ATTORNEYS' TEXTBOOK OF MEDICINE

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Chapter 51

ASBESTOSIS

Asbestos particles inhaled into the lung produce an exceedingly severe and perhaps fatal inflammation. This condition, called asbestosis, is not so important as many other forms of mineral irritation of lung tissue, because of its infrequency. However, it will become more prevalent as the industry grows. Care must be exercised in the event of claim, that the disability is properly chargeable under the policy, that the cost should not be pro-rated with previous carriers, and that the diagnosis is correct. Since asbestosis is incurable, and usually results in total permanent disability followed by death, care and caution should be used before a claim is assumed.

Asbestos Exposure in Industry

Asbestos is rapidly coming into use for a great variety of articles, particularly due to its heat, chemical, and electrical resisting qualities. A list of these will indicate that exposure may appear in many industries: cloth for theatre curtains or scenery, and for suits, aprons, gloves, mittens, leggings, and helmets; paper for filtering strong acids or bases, and for linings between floors or on pipes; as a thick paper for linings of stoves, furnaces, heaters, filing cabinets, automobile mufflers, and electric switch boxes; as a cloth covered with rubber cement for gaskets; woven with metal for break linings or clutch facings, and braided or woven tape; for non-inflammable rope; as millboard and shingles; and mixed with Portland cement as fireproof shingles and building material.

Fortunately the manufacture of many of these articles is

not accompanied with a great deal of asbestos dust, which is the only form in which this substance is dangerous. After refinement and the pressing or weaving into cloth, tape, rope, paper, or sheets, there is usually too little dust liberated to do much harm. We may, from a practical point of view, eliminate all plants as potential hazards in the production of asbestosis except quarrying, cleaning, spinning, weaving, and pressing of this mineral.

Although asbestos has been used since antiquity, asbestosis is a distinctly modern disease, the first known case having occurred in 1899, and only two were reported previous to 1928. How does it happen that apparently the disease did not occur before? Just as with silicosis, considerable of the mineral must be inhaled, and it was not until the advent of machinery that sufficient dust was produced. Particularly in the carding of the fibers, preparatory to making thread for weaving cloth, tape, and rope, enormous quantities of dust fly about. Moreover, in all other stages of manufacture by machinery until the fibers are imprisoned in some permanent form, the hazard is exceedingly great, unless the highest type of exhaust ventilation is employed. The hand processes in use until recent years were too leisurely to produce many particles suspended in air.

Composition of Asbestos

Asbestos is not a single chemical entity. The name is given to minerals that can be easily separated into flexible fibers. They are all silicates, compounds of metals, silicon, and oxygen, occurring principally as hydrous magnesium or calcium silicate. Specimens from different parts of the world vary widely in formula and in physical as well as chemical qualities, giving rise to many different names. It is not necessary to know them apart, since all are apparently quite poisonous if inhaled in sufficient quantity. The names of the common ones are given that you may recognize them as asbestos, and not be led astray, thinking them to be something else.

The Canadian mines supply the bulk of the world's supply under the name chrysotile, or serpentine. Chrysotile has a particularly long, strong fiber easy to spin, but of little use as a resistant to acids. The Russian and South African amphi-

bole, or hornblend, is poor for spinning, but makes an excellent acid resisting filter paper. Crocidolite and amosite are asbestos found in South Africa containing much iron silicate. Tremolite from Italy is largely magnesium silicate. Many local names have been given, as mountain flax when the fibers are particularly silky and flexible. When closely interwoven, it is sometimes called mountain leather, mountain cork, or mountain paper. That with very fine flexible fibers has been called earth flax, flexible asbestos, amiantus, or amianthus. There are many trade names as uralite, salamandrite, asbestolith, and gypsine. They are all asbestos.

Processing of Asbestos

Asbestos is usually quarried although it may be tunnel mined, especially in Arizona. After dislodging by blasting with dynamite, the best, long fibered pieces are separated with small hammers by hand. This process is called cobbing. If too wet the ore is dried. Then it is crushed by rock breakers, and finally divided between rollers. Fiberizers tear the fibers apart. On large screens, it is automatically shaken, with separation of sand or dirt, and graded by rotary screening. Then it is bagged, and ready for manufacture into the many, final products.

