CHAPTER XXX

ASBESTOSIS

Asbestosis is a comparatively new disease. The first comprehensive reports regarding asbestosis came from the English writers in the years between 1920 and 1925. In America in the early thirties studies were made by Lanza, Sayers, Bloomfield, and others.

Asbestos is a hydrated magnesium silicate. Occupational exposure occurs in those trades where it is used for packing, insulating, or fireproofing, or where it is combined with cotton or other material in textile processes.

Signs and Symptoms. As in silicosis,[1] the first sign is dyspnea, the cough is usually dry, and auscultation reveals few, if any, physical signs. Loss of weight is usually noticeable, the color is pale, and cyanosis is a fairly early sign. After some time, fine, crackling râles may be heard, and the chest appears emaciated and lacks the robust character noticed in silicosis. Early asbestosis must be differentiated from acute emphysema. If tuberculosis is present, the symptoms may take on the character of this disease, but this complication is far less frequent than in silicosis.

Pathology.—

PULMONARY CHANGES. The pulmonary fibrosis of asbestosis is diffuse in character, peribronchial, and basal in location in contrast to silicosis in which the fibrosis is nodular in character and present in the upper part of the lungs. As the process continues, the fibrosis extends into almost all portions of the lung tissue. Bronchiolectasis and bronchiectasis are present within the substance of the fibroid areas, and bronchopneumonia and acute tracheobronchitis often cause the death of these patients. The pleurae, especially in the basal regions, are thickened and adherent. In some cases the pleural sac is completely obliterated; in others a fibrous or serous exudate may be present within it.

Action of Asbestos and Silica. The difference between chrysotile asbestos and quartz silica in their mode of action has caused considerable speculation as well as experimentation. Gardner[2] particularly, at the Saranac Laboratory, carried on intensive study of these two materials. He felt that while the action of free silica is chemical in nature, the action of asbestos is mechanical. He stated:

"We have come to the conclusion that inhaled asbestos fibres are irritating not because they are silicates but because they are stiff fibres which mechanically irritate the lungs. Unlike the free silicas, these minerals will not stimulate fibroblasts in any part of the body; only those in the lungs are affected. It was inferred that these organs were

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affected because the movements of respiration are so much more rapid and continuous than those of other viscera. Then it was discovered that if asbestos was ground very finely so that few of the fibres were longer that 2 microns in length the irritating property of the asbestos was practically destroyed. Inhalation experiments with such fine chrysotile asbestos were carried out over a period of several years. No fibrosis has developed in spite of the fact that an average atmospheric concentration of 125 million particles per cubic feet of air has been maintained. In contrast in a previously reported experiment one-third this concentration of long fibre asbestos dust produced well marked fibrosis after about two years."

If the effect of asbestos were chemical, one would expect that a decrease in size would accelerate tissue response. With free silica large particles have little effect, but as their size decreases cellular reaction becomes more vigorous and even constitutional symptoms may ensue. With fibrous asbestos the reverse is true.

The histology of early asbestosis does not suggest a chemical injury. Even under the most extreme conditions that can be created by artificial injection there is no preliminary phase of tissue necrosis with infiltration of leucocytes as occurs with high concentrations of very fine quartz. The connective tissue cells merely multiply very slowly in areas where the asbestos fibres are caught in the bronchioles. As collagen forms and contracts, the air spaces are obliterated by scar tissue. In experimental animals, at least, this change is not a progressive one after cessation of exposure to the dust as is the case in the response to quartz. Perhaps the reason is the deposition of the peculiar iron-containing coating on the surface of the inhaled fibres giving rise to the characteristic "asbestosis bodies" (Figs. 50 and 51).

Finally, it is most suggestive that among dozens of different silicate minerals only the five known as asbestos, which are unique because they are fibrous in structure, should be commonly recognized as pulmonary irritants. The variation in chemical composition within this group is greater than that between them and many other silicates. In fact, chrysotile asbestos has the same chemical formula as a nonfibrous silicate, serpentine, which is physiologically inert. Obviously irritation would seem to be associated with the physical rather than the chemical composition of these minerals.

