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Characteristics and Etiology of Pleural Asbestosis

DEFINITION

Under this heading will be discussed the changes, other than neoplastic, that can develop in the pleura or pleural cavity as a result of the inhalation of asbestos dust. Four major changes are included: pleural thickening, pleural effusion, hyaline plaques, and calcified plaques, individually or in combination. These changes may or may not be accompanied by parenchymal asbestosis (as defined in Chapter 7) of various degrees, or by neoplastic developments (Chapters 10–12).

PATHOLOGY

In the last decade or so, pleural changes have received increasing attention in their own right not just as simple complications or extensions of parenchymal changes. Much of the following account of their pathology is drawn from reviews by Meurman (513), Chahinian (136), Lesobre (455), Israël-Asselain (367), Jones (379), Le Bouffant (446), Solomon (704), and their colleagues and extended by experience gained in the studies conducted at Mount Sinai.

Pleural Thickening

Simple thickening of the visceral pleura is commonly seen as an accompaniment of marked parenchymal asbestosis. In some of the cases

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described by pioneer investigators tuberculosis was present, and undoubtedly it contributed to the massive thickening described and to the adhesions that were present. But as the prevalence of tuberculosis declined and that of parenchymal asbestosis increased, the thickening responsive to the presence of asbestos became established. It consists simply of collagenization of the underlying connective tissue layer with no particular features to distinguish it from that caused by other agents. Changes in the visceral pleura are more commonly seen, however, in the lower two-thirds of the lung, as is parenchymal asbestosis. They vary in thickness from a thin layer of fibrous tissue to several millimeters. This fibrosis may be continuous with that of the interlobular septa and with that of the underlying parenchyma, as described in connection with "pleural drift" in the preceding chapter. Asbestos fibers and asbestos bodies may be found in the visceral pleura as well as in the parenchyma. The parietal pleura is generally more extensively and heavily involved than the visceral. Fibrosis may develop both posterolaterally and anterolaterally, over the diaphragm, and along the lines of the lower ribs. Every part of the parietal pleura may be affected—costal, mediastinal, diaphragmatic, and pericardial. The two pleural layers may be adherent but are often separable by manipulation. True adhesions may be present in advanced cases even in the absence of superadded infection.

Benign Pleural Effusion

A serofibrinous, or sometimes a serohemorrhagic, fluid may appear in the pleural cavity without provoking much in the way of symptoms or general signs. While it may be difficult to determine just when the condition started, the onset can vary from apparently acute through subacute to chronic. After a variable length of time the fluid may be spontaneously reabsorbed, but it is likely to reappear later on the same side or contralaterally. The fluid usually contains a number of lymphocytes, some erythrocytes, and perhaps a few neutrophils, but it is sterile. It is usually rich in albumin. Pleural thickening is usually, but not necessarily, present as well. Asbestos bodies are rarely found in the fluid, although they are generally present in the underlying parenchyma.

Hyaline Plaques

Whitish, glistening, raised areas, aptly termed plaques, appear on both visceral and parietal pleurae, but much more commonly in the latter, often in the midst of apparently normal tissue. Even when the surrounding pleura is thickened, the plaques are recognizable as distinct entities. A few mm thick, they may be scattered as small discrete nodules or, at the other extreme, in confluent masses up to 50 cm^2 or more in extent. The borders are fairly sharply defined and the transition to surrounding tissue abrupt. Many, if not most, of the confluent plaques have a mammilated surface.

On section, hyaline plaques are seen to have a laminated structure, which microscopic examination shows to consist of bundles of collagen fibers, separated by irregularly arranged spaces or pseudolacunae. The fibrous tissue is separated from the underlying fat-containing connective tissue by a thin network of elastic fibers. Its free surface is covered by mesothelium which is clearly not involved in plaque formation. The plaque grows from the underlying connective tissue by the action of fibroblasts. There are few cells in the substance of the plaque and these are surviving fibroblasts. There are no inflammatory cells present, although histiocytes may be found around the edges of the plaque, probably as a tissue reaction to what is essentially a foreign body. No inflammatory process is seen preceding plaque formation.