The process of cloth making is very similar to that followed with cotton, necessitating carding, spinning, and weaving. The manufacture of paper, thick sheets, and fireproof board is preceded by sorting and so called "opening up," wherein the fibers are pulled apart.

Dust

All of these processes are accompanied by a tremendous volume of very fine dust, until such time as the tiny, loose particles have become separated, and the fibers trapped as in thread, paper, or board. Then, handling is relatively harmless. The greatest dust hazard occurs in the carding rooms, although considerable danger is present until the fibers have become bound in some permanent form.

A microscopic study of asbestos and of the dust is necessary to a clear understanding of the lung changes occurring in this disease. The bulk of asbestos consists of translucent, glisten-

ing fibers. A relatively small quantity of dark, opaque, angular particles are found scattered here and there. Tiny black granules are seen as integral portions of the fibers. The dust contains not as many fibers as would be expected, but a relatively small quantity, while the particles and granules are present in great profusion. The assumption is clear that the dust consists mostly of particles that have been mechanically loosened from the fibers, and that but little of the latter are found, since most of them are too large to fly freely about, or have remained stuck to the remainder of the asbestos.

We shall find that the fibers and the black material play two different parts in the production of asbestosis. It is therefore necessary that we know their chemical composition. The fibers are silicates whose formula depends upon the particular variety of asbestos present. The dark material is very rich in iron oxide and carbon.

Action of Dust on Lung

Fibers may be inhaled in such tremendous quantities that actual plugging of bronchioles occurs. Examination of the lung will disclose asbestos fragments of all sizes, from very thin spicules 1 micron (1/25,000 inch) in length to others extending 360 microns (over 1/10 inch). They are the exact counterpart of those found in asbestos dust. When a bronchiole is plugged, no longer permitting the passage of air, that part of the lung cannot function. It dies, and either becomes abscessed, or replaced by scar tissue.

Fortunately, the majority of fibers are too small to prevent passage of air, or are not inhaled in sufficient quantity. However, unlike most foreign bodies that enter the lung, they cannot be swept out by the lashing motion of the hair-like cilia on the mucous membrane, whose function is to remove dust from the lung. Asbestos is too irregular, and the fragments usually become lodged, to remain within the lung, only to begin the slow, insidious poisoning that will continue for many years.

It is not entirely clear what occurs, but apparently the following complicated steps give the best accepted explanation of the changes found in the lung, so far as the asbestos fiber is concerned:

1. The asbestos spicule or fiber lodges in a bronchiole or an alveolus. (Dilated structure at end of bronchiole where oxygen absorption occurs.)
2. Minute extravasation of blood or serum envelops the spicule.
3. The minute soluble portion of the asbestos dissolves in the serum and mucus.
4. The surface of the fiber adsorbs, or holds thereto the colloidal solution of asbestos and serum, or mucus.
5. Chemical reaction occurs between the soluble portion of asbestos and tissue fluids with loss of water, so a permanent union results, coating the fiber.
6. This jelly-like envelope becomes molded by the alveolar or bronchial currents in the normal flow of mucus in the lung.
7. From time to time these "curious bodies" become loosened and carried out of the lung by the action of the cilia. Then they appear in the sputum.

These "curious bodies" gain their name from the variety of shapes they may assume, and the fact that their nature was not at all understood for some time. A better name, of late suggested, is "abestosis bodies." Their presence in any considerable number, either in the sputum or the lung, is proof positive of asbestos exposure, since they have never been found due to any other condition. It is not necessary to go into an elaborate description of the very complicated steps needed to prove the identity of alleged "asbestosis" or "curious bodies." It requires the most expert laboratory work to complete this cardinal proof of diagnosis. Since it is but rarely that a docter can be found who has seen them, you should consult a very able pathologist, and ask him to make the necessary tests, in accordance with technique such as outlined by Merewether under the title: "The Occurrence of Pulmonary Fibrosis and Other Pulmonary Affections in Asbestos Workers," reported in the Journal of Industrial Hygiene for June 1930 on Page 239 (6).

