ASBESTOSIS

by

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Asbestosis is a pneumoconiosis caused by the inhalation of asbestos dust. It is distinct from silicosis in its pathology and clinically. Whether asbestosis will remain a distinct form of pulmonary dust disease or will prove to be of a type common to a number of dusts remains to be seen. So far, it is the only pneumoconiosis, other than silicosis, which has received any considerable amount of study from pathologists, clinicians, and industrial hygienists.

Whereas silicosis has been recognized for many centuries, asbestosis is a newcomer. Asbestos (Canadian) is a hydrated magnesium silicate containing no free silica, but about 44% of combined silica, 43% magnesium, nearly 13% of water, and traces of iron and nickel. While asbestos was known to the ancients, the fabricating of asbestos on a large scale is comparitively new and received a tremendous impetus from its use in connection with automobiles and as an insulating material and a heat resistant for a great variety of mechanical purposes.

While Hoffman, in his celebrated Bulletin on Mortality from Respiratory Diseases in Dusty Trades,(1) called attention to the possible harmfulness of asbestos dust in 1918, it was not until February, 1927 that asbestosis was, so to speak, officially recognized in this country by the filing of a disability claim for workman's compensation in Massachusetts. The claimant was a foreman in the weaving department of an asbestos plant and the claim was upheld by the Massachusetts Industrial Accident Board. This was twenty-seven years after the first fatal case was reported in England by Dr. Montague Murray. In 1910 and again in 1924, a fatal case was reported in England and in 1928, the British Factory

Department conducted an investigation and enacted laws for the protection and compensation of the worker against this hazard. The whole subject in England has been very well summarized by Merewether in a series of articles in Tubercle.(2) At the present time, as nearly as can be estimated, there are about 12,000 individuals employed in the chief asbestos plants in the United States of whom, 10,000 might be exposed to asbestos dust.

In 1927, a fatal case of uncomplicated asbestosis was reported to the Medical Society of South Carolina(3) and since then, including this case, there have been eleven fatal cases reported in the United States, eight uncomplicated and three complicated by tuberculosis. These reports, together with the fact that asbestosis figured in the extraordinary occupational disease litigation that has spread over this country, resulted in both laboratory and field studies of this new hazard. Gardner and Cummings at Saranac Lake commenced animal experimentation with asbestos in 1928 and reported their findings in 1931.(4) These two authorities in the United States and Gloyne in England(5) have produced masterly descriptions of the pathology. While silicosis is predominantly parenchymatous, asbestosis is mainly interstitial, nor is asbestosis characterized by the nodular formation so distinctive of silicosis.

A search of all the death records on file in the Metropolitan Life Insurance Company revealed that asbestosis had been given as a cause or contributing cause of death in only nineteen cases. The first case noted was in 1924, two in 1927, one in 1931, and the rest since 1933. The diagnosis was supported by autopsy in only six. In one of these six, the primary cause of death was carcinoma; in another, glioma of the brain; and in another, pulmonary tuberculosis. In the other three, asbestosis was given as the primary cause with cardiac failure as the contributing cause. Some of these cases were reported in the literature.

Asbestosis complicated by heart disease was given as the cause of death in seven cases. There were no autopsies in these seven and pulmonary tuberculosis was diagnosed in several by the attending physician, so perhaps too much weight cannot be given to these certificates.

Diabetes and pulmonary tuberculosis were also mentioned in the remaining six cases, in which there were no autopsies. All these nineteen cases were males and all were white with one exception.

In our studies of asbestos mines and fabricating plants, (6) the clinical picture of asbestosis was milder than that of silicosis. To be sure, the individual patient with marked asbestosis will greatly resemble the individual with silicosis. There is the same dyspnea on exertion, the same dry cough, the more or less indefinite physical signs elicited by the stethescope. The patient with asbestosis is apt to have clubbed fingers - not usually seen in silicosis - and he is apt to be pasty faced and even show a slight cyanosis, while the silicotic is apt to be fairly robust looking. Of course, in each instance I refer to patients whose disease is not complicated by infection.