An alternative concept of plaque formation is that fibrin is first deposited on the surface of the mesothelium and that this is transformed into collagen without the intervention of fibroblasts. At a later stage, mesothelium is believed to grow over the collagenized deposit from the periphery.

When hyaline plaque tissue is examined by electron microscopy, electron-dense bodies are seen that are identifiable as incipient calcifications. Le Bouffant, from whose account much of this description is taken, makes the important point that these incipient calcifications appear in the hyaline plaques on the visceral as well as in those on the parietal pleura, and that they may be found in thickened pleura away from plaque formation. Analysis shows that hexosamine is high in the collagenous material and that mucopolysaccharides, removable by the action of hyaluronidase, are present. Neither asbestos fibers nor asbestos bodies are generally found in hyaline plaque material, but Fondimare and colleagues report finding 1000 asbestos bodies in 4 g of hyaline pleura after digestion, and uncoated fibers by electron microscopy in pleural plaques (250). Le Bouffant implies that the hyaline plaques represent a nonspecific reaction, but that extension of the calcification beyond the "incipient" stage may very well be a specific reaction to asbestos.

Calcified Plaques

Calcified plaques are found much more commonly on the parietal than on the visceral pleura. They have a more opaque white or ivory

color, are hard, and fracture easily when peeled off or bent. The calcification tends to be distributed irregularly in the plaque and appears first toward the center of it or in the centers of the mammilations. The calcified plaques are apparently formed by extension of the "incipient" calcified deposits of the hyaline plaques. If Le Bouffant's thesis is substantiated, a critical question may be not why plaques are found more commonly on the parietal pleura, which seems to be the note sounded by many, but why the calcification extends to the rest of the plaque preferentially in those on the parietal pleura.

When the calcification is removed by treatment with acid, it is seen that the underlying collagenous matrix is indistinguishable from that of the noncalcified portions or from that of the hyaline plaque. The full-fledged calcified plaque consists of about 30% organic and 70% mineral matter. The mineral portion contains about 84% phosphate and 9–11% carbonate. Israël-Asselain speaks of lipids being involved in the calcification process as they are in other deposits such as in atheroma plaques but gives no details. Meurman did not find any asbestos bodies in the plaques of the large number of cases that he examined, but did find them in the lung substance of 86% of those with bilateral calcified plaques. Le Bouffant *et al.* go further in saying that, while they may be found in the visceral pleura, they do not occur in the parietal pleura, in the plaques, or in mesotheliomas that are clearly parietal in origin (447). Fondimare and colleagues, however, found 1000 asbestos bodies in 4 g of hyaline pleural tissue after digestion (250). Asbestos fibers have been found in calcified plaques by at least three investigators (355, 446, 738). The plaques are similar to the "sugar-icing" of unknown etiology sometimes found on the peritoneal surface of the liver and spleen and occasionally on the serosal surface of the intestine (513).

CLINICAL CHARACTERISTICS

Here again we have drawn largely from the classical descriptions of the disease state and have supplemented the information with the experience gained at Mount Sinai. Our chief sources have been Meurman (512,513), Kiviluoto (401,402), Chahinian (136), Israël-Asselain (367), Anton (48), Lesobre (455), Jones (379), and their associates who will not be specifically cited as we present the consolidated information.

History and Physical Examination

For the most part, the development of pleural changes adds little to the symptomatology of the patient. Those who are already inflicted

with parenchymal disease may experience some slight aggravation of their dyspnea or sense of restricted breathing; those who have little or no parenchymal involvement may be surprised when routine examination reveals abnormalities. Infrequently, however, pleural fibrosis may be so extensive as to result in severe or even fatal dyspnea, especially if there is much concomitant parenchymal fibrosis. Pleural effusion may provide something of an exception if the onset is acute, but even here the increase in the patient's complaints may be minimal. Under these circumstances, the patient may not realize the connection with exposure to asbestos or may not even be aware of any exposure. As will be brought out in considering the etiology of the condition, exposure may have been limited to living in the neighborhood of an asbestos plant or even to residing in an area where asbestos minerals are prevalent in the soil. It may take a series of leading questions to establish the opportunity of exposure, the significance of which may not be clear even to the physician, until the cases are seen as part of a statistical series.