It will be recalled that asbestos dust not only contains fibers, but that many particles are also present. They are mostly either of carbon or various compounds of iron, magnesium, or

are silicates, as mica. These particles may be seen interspersed throughout the asbestos ore, and have become dislodged when the fibers were torn apart. Thus, finished asbestos products contain but few, while the dust, especially of the carding rooms, is loaded with them.

As would be expected, the lung of an old asbestos worker contains many "particles," inhaled through the years. Since they are practically insoluble, they remain in the lung if small enough to enter the alveoli, or are not swept out by the cilia of the mucous membrane. They do not remain at the spot where they lodged, but are seized upon by the "dust cells" of the lung and the phagocytes, or white cells that act as the scavengers of the body. These ingest the particles, dissolve out any digestible material, and carry the remainder away.

Similarly, the tiny asbestos fibers, undergoing chemical change, are carried off in nature's effort to rid the body of their poisonous presence. The "dust cells" and phagocytes do not always succeed in their task of elimination. The journey from the alveoli and bronchioles to the lymphatic glands is but slowly made. Many of the "scavenger cells" die on the way, leaving the ingested "particles" and "curious bodies" behind. This is particularly true where the lymphatic channels which they follow join one another. Then plugging may occur at these points, due to the great quantity of insoluble and poisonous material being carried along. So, masses of "particles" and "curious bodies" will be found here and there at the junction of lymphatics and in the various lymphatic glands of the lung.

While the "particles" do not undergo very important chemical changes, leading to the death of many cells, the asbestos fibers seriously disturb the life processes of surrounding tissue. Local inflammation and death result. Areas of lung are destroyed and healing occurs through replacement with fibrous cells, similar to scar tissue. The fibrosis of asbestosis ensues.

Morbid Anatomy

A description of the lung changes will clearly explain the symptoms of this disease. A complete and masterly discus-

sion of this subject has been given by E. R. A. Merewether (5) in the Journal of Industrial Hygiene for June 1930.

Briefly, the changes are divided into two stages, tissue destruction and reparative processes. The poisonous action of the asbestos leads to a destruction of mucous membrane in the bronchioles and alveoli, as well as of the lymphatic channels through which the "scavenger cells" attempt to carry the dust away to the lymphatic glands. Thus a chronic bronchitis is set up. If bronchioles become mechanically plugged, necessitating increased air supply to uninvolved portions of the lung, many dilate, producing a bronchiectasis, or dilatation of bronchi. Complete stoppage of air channels leads to death of that part of the lung which no longer functions. Sufficient air cannot be absorbed, so breathlessness on exertion ensues.

X-Ray Findings

Nature tries to overcome the difficulty by replacing destroyed structures with fibrous tissue. The degree of fibrosis will, of course, depend upon how far advanced the changes are, but the most characteristic x-ray finding is the so-called "ground-glass" appearance, wherein the entire picture has a hazy, indefinite, fogged effect, due to the presence of fibrosis scattered throughout the lung. Close examination shows that the haziness is not entirely vague, but consists of fine lines during the early stages, with tiny dots appearing later, as masses of dust are deposited at the junction of lymphatics. The dots become larger, with a coarse mottling of the picture. Finally, large masses of scar bind the layers of the pleura together, and adhesions drag various parts of the lung away from their normal positions.

The x-ray findings, as a general rule, are more pronounced in the lower third of the lungs during the early stages of the disease. Later, they are manifest everywhere.

Symptoms

As may be expected, the symptoms are very largely due to the destruction of lung tissue and to the bronchitis. Dyspnea, or shortness of breath on exertion is a cardinal symptom in

52% of cases. Cough, due to bronchitis, occurs in 59%. Because of insufficient lung tissue to properly aerate the blood, cyanosis, or a blueness of the skin is apparent in 56% of patients. Bronchitis accounts for marked raising of phlegm with 34%. Adhesions of the pleura produce pain in 11% of cases.

Shull (8) presents an interesting study of seventy-one cases showing certain progressive changes that occur during the course of the disease. He divided his patients in accordance with the degree of lung fibrosis shown on x-ray, into slightly advanced 23%, moderate 49%, and far advanced 28%.

Shull (8) found hypertrophy of the right heart in 37.5% of his early cases of asbestosis. This condition was evident in 62.8% of the moderately advanced. In those with marked lung fibrosis, 95% had developed enlargement of the right heart. Thus it may be clearly concluded that asbestosis seriously embarrasses pulmonary circulation, contributing to the most common subjective symptoms, shortness of breath and cyanosis, blueness of the skin.