We did not find in communities where asbestos was mined or fabricated the familiar picture of disability and tuberculous infection so characteristic of hard rock mining communities. Our personal observations were supported by the statements of physicians practicing in these communities. All the cases of asbestosis we detected were, with one exception, working steadily at their trades. In only one did we find evidence of active tuberculosis and that diagnosis was based on X-ray appearance only, and showed healed tuberculosis. Gardner and Cummings in their reports called attention to the difference between the action of asbestos dust and silica dust in relation to tubercle infection in experimental animals and their findings tend to bear out our clinical observation.

In all, our clinical data are based on 126 physical examinations of asbestos workers, all of whom had more than three years' exposure and who were selected at random. 63 of these showed an X-ray appearance which we thought indicated a pneumoconiosis but their symptoms were indefinite and inconclusive. We called these cases first stage. 4, with evident pulmonary symptoms and corrobative X-ray, we termed second stage. Of these 67, 29 had over ten years' exposure, and 15 had over 15 years. We are still continuing our asbestos studies and hope to secure additional information on the time element in development and the tendency and rate of progress.

One feature that has impressed us is that the British investigators found asbestosis more severe and more menacing than we did. This difference may be more apparent than real but it is possible that the English factories may be more dusty than ours. There were not available any comparative dust counts but this impression is based on their reports and statements made to myself and colleagues by persons familiar with the English conditions. One process, described in the British reports as "mattress making" and stated to be extremely dusty, does not appear to have a counterpart in this country. In both countries, energetic steps have been taken to control the dust hazard in asbestos plants so that it is probable that further cases of disabling asbestosis will be rare.

As in silicosis, the diagnosis centers upon the X-ray film. However, the whole matter of attempting to interpret these films and correlate them with the clinical evidence, if any, is difficult and elusive. If, in our studies, we had found a more clear cut and severe type of pneumoconiosis

with marked symptoms and disability as well as a distinctive X-ray appearance, such as my colleagues and myself had been accustomed to find in our previous investigations of silicosis, our task in attempting to make a positive diagnosis and estimate the extent of the disease would have been easier. There is no doubt that, especially in the beginning, we were handicapped by endeavoring to evaluate asbestosis with a silicosis foot-rule.

The X-ray appearances are not clear cut nor distinctive as in silicosis and do not lend themselves to ready grouping into progressive stages. There is less evident pathology in these films, the shadows are finer, more granular, and softer, than in silicosis. The asbestosis film gives the impression of ground glass and there is no nodulation with the consequent tendency of the nodules to coalesce and give dense opaque areas in the films. The distribution of the shadows is somewhat different, occupying the lower third of the lung, except in far advanced cases when the shadows may occupy the major portion of the lung. We noticed frequently the well marked outline of the interlobar septum on the right side. We also noticed that a number of the films of cases of asbestosis had enlarged hearts, and this might be expected when one considers that the pathological process tends to constrict the pulmonary blood vessels and they ramify along with the bronchioles. (7)

It is possible that the X-ray appearance of asbestosis may not be distinctive of this disease alone but uniform in appearance with pneumoconiosis due to other silicate dusts. Much more investigation and study of X-ray films of industrial workers exposed to all sorts of silicate and other dusts are needed before it will be possible to speak definitely on this, the most important phase of the diagnosis of pneumoconiosis.

One thing is certain. The utmost care and patience are needed to elicit the occupational history of the patient and to correlate this and the clinical picture with the X-ray film before a diagnosis of asbestosis is justified. As noted in relation to other occupational health hazards, there is an all too frequent tendency to make a diagnosis of a specific occupational disease because of presumptive or actual exposure without the corroboration of other essential factors to a correct diagnosis. We are not justified in making a diagnosis of asbestosis, any more than of silicosis, in the presence of an X-ray film showing no distinctive pulmonary pathology.

Associated with exposure to asbestos dust is the occurrence in the sputum and pulmonary tissues of a peculiar formation known as asbestos bodies. These asbestos bodies have been described by a number of observers and are due apparently to the action of the tissues upon the asbestos fibre. Their exact significance is doubtful but it is commonly agreed at the present time that they are not diagnostic of pulmonary fibrosis and indicate merely that the individual has been exposed to asbestos dust.(2) (8) Some observers believe that where these asbestos bodies appear in the sputum in clumps, they indicate actual disintegration of lung tissue.

