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Asbestosis as Differentiated from Other Pneumoconioses

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The preceding discussion has shown that the roentgenological appearance of a well-defined asbestosis is quite distinctive and that it differs materially from any of the other pneumoconioses. For that reason, I shall not limit my part of the discussion to the differences between this disease entity and the other dust diseases but shall include other conditions of the lungs which may be misdiagnosed as asbestosis.

The first and commonest cause of misdiagnosis is poor film technique. A perfectly normal chest can be made to look like one with definite first-stage asbestosis by slight underexposure, by lack of contrast, and by blurred vascular markings due to too long exposure time. Such films are especially common in overweight persons, the heart shadow usually being horizontal and often presenting a shaggy appearance due to compression of the vascular shadows in the lower lobes. Films lacking proper penetration, sharply defined detail, and lack of contrast should be rejected for the diagnosis of any occupational disease of the lungs but particularly of asbestosis.

Another condition which causes diagnostic trouble at times is emphysema due to any cause, when there are one or more adhesions of the diaphragm due to past pleurisy. The "ground-glass appearance" is very easily read into such a film. When to this are added some retained secretions in the lower lobes, which often are bronchiectatic in emphysema, the misdiagnosis is established.

A pneumoconiosis of which we have become increasingly aware in recent years is that due to excessive deposition of coal dust. It appears quite certain now that free silica is not needed as an essential component of the dust to develop the characteristic changes. The fine lacy character of the shadows plus some emphysema gives a pattern which looks very much like "ground glass" and is easily mistaken for early asbestosis.

All these examples I assume represent patients who present themselves to their physicians with symptoms of dyspnea, with a history of some exposure to asbestos dust, and with chest films of the character I have mentioned. How easy it is to fall into the trap of a gunshot misdiagnosis! Unless other possible causes for the x-ray changes are considered first and a detailed past occupational history is evaluated, along with the character and extent of the most recent dust exposure, another worker who actually needed reassurance will be told that his lungs are full of asbestos dust and that he should quit his trade! From that point on, his symptoms usually increase to a marked degree, and he has developed what to him is a real disability. In my experience, such doctor-induced disability is commoner in some areas than is the disability from the disease itself.

Because of the complete lack of unanimity of opinion about this disease among physicians and because of the need for more clearly defined criteria for diagnosis and advice on continued employment in the trade, the medical and industrial hygiene advisers of members of the Asbestos Textile Institute

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have set up a so-called Air Hygiene Committee. This committee has met on several occasions to determine whether those who are most intimately associated with this disease can agree on the various medical and hygiene phases of the problem.

At the last meeting, each medical member of this committee brought with him sample films of the various stages of asbestosis, including some he had classified as essentially negative. All films were reviewed by the eight physicians who were present. We found fairly good agreement on the more advanced stages of asbestosis but practically none on the borderline degrees. A number of my films from the textile mill in North Carolina for which I am consultant, representing cases which I had classified as "early or first-stage asbestosis," were called "essentially negative" by a number of physicians present who had had long experience with this disease. The reverse also was true; some films I had called negative which others thought represented first-stage asbestosis. The same disagreement was found with the other films which were presented. It was our final conclusion that it is impossible to clearly define a first-stage case and that it can be called "essentially negative" one day and "first stage" the next by the same reader. This was not new. It has been emphasized repeatedly by Pendergrass, beginning in 1938.

Our group finally agreed that little attention should be paid to the first-stage diagnosis, that little, if any, disability has been shown to exist with the borderline stages, that no one should be advised to stop work with these questionable degrees of change, and that workers should be kept at their regular jobs but the dust control should be so improved that their cases will not progress to the stage where everyone agrees that they have asbestosis.

Regarding recommendations of transfer to less dusty or nondusty work, we agreed to the following:

1. Persons under 40, when the diagnosis is clear cut (beyond first stage), should be moved to a nondusty job if possible.

2. Where progression is seen on serial films, regardless of age, less dust exposure is clearly indicated.

3. Exceptions should be made if there will be material improvement in dust control on the present job within a very short time.