Hypertrophy of the right side of the heart is to be expected as Lanza (9) explains, because asbestosis leads to a constriction of the tiny pulmonary arterioles in their course along the bronchioles. The increasing fibrosis involving the smaller air passages inevitably includes thickening of the blood vessel walls, impeding blood circulation, demanding enlargement of the right side of the heart, responsible for pulmonary blood pressure.

Thickening of the pleura and pericardium progress with advancing asbestosis, as disclosed by Shull's series. In those classified as early, noticeable thickening had not occurred in a single patient, while 28.6% of the moderate cases showed these involvements, and 45% of those with advanced lung fibrosis also had thickening of the pleura and pericardium.

The left side of the diaphragm was distinctly higher than the right in only 12.5% of the early cases, in 48.6% of the later stage patients, while 80% of the advanced cases disclosed this finding.

Difficulty with breathing tends to a compensatory enlargement of the entire chest cage, the emphysematous chest. In Shull's early cases, the finding was infrequent, only appearing

in 6.3% of his patients, 65.7% of those classified as moderate presented this finding, while 95% of those with marked lung fibrosis had emphysematous chests.

Diagnosis

This disease occurs rarely in the experience of most doctors, and they usually received no teaching regarding probable findings. Thus they cannot be expected to recognize a case without special study. The similarity to silicosis leads to mistake unless great care is used in the interpretation of x-rays. Even the pictures may be so similar that the history of exposure to asbestos or silicon dioxide must be carefully considered.

Frequently the findings and x-ray studies indicate a fibrous tuberculosis, which may also be independently present. Bronchitis in an asbestos worker does not necessarily indicate asbestosis, nor does bronchiectasis, although both are very suggestive. Cyanosis and breathlessness may be entirely due to some other condition, as heart disease or asthma.

It is clear that the most expert advice is necessary to a true determination, which must usually depend upon a careful study of excellent x-rays, a search for "asbestosis bodies" in the sputum, a study of the asbestos and other dust exposures over all the patient's life, and a careful elimination of other possible diseases as causative factors in the production of symptoms.

Treatment

This is a very serious disease and is practically incurable. Particles once ingested continue their slow, insidious, tissue destruction through years, even though exposure may long have terminated. We know of no way to stop the action or to bring about the elimination of asbestos fibers. While those too large to be carried toward the lymphatic glands may become loosened in the bronchioles from time to time and eliminated in the sputum, appearing as "asbestosis bodies," the smaller ones are usually in the lung to stay.

Of course, the bronchitis may be alleviated by usual treatment directed toward this disease, but it will continue. The patient's general strength should be maintained by study of his hygiene. Usually, death is not due to the asbestosis, but

to some other disease particularly of the lung. Therefore, pneumonia must be guarded against. The shortness of breath on exertion can only be met by avoiding heavy work. In time, the patient probably will become a total permanent disability. Death usually occurs within a year after the patient can no longer work.

Complications

It is sometimes alleged that tuberculosis is likely to result from this disease, but Merewether (6) points out that only 9.9% of patients with asbestosis show signs of tuberculosis, and concludes from his studies that there is no great susceptibility to pulmonary tuberculosis among workers in asbestos, even though they already had fibrosis, since about the same amount of the disease occurred in the general population.

Bronchitis and bronchiectasis are almost inevitable.

Duration of Exposure

The discussion relative to symptoms, treatment, and likelihood of total permanent disability is based upon the assumption that definite, disabling asbestosis has occurred. If the exposure is slight, as in a well guarded plant with good exhaust ventilation, insufficient asbestos will be absorbed to destroy a vital amount of lung. Again, exposure may not continue over enough years to have brought about the inhalation of sufficient dust. Usually the slow beginning symptoms of bronchitis or shortness of breath cause the employee to change his work to a less dusty or laborious occupation.

The average exposure before the appearance of the disease is 13.5 years. It is very rarely found with less than 5 years labor with this dust, rapidly increases after this duration, and beyond the tenth year, the likelihood of asbestosis is exceedingly great.