Dr. Miller of the United States Public Health Service, in the Public Health Reports,(9) describes the technique which he employes in the intraperitoneal injection of finely divided dusts in a state of suspension. Miller defined three types of reaction - Absorption, Inert, and Proliferative. The first is produced by relatively harmless or inactive dusts; the second by dusts which could cause pulmonary fibrosis; and the third is the typical reaction of silicosis. Recently, in a personal communication, Miller described the effects of the injection of three varieties of asbestos, namely chrysotile,

crocidolite, and amosite. Chrysotile is Canadian asbestos and, as previously stated, is largely magnesium silicate. Crocidolite and amosite contain only a very small quantity of magnesium, containing instead iron silicate in approximately the same quantity.

The three types of asbestos produced the same type of reaction - namely the one described by this investigator as inert. "We have been assuming," he states, "that the dusts producing this inert reaction cause pneumoconiosis of the diffuse fibrosis type as distinct from the proliferative reacting dusts which cause nodular fibrosis. This assumption, as you know, has not been proven by corresponding animal experiments with the same dusts, but results from observation of pathologic material from autopsies." He further states, "I believe that the gross behavior of asbestos in the tissues has further strengthened the value of our classification of dusts and the intraperitoneal test as a means of determining the harmful dusts by its clinical correlation."

Much work remains to be done before we can speak as authoritatively about asbestos as we do about silica. In the meantime, asbestos plants are being cleaned up and the dust controlled. This, together with the small number of persons employed, implies that we will probably never have the wealth of clinical material that has been available in silicosis. It is by no means certain that asbestosis progresses as does silicosis after withdrawal from dust exposure, nor does infection seem to be as closely and intimately associated with asbestosis as with silicosis. The answer to these and other problems resulting from exposure to silicate dusts demands further study both in the field and in the laboratory.

BIBLIOGRAPHY

(1) MORTALITY FROM RESPIRATORY DISEASES IN DUSTY TRADES - Frederick L. Hoffman - U. S. Department of Labor - Bulletin of the U. S. Bureau of Labor Statistics, No. 231, June, 1918.

(2) A MEMORANDUM ON ASBESTOSIS - E. R. A. Merewether, One of H. M. Medical Inspectors of Factories (Great Britain) - Tubercle - November, 1933; December, 1933; January, 1934.

(3) PULMONARY ASBESTOSIS - Kenneth M. Lynch and William Atmer Smith - American Review of Tuberculosis, Vol. XXIII, No. 6, June, 1931.

(4) STUDIES ON EXPERIMENTAL PNEUMONOKONIOSIS, VI. INHALATION OF ASBESTOSIS DUST: ITS EFFECT UPON PRIMARY TUBERCULOUS INFECTION - Leroy U. Gardner and Donald D. Cummings - The Journal of Industrial Hygiene, Vol. XIII, No. 2, February, 1931.

(5) THE MORBID ANATOMY AND HISTOLOGY OF ASBESTOSIS - S. Roodhouse Gloyne, M. D., D. P. H., - Tubercle - Vol. XIV, No. 10, July, 1933; No. 11, August, 1933; No. 12, September, 1933.

(6) EFFECTS OF THE INHALATION OF ASBESTOS DUST ON THE LUNGS OF ASBESTOS WORKERS - A. J. Lanza, Asst. Medical Director; William J. McConnell, Asst. Medical Director; and J. William Fehnel, Chemist; - Metropolitan Life Insurance Co., Public Health Reports, Vol. 50, No. 1, January 4, 1935.

(7) DISCUSSION - James L. Dubrow - Journal of the American Medical Association - Vol. 103, No. 11, September 15, 1934 (p.814)

(8) PULMONARY ASBESTOSIS - W. Burton Wood, M. D. Camb., M.R.C.P. Lond., D.P.H. and S. Roodhouse Gloyne, M. D. Leeds, D.P.H. - The Lancet, December 22, 1934.

(9) THE PHYSIOLOGICAL RESPONSE OF THE PERITONEAL TISSUE TO DUSTS [illegible] AS FOREIGN BODIES* John W. Miller, Acting Assistant Surgeon, R. R. Sayers, Surgeon, U. S. Public Health Service- Public Health Reports, Vol. 49, No. 3, January 19, 1934.