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Asbestos and Disease

By

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those exposed for more than 20 years, with increasing frequency with longer employment.

The incidence to be expected falls as the exposure level decreases and the time to appearance of significant disease increases. But even at the level of 5 mppcf, some disease can be expected to appear after 20 years of exposure.

Familial and neighborhood cases can be expected at lower frequencies than those seen in persons occupationally exposed.

As the exposure level falls, the character of the disease tends to change from predominantly parenchymal to that of pleural involvement, which, although less productive of disability is less readily diagnosed, and may indicate that the population is liable also to the risk of mesothelioma.

Some pleural plaques may be found in persons with no ascertainable exposure to asbestos or talc, or to a less provocative mineral, mica. This may be particularly true in areas where asbestiform minerals are common in the soil. In any one particular case, the cause may be conjectural.

Meurman has provided a useful table of the reported prevalence of pleural plaques and their relations to exposure which is reproduced as Table 9–12 (513).

DIAGNOSIS

In comparison with other diseases, medical science has not yet provided very much in the way of reliable and practical methods of arriving at a firm diagnosis of asbestosis in the absence of a reliable history of occupational or other established exposure. As the field of opportunities for exposure widens, the possibility of asbestosis and particularly that of the pleura increases, and the range in which diagnosis may be doubtful is at the same time increased. This is particularly true in the incipient stages of disease when further decrease in functional disability and shortening of life might be somewhat reduced by removal of the affected person from further exposure, and perhaps new skills learned that would be compatible with the patient's functional capabilities. The art of diagnosis, always a matter of weighing probabilities and of looking at the total evidence, is seldom subjected to greater challenge than in the lesser stages of asbestosis (532,534,602). Even the demonstration of fibers or of asbestos bodies in lung biopsy, itself not a procedure to be undertaken lightly, lends supporting rather than conclusive evidence.

The interstitial pulmonary fibrosis produced by asbestos and termed parenchymal asbestosis, described in Chapter 7, is essentially similar to that seen in some persons with no history of exposure, to which the terms Hamman–Rich syndrome or "idiopathic" diffuse interstitial fibrosis are sometimes applied (285,753). This unhappy dependence on a history of exposure for the making of a diagnosis is not uncommon in occupational medicine, as witness the state of diagnostic procedures in so-called coal workers' pneumoconiosis, byssinosis, and berylliosis. It is only late in the disease that the indications add up to a virtual certainty, and by then very little can be done for the patient or for his resultant socioeconomic problems.

The relative evidence can be conveniently considered in four categories: exposure history, functional impairment, radiographic changes, and pathological indications. Many of the items have been considered in the two preceding chapters (Chapters 7 and 8), and will be reviewed here only as they pertain to diagnosis.

Exposure History

As indicated above, a history of exposure is a most important element in the diagnostic evidence. Unfortunately, it may be quite difficult to acquire. Persons currently employed in the mining, milling, manufacture, or utilization of asbestos constitute only a part of the population at risk. Furthermore, the dustiness of their workplace 20 years ago, when the disease process was initiated, was probably much greater than it would be today. Persons exposed for only short periods many years ago, as for example in wartime temporary employment, may not think the experience worth mentioning, or they may actually have forgotten about it. Even short periods a couple of decades ago, when controls were not so stringent, may have been quite sufficient for pathogenesis to begin. Such short exposures are easily overlooked, but they cannot be lightly dismissed. The possibility of short exposure needs to be borne particularly in mind where the presenting picture is one of pleural change with little in the way of parenchymal involvement. (We will see later in Chapters 10 and 11 that these short exposures are even more critical in the induction of mesothelioma. In any case of interstitial pulmonary fibrosis of undetermined origin, of pleural calcification, or of alveolar capillary block syndrome, no effort should be spared to elicit possible exposure to asbestos. A correct diagnosis is not an academic matter; the patient's entitlement to compensation or care may be jeopardized, and he has little else to look forward to. Appropriate

clinical care, moreover, depends on the diagnosis: steroids, often used in cases of Hamman–Rich syndrome, are contraindicated in asbestosis, and persons with asbestosis should be kept under surveillance for the possible development of neoplastic changes.

The possibility of incidential or vicarious exposure must also be remembered. Mention was made in discussing the etiology of parenchymal asbestosis (Chapter 7) of the possibility of neighborhood or family exposure and of the appearance of disease in persons working around but not in the actual handling of asbestos, as in shipyards or building operations. This is certainly important in the construction industry (with approximately four million employees in the United States alone), in the shipbuilding and ship repairing industry, in power plants, in brake repair and maintenance operations, and in railroading as well as in various other major industries. One cannot neglect the fact either that there are over 3,000 asbestos consumer products on the market that may permit exposure in the course of their manufacture, use, or disposal. We have as yet only partial estimates of how important this last category of exposure may be.