I have deviated from my assignment of differential diagnosis, because the diagnostic criteria are so intimately associated with it. Until we have some agreement on x-ray interpretation, the present chaotic state of affairs will continue.

In the differential diagnosis, it is my belief that a new approach is essential, not only with asbestosis but also with nonoccupational diseases as well. Because textbooks in medicine are written by diseases, each followed by a listing of other diseases which must be differentiated from it, the disease itself becomes fixed first in the diagnostician's mind. He says to himself, "This is it," and he pays only cursory attention to the diagnostic criteria of the disease and to the diseases from which it must be differentiated. A more scientific approach would be to list all the conditions and diseases which are compatible with the x-ray pattern which the patient presents and then, after a complete and detailed medical and occupational history, physical examination, and laboratory studies, to see which of the positive findings more closely fit the listed diseases.

I should like to cite a case which undoubtedly would not have been misdiagnosed had this approach been used.

A railroad shop repairman, aged 57, had to stop work because of increasing shortness of breath several years ago. Cyanosis and dyspnea became progressively worse, and he died of anoxia and right heart failure several months ago. His work in the railroad shop included welding, unpacking, and repacking asbestos insulation around locomotive boilers and doing some fitting and grinding. The chest x-ray film showed a diffuse mottled and micronodular pattern in both lungs, and compressed lower lobes due to high position of the diaphragm, with numerous annular shadows of less density scattered throughout both lungs. Asbestosis was diagnosed by the man's physician, because there had been some asbestos-dust exposure, because he had always heard that the x-ray pattern of asbestosis was rather bizarre (and this certainly was), and

because it did not look like the siderosis of welders about which he had read something recently. The degree of asbestos-dust exposure was not inquired about (it was spasmodic and minimal). Other diseases and conditions of the lungs were not considered. The annular shadows should have brought emphysematous blebs or air cysts into the differential diagnosis, as well as the granulomatous diseases. Miliary tuberculosis had to be ruled out as well as berylliosis with emphysema. Sarcoidosis also had to be considered, although the annular shadows were against it. After definitive study, the diagnostic possibilities should have been reduced to emphysematous blebs plus pneumonitis or polycystic disease plus pneumonitis. Also, there were none of the characteristics of asbestosis and no pleural involvement.

After postmortem study the diagnosis was clarified—a widespread congenital cystic lung, with interstitial fibrosis due to long-standing infection. The cuboidal and columnar epithelium lining of the cysts is noteworthy. Also, there was complete absence of anything resembling asbestosis bodies, an absence of pleural thickening, and no evidence of any hyaline fibrosis. Even with this clear-cut postmortem evidence, the physician who originally made the diagnosis still believes that this was a case of asbestosis and is sending the tissue slides to various pathologists. He should be convinced soon that he most likely was wrong, which will be most embarrassing to him.

This case, along with many others which could be cited, clearly points up the need to consider all possibilities in cases presenting bizarre x-ray patterns. Each case must have painstaking study, including not only the immediately preceding occupational history but also a history of every job from the first one on. An example is a foundry worker with a chest film which was characteristic of a moderately well-developed asbestosis. Careful study of the occupational environment revealed no possibility of asbestos exposure in this shop. More detailed early occupational history revealed that the man had been a plumber's helper during the late teens and early twenties, doing all the sawing of asbestos pipe coverings, usually in confined basement rooms.

When the most detailed analysis and study will not reveal the true diagnosis, which happens in occasional cases, taking a biopsy specimen of the lung should be considered. This has become a rather simple procedure with little danger of complications. It must be remembered, however, that because of the relatively small piece of lung usually obtained for biopsy, there always is some danger that the diagnostic pathological change will not be revealed.

In conclusion, as with all chronic chest diseases, so especially with asbestosis, all possible causes of the pathology revealed by the chest film must be considered. Poorly studied cases result in misdiagnoses which not only are embarrassing to the physician but which may also cause irreversible harm to the patient. We owe it to our patients and to the medical profession to so thoroughly investigate every obscure case that such unfortunate situations do not arise.