The danger is proportionate to the dust count. If this is low enough, elimination practically equals the rate of inhalation. Relatively little dust occurs in those processes after spinning, because the "particles" have become freed, and the fibers have been trapped into a permanent position. Since asbestosis practically does not occur with spinning, unless there has been previous exposure to more dusty processes, it

is considered that the dust concentration of the spinning room is safe. Merewether (6) found the dust count in this department to be as low as 506 to the cubic centimeter, equal to about 13,500,000 to the cubic foot. In the dusty departments, the count ran as high as 170,748,000 to the cubic foot.

Investigation

Investigation must be thoroughly conducted along three lines; duration and intensity of exposure, and the true cause of the disability.

Even though the patient may have asbestosis, and has worked for some time with considerable exposure, this is not sufficient grounds to charge the disability to the present employment alone. The average period of exposure is 13.5 years. Very few men have continued to work this long for a single assured, and but rarely is the same carrier on the risk throughout the entire exposure the patient has had. Obviously the disease was caused by all of the asbestos inhaled, not the fibers respired during the past few months or years alone. Therefore, the disability should be charged pro-rata to all carriers during the entire time of asbestos exposure.

In fact, to be scientifically accurate, probably little or none of the asbestos inhaled within many months has had time to go through the chemical processes necessary to destroy tissue, and thus is not a causative factor. This fact probably would not convince a jury that the disease should be charged only to carriers in past years, but it is true that all of the exposure the patient has had throughout his life contributed pro-rata to the disability in direct proportion to time and concentration of asbestos dust.

The intensity of exposure is important, even in the proven case of this disease. Unless the dust count exceeds that usually present in the spinning room, of about 13,500,000 to the cubic foot, the condition is probably due to previous exposure alone. The protective mechanism of the body is able to handle this amount indefinitely.

The true cause of disability must be determined. This necessitates expert study of x-rays, search of the sputum for "asbestosis bodies", and definite conclusion that other disease is not the cause. Unless the experts have had experience

with this disease, the attorney should ask them to read this chapter, since their text-books will be of little avail, and to particularly study the articles of Merewether listed as (5) and (6) in the *Bibliography*. It is not surprising that doctors have had but little experience when it is recalled that only two cases were reported up to 1928.

Risk Control

IN THE CONTROL OF A RISK, THE FOLLOWING POINTS SHOULD BE CONSIDERED:

1. In what departments do asbestos dust counts exceed the threshold limit of 13,500,000 per cubic foot during any considerable period of the day?
2. May the concentration be reduced in such departments through mechanical means, or the number of exposed workers reduced through partitions?
3. Respirators must be worn when exposed to concentrations exceeding 13,500,000 per cubic foot.
4. The use of respirators must be enforced by monitors with police power, and discharge as the penalty for disobedience.
5. Physical examination before employment should be required. Workers should not be exposed to asbestos if they show positive evidence of chest disease.

IN THE STUDY OF AN INDIVIDUAL CLAIM, THE FOLLOWING POINTS SHOULD BE CONSIDERED:

1. All records of physical examinations should be carefully scrutinized by an expert trained in the detection of asbestosis and of other chest diseases.
2. Determination should be made relative to whether the entire disability is due to asbestosis or other disease a direct outcome thereof, and what part of the disability is due to conditions not caused or aggravated by asbestos inhalation.
3. A careful history should be taken covering all exposure to asbestos throughout life, unless claim is brought under a compensation act which does not permit prorating between present and past employers.
4. The history should endeavor to determine the approximate lengths of exposure at various concentrations, and a rough estimation of the asbestos dust counts for each should be made.

5. **The degree of exposure for the past and present employments should then be compared.**
6. **Has work with the present employer appreciably exceeded asbestos inhalation of 13,500,000 particles per cubic foot?**
7. **Is it certain that diagnosis has not been made largely on x-ray findings and employment with asbestos, without making certain that some other disease as bronchitis or silicosis may not be the real causative factor?**

Obviously these studies necessitate the highest class technical experts for determination. Since each case is a probable fatality, any reasonable expense is warranted. Only in this way can the attorney avoid a determination based on history of exposure or sheer guess work, and without properly charging disability to all carriers entering into the exposure. Justice must be done to both the patient and the Company.

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