Functional Impairment

Meiklejohn, says Hutchinson, claimed that he could tell which of the girls in a factory had asbestosis by dancing with them (363). Our more prosaic generation seldom indulges in this approach to diagnostic evidence, and seeks perforce more quantifiable and perhaps more objective information. Even in fairly early stages of the disease, there are some indicators that can be sought once a suspicion of exposure has been aroused or in the course of a routine examination of exposed personnel. One that sometimes develops quite early is a reduction in the carbon monoxide diffusion capacity of the alveoli (243,535,602), which may be detectable before any complaint of dyspnea is offered. Changes in compliance are considered by some to provide an early suggestive sign (205,495). Reduced rate of lower lobe emptying in expiration, as revealed by an admixture of radioactive xenon, is advocated by others (711). At a somewhat later stage, dyspnea accompanied by reduced vital capacity and lung volume, and the presence of persistent, fine rales at the bases of the lungs constitute a very suggestive combination, even in the absence of demonstrable radiographic changes.

Later in the disease, clubbing of the fingers is often seen and cyanosis may be present. By this time, however, dyspnea is often marked and

radiographic appearances consistent with the diagnosis have usually appeared.

Radiographic Changes

For systematic evaluation of chest radiographs in suspected cases of asbestosis, the UICC classification, developed for silicosis and coal workers' pneumoconiosis, has been modified to the form known as the ILO–U/C classification, and is particularly useful for the recording of pleural changes and irregular parenchymal infiltrates, as described in Chapter 7 (365).

Parenchymal changes provide few radiographic features that are characteristic of asbestosis, but some are highly suggestive particularly when they occur together (Plates 7–2,a–h). The irregular, reticular, and nodular changes are more commonly seen in the lower two-thirds of the lung fields and are generally more easily visible on the right side. The apices remain lucent and massive confluent shadows are uncommon. Hilar enlargement is not usually marked until pulmonary hypertension develops. A shaggy heart outline is not rare, interlobar septa may be visible, and shadows described as resembling Kerley "B" lines may appear. As lung volume diminishes, the apicocaudal diameter is diminished, the costophrenic angle is narrowed, and the lesser interlobar fissure descends.

Pleural thickening may lead to obliteration of the costophrenic, and sometimes of the cardiophrenic angles, and to tenting of the diaphragm, although the latter may be found without adhesions. The pleural fibrosis may appear as soft tissue between the lung periphery and the chest wall, and adhesions may distort the lung shadow. This is particularly true in the region of the lingula or mid-lung fields ("mid-lung tug"). The fibrosis may give some graded shadowing over the peripheral field where the rays become tangential to the thickened pleura.

Hyaline pleural plaques may appear as faint diffuse shadows that persist in repeated films when viewed *en face*, and may be seen a little more clearly when viewed end on. The presence of calcified pleural plaques, distinguished from intrapulmonary calcification, are practically pathognomonic of exposure to asbestos, talc, or (occasionally) mica, especially in the absence of past thoracic hemorrhage, trauma, or tuberculosis (Plates 8–1,a–o).

Asbestos exposure must be considered when pleural effusion, free or loculated, has come on quietly, particularly if it undergoes spontaneous

resorption. The question of mesothelioma also enters into the differential diagnosis of this condition.

Pathological Confirmation

While the sum of clinical evidence may strongly suggest the disease and warrant the diagnosis in an occupationally exposed person, in most instances it will be rendered unequivocal only after microscopic examination of lung tissue obtained by trans-bronchial biopsy, needle biopsy, open chest biopsy, or autopsy. The extent to which surgical intervention is justified in the search for an unequivocal diagnosis is a matter for clinical judgment in each individual case. In general, intervention is not essential. The hazards mentioned in Chapter 7 may be minimal in trans-bronchial biopsy with fiberoptic techinques, but even this procedure is not free of risks (383). The appearance of typical interstitial alveolar fibrosis accompanied by numerous asbestos bodies, will serve to confirm a diagnosis that functional and radiographic evidence may have left equivocal; but for reasons given in earlier chapters, the presence of asbestos bodies, or even of asbestos fibers alone is not sufficient evidence of asbestosis (208,440,473). The histological appearance of pleural thickening, while highly suggestive, is not specific for asbestosis, but the presence of numerous asbestos bodies or fibers in the tissue or in contigous parenchyma will serve to support the diagnosis. The presence of hyaline, and particularly of calcified, plaques in the typical locations mentioned above would very strongly support the diagnosis. [A]

Differential Diagnosis

As pointed out by Turner-Warwick, widespread pulmonary fibrosis may be the end result of a variety of lesions occurring in the lungs (753). She groups the possible preceding events into four broad categories: (1) granulomas from many causes, e.g. extrinsic allergic alveolitis, sarcoidosis, berylliosis; (2) alveolar exudates from drugs, persisting left ventricular failure, chronic renal failure, etc.; (3) pneumoconioses; and (4) unknown, e.g. cryptogenic fibrosing alveolitis.

Intrathoracic calcification can also result from a variety of causes, as can pleural effusion. Many possibilities have, therefore, to be taken into account in the diagnosis of a disease that has few really characteristic features. One frequently has to fall back on exposure history, but for reasons that have been stressed in preceding pages, histories, particularly negative ones, can be misleading.