefferson Hospital were tubercle bacilli found in his sputum. There were a moderate secondary anemia, a slight elevation of nonprotein nitrogen, and negative results from Kahn and Wassermann tests. On roentgen examination, Sept. 20, 1931, there was multiple small cavitation in the right upper lobe, and the interlobar pleura below it was much thickened. In the lower lobe, right, and in the middle and lower lobes, left, there was an increase in the markings that resembled a dust change. The heart appeared normal.

At the postmortem examination, the heart weighed 280 Gm. and measured 13 by 8 by 6 cm. The myocardium was mottled red and yellow, and appeared soft and dull. There were many firm, gray, translucent nodules on the free margins of the mitral valve leaflets. The coronaries were thickened and tortuous, and contained atheromatous plaques. The parietal pleura on both sides was thickly studded with small, discrete, gray, pinhead-sized nodules, firm in consistency. Similar ones were present, but much less frequently, on the visceral pleura. Over the bases of both lungs, firm fibrous adhesions completely obliterated the pleural cavities. Between the bases of the lungs and the diaphragm, these adhesions were converted into a thick, tough, yellow, homogeneous substance having the appearance of hyaline cartilage. The right lung weighed 960 Gm. and the left 830 Gm. The consistency of both lungs was much increased over the normal. The bases were particularly firm and tough, and dark brown. The upper half of the right upper lobe was excavated by a large multilocular cavity. The wall of this did not differ in density from that of the adjacent lung tissue, giving the impression that the cavity had formed so rapidly that a limiting fibrous wall was not thrown out. The cavity measured 6 cm. across, and through it coursed many thrombosed blood vessels. It had a foul, fetid odor as of gangrene. Throughout the remainder of this lung there were patchy areas elevated slightly above the cut surface, gray and red, moist, and measuring about 1 cm. in diameter. In the left lung, in addition to what has already been described for it, were two round nodules, similar in appearance, one in the upper portion of the lower lobe and one in the lower portion of the upper lobe on the outer aspect near the pleura. The one in the lower lobe was slightly larger, measuring 4 cm. in diameter. It cut with increased resistance and on section was composed of thick, dense strands of fibrous tissue, intermingled with gray, cheesy material. The regional lymph node draining this area was similar to this nodule. There were no cavities in this lung. In many fresh smears examined from these caseous areas, no tubercle bacilli were found.

Microscopically, the parietal pleura was studded with tubercles. These were composed of epithelioid cells, a few lymphocytes, giant cells and fibrous tissue in considerable amounts. Many were hyalinized to a considerable extent. Caseation necrosis was practically absent. They were of the productive type, with a few well formed tubercles.

The nodules in the left lung were large, caseous areas, divided up into lobules by strands of fibrous tissue. Sections from them and from the tubercles in the pleura were examined for tubercle bacilli, but none were found. In sections taken from the large cavity in the upper lobe of the right lung there were fresh liquefaction necrosis, very little polymorphonuclear and lymphocytic cell infiltration, little hyperemia and a thin, newly formed layer composed chiefly of young fibroblasts.

Fig. 1 (case 2).—A small bronchiole completely filled with cellular exudate and "asbestosis bodies." The fine segmentation and the clubbed end in some can be distinctly seen; × 406.

The principal lesion of the lungs was a diffuse fibrosis, particularly marked in the lower lobes. In many places, the alveolar walls were markedly thickened; in others, the alveoli had been completely replaced by fibrous tissue. Many bronchi were inflamed, and there were many areas of pneumonia present. A high percentage of the arteries were the seat of an obliterative endarteritis. The most striking feature in the lungs was the "asbestosis bodies." They were present in great numbers in the fibrosed areas, in some alveolar walls and spaces and in the

smaller bronchial branches. Particles of them were fragmented and were seen in phagocytic cells. Mainly, they lay loose in small clusters with collections of polymorphonuclear leukocytes and polyblasts about them, often completely occluding a small bronchiole or an alveolar duct. They measured roughly from 10 to 200 microns and were golden brown. They had a variety of appearances, but a characteristic morphology. They were seen often as a series of regular disks very much as are red blood cells in rouleaux formation. Frequently a series of disks ended in a club form on one or on both ends. Sometimes they were seen as long delicate filaments ending in clubbed ends; or again as short clubbed forms, single or double, suggesting a dumbbell. Occasionally, single, paired or coccal forms in chains were found, or even sporelike aggregations. They were present in the juice squeezed from the lungs. They did not stain with hematoxylin-eosin or Gram's stain. They did, however, give the prussian blue reaction for iron in varying degrees of intensity, in the main rather pronounced. In the caseous tuberculous areas in the left lung, these asbestosis bodies were fewer in number than in the adjacent lung tissue. A few were [illegible] many more pale with indistinct

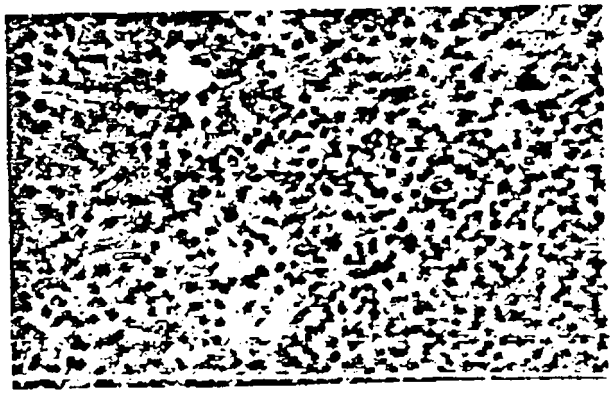

Fig. 2 (case 21).—Spleen. An asbestosis body is lying in the center of the field. One end is clubbed; the other pointed; × 400.