Most investigators comment on the lack of functional impairment to be found in those who have minimal or undetectable parenchymal involvement (444,531,562), although Becklake and her colleagues have pointed to the finding of some diminution of respiratory function in such circumstances (69). We have made similar observations. There may be a feeling of tightness in the chest or difficulty in taking a breath, but only occasionally does any complaint of pleural pain arise. The dyspnea and pain present in Chahinian's cases may well have been due more to the accompanying parenchymal involvement than to the effusion. Other reports speak of the onset of effusion as being virtually symptomless (262,455). We have been surprised to find, upon occasion, that pleural friction rubs are heard with no pain being volunteered by the patient. Perhaps the slow development of the pleural thickening explains the lack of pain. [A]

Where there is grossly thickened pleura, restriction of chest expansion may be seen, and there may be some dullness on percussion and some diminution of breath sounds. A friction rub or pleural crackling may be heard. Effusion would increase restriction of chest movement and produce marked dullness on percussion, shifting with the patient's position unless it is loculated. Hyaline plaques and calcified plaques add little to the background of physical signs that may be present from accompanying parenchymal or other pleural change.

Mackenzie and Harries speak of pleural abnormalities causing marked restriction of ventilatory capacity without any effect on gas transfer (471) and Soutar *et al.* found a significant correlation in 11 cases between the extent of pleural shadowing and reduction of vital capacity (706). Becklake *et al.* report that the group mean values for all pulmonary

function tests were significantly more impaired in those with radiological evidence of pleural changes but with the same degree of radiological change in the parenchyma (69). But individuals will vary greatly in the extent to which they reveal such changes.

Radiography

Pleural thickening may result at a fairly early stage in some obliteration of the costophrenic (Plate 7–2b) and sometimes, of the cardiophrenic angles. The border between the lung and the pleura may become ill-defined as thickening develops (215). Adhesions may occur with progressive obliteration of the pleural cavity (421). Pleural changes occur earlier and are usually most marked over the lower and middle portions of the lungs, but in a small proportion of cases may extend upwards towards the apex (677). They may be unilateral, but are more commonly bilateral, and are best seen on the lateral aspects and in the paravertebral sulci (421). When circumscribed, the thickening may resemble an encysted pleural effusion or a pleural neoplasm. The interlobar fissures may be rendered visible (704) (Plate 7–2g). The diaphragmatic outline may become ill-defined and the contours of the heart shadow may be ironed out by pleuritic change. In advanced cases the cardiac shadow may be distorted.

Pleural effusion appears as a denser shadow occupying a gravitational position in the lower portion of the cavity. It will shift with change of position of the patient when it is free; but it may be loculated by adhesions.

Circumscribed hyaline plaques are not always seen in standard P–A radiographs, but oblique views may reveal those on the parietal pleura as rounded, oblong, somewhat denser areas on the diaphragm or costal areas, or as guttae along the lines of the ribs (215). Those on the visceral pleura may be indistinguishable from more generalized pleural thickening. Many more will usually be revealed on thoracotomy or at postmortem examination. In this connection, Anton summed up by saying that radiographic visualization depends on the thickness of the plaque and their orientation to the X-ray beam (48). Narrow plaques may appear simply as nodular opacities at the periphery of the lung fields; with thicker plaques *en face* faint shadows may be seen, usually limited to the outer parts of the lung fields, and a vertical lucent band often separates them from the plaques seen in profile. The faint shadows may be rounded, but occasionally display the bizarre contours found with calcified plaques.