One point of practical significance may be indicated by these observations; namely, that very finely ground asbestos is not dangerous. This conclusion has support in clinical observation, for it has long been known that at the Thetford Mills there was no clinical asbestosis even though in former years the atmosphere was very dusty and the dust was extremely fine. The fact that fabrication of fibres of the same mineral in American plants could produce disease was one of the puzzling fea-



Fig. 50.—Right lung, showing markedly thickened pleura with fusion of interlobar pleura. Dense fibrosis fogs over entire surface. (U. S. Public Health Service, Bulletin 241.)

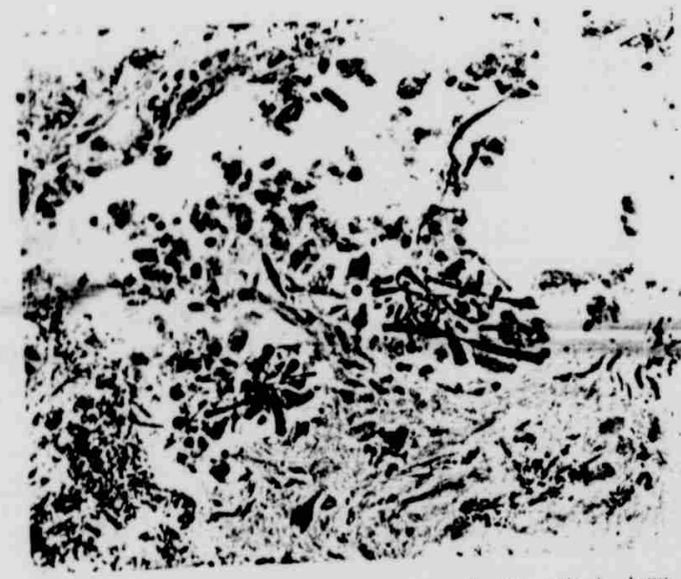


Fig. 51.—Asbestosis bodies accompanied by giant cells. (U. S. Army Medical Museum, Specimen 5225.)



Fig. 52.—Section of a lung in asbestosis. (Gardner.)



Fig. 53.—X-ray film of a patient with asbestosis. (Vonbergmann.)

tures of this disease. But these experiments offer a plausible explanation. The fine dust in the mills is composed of serpentine and extremely short chrysotile fibres; that in the spinning and weaving mills contains many more long fibres.

Microscopic Appearance. In the early phases of the disease there is a thickening of the alveolar septa as a result of fibroblastic proliferation. The alveolar spaces contain numerous alveolar phagocytes. With progression of the disease, fibrosis becomes more marked and the alveolar structure gradually disappears, and in its place there is now dense fibrous tissue. The few remaining alveoli which lie in the area of fibrous tissue are lined by a low cuboidal epithelium and have a glandular appearance (Fig. 52).

Scattered throughout the lung in both the diseased and healthy parts are spindle-shaped structures (first described by McDonald*) known as asbestosis bodies. These bodies are slender in their center and bulbous at each extremity. Many are arranged like strings of graduated beads with the largest at the end of the chain.

X-Ray Findings. Certain peculiarities exist here as contrasted to silicosis. In asbestosis, the lesions may be bilateral or largely unilateral. According to the experience of Pendergrass,[3] the roentgenologic findings in asbestosis are largely limited to the lower half or two-thirds of the lung fields, while in silicosis the upper portions of the lungs are also involved. In moderately advanced asbestosis, x-rays will reveal lessened ventilation of the lung fields, the parietal pleura is thickened, and there is a "ground-glass" appearance to the picture. The vascular shadows lose their identity. Nodulation is absent. Pendergrass[3] feels that a roentgenologic diagnosis of early asbestosis is unreliable and that the condition must be moderately or markedly advanced to make a differential diagnosis from the roentgenograms (Fig. 53).

Medicolegal Aspects. It is felt that the time necessary to develop asbestosis is, on the average, from seven to nine years in a fairly high concentration; with a less severe concentration, from fifteen to twenty years. The allowable concentration is 10,000,000 particles per cubic foot of air, of a size between 0.5 and 5.

Once established, the disease is progressive, even after the cessation of exposure. The basis for diagnosis is similar to that given under Silicosis.

References

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