DISCUSSION

DR. KARL T. BENEDICT, West Boylston, Mass.: It is a privilege to substitute for Dr. Harriet Hardy; she has helped me many times, but a substitute is always a "second." The assignment is "the differential diagnosis." I have had no experience with asbestosis and, furthermore, I believe that experience with most pneumoconioses, as seen today, is gained only after 20 to 30 years' study, because it often takes that long to produce such disease.

In spite of the foregoing, I believe I am qualified to make certain remarks, because I have been practicing industrial medicine for 15 years in one of the world's largest artificial abrasive plants. Since 1940, we have taken more than 25,000 chest x-rays on some 5,000 abrasives workers. From 1911 to 1940, except in rare instances, we simply did so-called routine physical examinations.

I agree with Dr. Sosman that the physical examination is not worth very much in these circumstances. Thanks to Phil Drinker and others, dust control is much better in our plants today. In one large plant our dust counts run consistently 3,000,000 to 4,000,000 particles per cubic foot. We handle a great variety of dusts, for that is our business, and safe handling is essential.

Recently at the Seventh Saranac Symposium, several world-renowned authorities on pulmonary diseases attempted to define pneumoconiosis. I shall not. There was no definition there either. Instead there were minority and majority reports. One

group insisted that pneumoconiosis simply means dust in the lung. The other group restricts it to those conditions producing demonstrable disability. The hitch is, Who demonstrates the disability? The roentgenologist sees nodular fibrosis in the x-ray film: that is pneumoconiosis to him. At once, or sometimes much later, the industrial physician and hygienist set about to correct conditions to prevent progress of the pneumoconiosis or development of new cases. Later, though the worker still may not know of his trouble, the family physician has difficulty clearing an ordinary chest cold or the surgeon is faced with anesthesia worries. The occupational disease or the health department specialists in our state government want reports when "injury" has resulted. Compensation and insurance authorities talk about the number of pneumoconiosis cases, based upon those workers whose earnings have suffered. The compensation lawyer appears later in the picture, and alas sometimes the last, the pathologist, may be the first in proving that a case of tuberculosis or cancer is in reality pneumoconiosis.

I believe we all know when the man is suffering from pneumoconiosis, whether we be laymen or physicians. What he wants to know is, What is wrong? What can be done about it? Is he permanently disabled? Is his life shortened?

There can be only one answer: not better diagnosis or differential diagnosis or function studies but adequate dust control. Since different dusts require different degrees of dust control, witness the different effects from inhaling small amounts of aluminum (therapeutically) and beryllium or radioactive dusts. There is, therefore, good reason for you and me to continue to study pneumoconioses and their differential diagnosis. We in the abrasive industry are continuing this research. Specifically, we believe that under reasonable control alumina dust is no problem. You have heard of the "carborundum lung" no doubt; this is an unjust label, as my good friend Dr. Eddy, of the Carborundum Company, well knows, for Carborundum is just a trade name for the fusion product, silicon carbide. Furthermore, just what disease, disability, or pathology silicon carbide per se will produce in man has never been proved conclusively, I believe. It is entirely possible that this lung condition is caused by significant free silica contaminations. Further study is needed, and we intend that it shall be made.

Shaver's disease has been mentioned before. In spite of several years' research tending to prove that alumina fume is the cause, there are those who believe that silica fume is to blame. Industry is somewhat discouraged by the inconclusive nature of these and similar medical studies.

We all know about the problems related by beryllium and radioactive dusts. On the other hand, our concepts of clay and talc pneumoconioses have changed in recent years. And what do we know about zirconia or titania or magnesia or boron carbide or graphite dust inhalation? In our industry organic substances have caused only two minor cases of asthma. And we have seen no cases of cancer of the lung among our abrasives employees.

In conclusion, our goal is not a specific M. A. C.* figure but maximum dust control to eliminate all pneumoconioses. This goal is not easily attainable. Adequate differential diagnostic knowledge will help us to achieve it.

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* Maximum allowable concentration.