As calcification develops, the plaques become more easily visible (Plates 8–1,a–e) but it is still not uncommon to find many more at

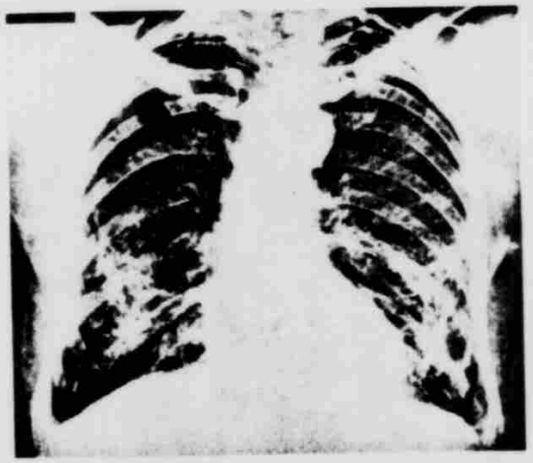


Plate 8–1a. Bilateral pleural calcification.

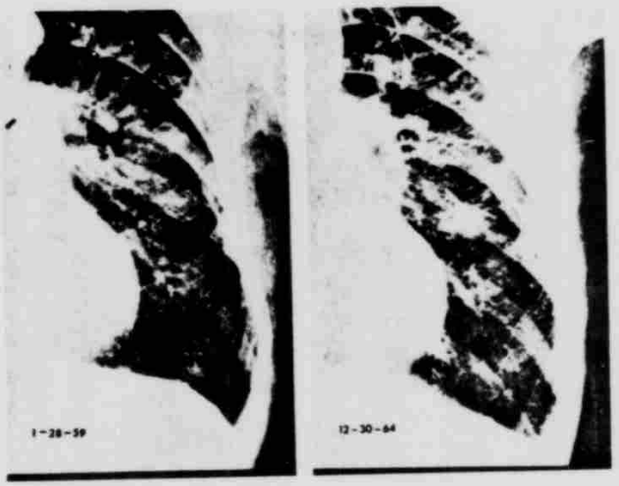


Plate 8–1b. Pleural calcification developing over five years.



Plate 8–1c. Bizarre pleural calcification pattern.

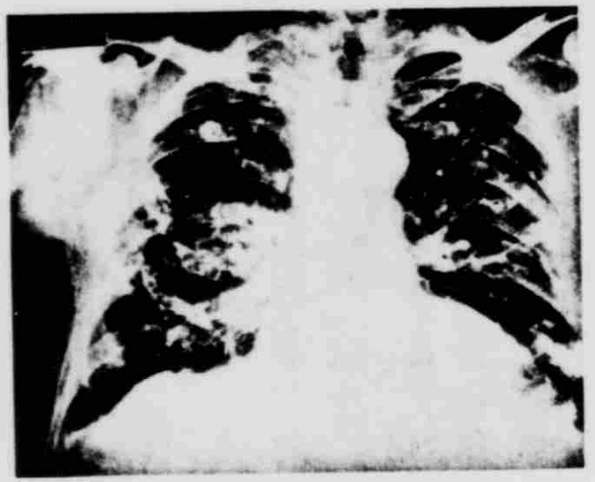


Plate 8–1d. Bilateral bizarre pleural calcification pattern.

autopsy than were seen on the film (355). They are best seen and their extrapulmonary location in the lateral portion of the chest demonstrated by oblique views (Plates 8–1l,m) or when pneumothorax is present. On fluoroscopy they are seen to move with the parieties rather than the lung. They may be few and so small as to be barely noticeable, or so extensive as almost to form a cuirass (681). The plaque shadow is usually of uneven density. Calcified plaques commonly appear bilaterally and in generally symmetrical arrangement as irregular patches, mainly in the middle and lower zones. However, unilateral plaques are not uncommon. Their edges appear clear cut, but the shape is usually irregular. Bizarre, holly-leaf, racemose, and lacelike are other terms often applied to their appearance (Plates 8–1c,d). Seen end-on, they may be found as linear densities following the ribs (Plate 8–1h), on the diaphragmatic contours (Plates 7–2g, 8–1k), or in a lateral view behind the sternum (692), and are sometimes then described as being like inverted plates or saucers. Plaques, unlike pleural thickening, do not involve the costophrenic angle. Calcification with a clear costophrenic angle is seen only with asbestosis. Calcification with an obscured costophrenic angle can occur with tuberculosis as well as with asbestosis. In some cases plaques may appear on the surface of the pericardium