outline. When these caseous areas had been stained for prussian blue reaction following the staining of them for acid-fast bacteria (carbol fuchsin saturated solution 95 per cent alcohol, decolorization with 25 per cent nitric acid, Loeffler's methylene blue), the asbestosis bodies stained a pale green instead of a distinct blue, and their morphology was less regular, but they could still be recognized. In addition to the asbestosis bodies there was considerable golden-brown and black, granular pigment present in phagocytic cells. Much of this gave the iron reaction. In the spleen the reticulo-endothelial cells were loaded with a fine amorphous golden-brown and sometimes blackish dust. Some of this dust was extracellular. A few large, typical asbestosis bodies were found. Practically every hepatic cell contained a few granules of a golden-yellow pigment. Many of the Kupffer cells contained considerable quantities of light golden-yellow and blackish granules of pigment. About the portal radicles it was common to see a few cells loaded with pigment. In the mucosa of the stomach there were many phagocytic cells filled with fine, golden-brown pigment granules. In the kidney, many cells were seen in the capillaries of the glomeruli which were filled with coarse granules of a golden-brown and blackish pigment. The lymph node draining the caseous area in

the left lung contained a caseous tuberculous area similar to the one described in that lung, and in addition, many well healed tubercles. All bronchial nodes were deeply pigmented and fibrotic. The pigment was in the form of fine brown and black granules, mainly within phagocytic cells, and a great deal of it stained for iron. A few asbestosis bodies were present.

COMMENT

The two cases cited illustrate nicely the extremes and some of the features of the disease. In the first case, as far as the patient's health and life were concerned, the presence of pulmonary asbestosis in the lung was relatively unimportant. He had symptoms doubtfully attributable to it. It was only through the history, which was confirmed by postmortem examination, that the possibility of such a condition was suggested. It is, however, important to point out two considerations in this case. First, it is a question whether the asbestosis had anything to do with his emphysema and cough. As an etiologic factor, the asbestosis cannot be ignored. The other point is that one has to do with preventive medicine. The exposure was for nine years in a comparatively well ventilated factory in which precautionary appliances were used, and yet the patient developed a minor grade of the disease. It would seem that further precautionary means are necessary in this occupation to obviate a disease in itself so preventable.

In the second case, the patient worked in an asbestos factory for a short time, but without a mask. This was long enough to permit his lungs to become densely filled with the dust. Obviously the symptoms did not result from the asbestos per se, but were due to the marked fibrosis resulting from the deposition of the pigment. This demonstrates that the symptoms of a pneumokoniosis may come on years after a short exposure and emphasizes again the absolute necessity for wearing masks in even the less dusty atmospheres.

The marked fibrosis of the pleura, particularly of that interposed between the base of the lung and the diaphragm, is characteristic. The finding of rapidly liquefying areas in the lung is not unusual either, but the presence of such an extensive freshly excavated area is.

We have been able to demonstrate two large tuberculous lesions in the midlung just beneath the pleura, tuberculosis of the regional lymph nodes and tuberculosis of the parietal pleura. The analogy between this form of lesion and that of the tuberculosis of childhood is, to say the least, striking. Gardner and Cummings have shown the course of asbestos inhalation with coincident tuberculous inoculation. [illegible] ner, in a personal communication to us, replied in the negative to our question as to whether or not any experimental work had been done on the influence of asbestos inhalation on a healed primary focus. It seems to us this would throw light on the subject, for it does not appear that the asbestosis and the tuberculosis were coincidental lesions.

Our second case confirms previous observations that the asbestosis bodies have a tendency to disappear in a caseous area. We have also shown that in such an area following an acid-fast stain (25 per cent nitric acid) the bodies do not stain so well for iron and have a less definite morphology, but are still recognizable as such, so that if these are present in the sputum in any case, they should be recognized on a routine smear even after it has been treated with acid.

The finding of tubercle bacilli in the sputum in the second case on two different occasions, plus the gross and microscopic features characteristic of tuberculosis, is sufficient to establish such a diagnosis. It is significant, however, that many more specimens of sputums were examined for acid-fast bacilli in which none were found, and that no one noted the asbestosis bodies in the sputum, although these were not being looked for specifically.

An important additional finding in case 2 was that of asbestosis bodies in the spleen. Although we have diligently searched the literature, mention of such a finding, to our knowledge, never previously has been made. Whether these bodies get there by the blood stream as emboli or whether they are carried there by phagocytic cells, we are not prepared to say and have no evidence on which to base a conclusion. The fact that they are in that organ, irrespective of how they got there, seems to be significant.

It is interesting that neither in case 1 nor in case 2 (despite the marked pulmonary fibrosis and the thickening of the smaller pulmonary arteries) was there any dilatation or hypertrophy of the heart on either side.

SUMMARY

Two cases of pulmonary asbestosis are reported. In the first case, the symptoms were relatively unimportant. In the second case, the disease was concomitant with tuberculosis and, with it, was the cause of death. The similarity between some of the features of tuberculosis in childhood and tuberculosis complicated by asbestosis is suggested.

In asbestosis the lung is not the only organ invaded by asbestosis bodies. As we have shown, the spleen and the lymph nodes may harbor large asbestosis bodies.

From the histories there appears to be a further need for study along the lines of prevention of this disease.