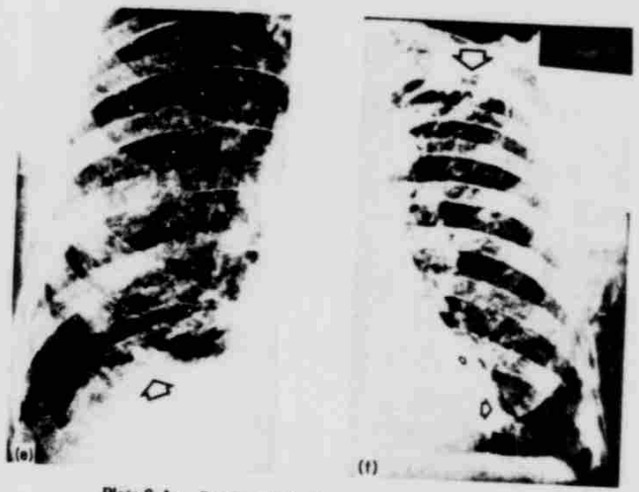


Plate 8–1e. Button calcification.
Plate 8–1f. Apical pleural and pericardial calcifications.

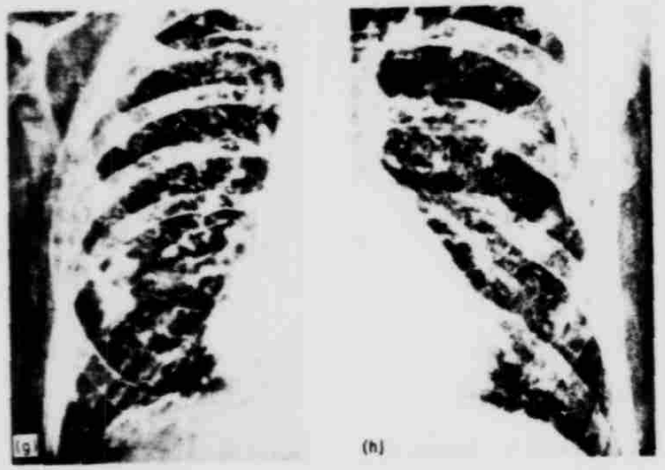


Plate 8–1g. Mass shadows calls for differential diagnosis between pleural calcification, mesothelioma, and carcinoma.

Plate 8–1h. Pleural calcifications following rib lines.

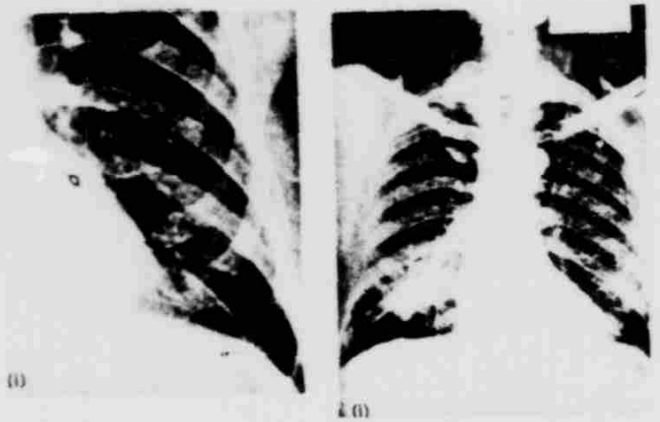


Plate 8–1i. Calcification of pericardium. Patient died from peritoneal mesothelioma with asbestosis.

Plate 8–1j. Marked pleural calcification. Patient gave history only of three months' exposure to rovings.



Plate 8–1k. Pleural plaques on diaphragm more visible with high penetration radiograph. Only domestic exposure to asbestos.



Plate 8–1l. Calcification behind heart revealed by oblique radiograph. Question raised of differential diagnosis. Death from lung carcinoma.

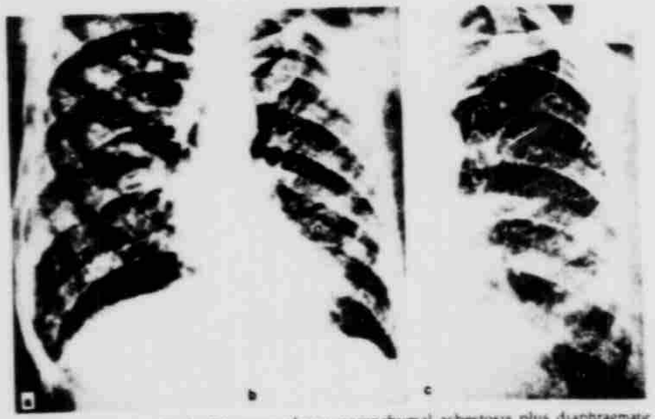


Plate 8–1m. a, Right PA views shows parenchymal asbestosis plus diaphragmatic plaques. b, Left PA view shows parenchymal asbestosis only. c, Left-oblique view reveals pleural plaque. At autopsy patient was found to have carcinoma of the lung and peritoneal mesothelioma. An outstanding example of all four pathogenic processes developing in the same individual.

(Plate 8–1i), and occasionally they may appear in the interlobar fissure or on the surface of the mediastinum (661) (Plate 8–1n).

The incidence of pleural calcification will be greatly underestimated if only routine posteroanterior films are taken. Oblique views that permit looking at plaques end-on permit many more to be seen. A more penetrating high kilovoltage technique, combined with the use of a fine grid, is also helpful in increasing their visualization (Plate 8–1k,o). Tomography is useful in revealing calcified plaques that might otherwise remain hidden and in differentiating them from the calcification occasionally seen in the pulmonary parenchyma. Computer measurement of attenuation by area has recently been added to the diagnostic armamentarium (420).

ETIOLOGY

The etiology of such pleural changes as accompany parenchymal responses to asbestos dust need not be further discussed since they share causation with the parenchymal responses, except to say that in

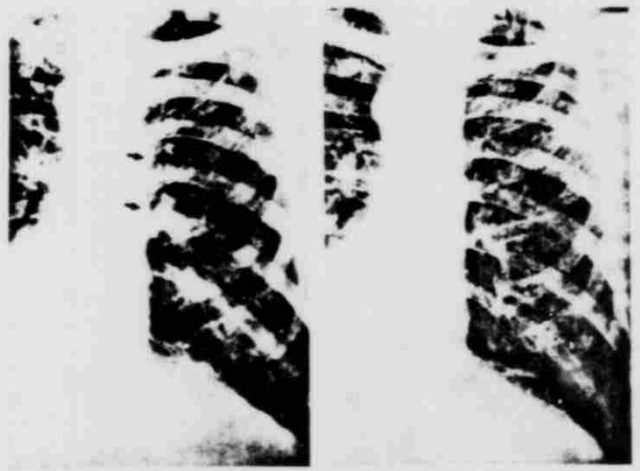


Plate 8–1n. Mediastinal calcification revealed by oblique view.

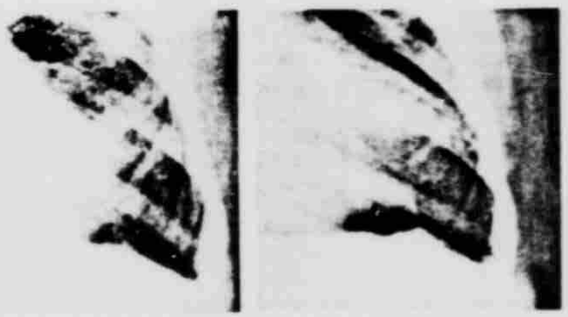


Plate 8–1o. Pleural calcification revealed by higher kilovoltage (R). Patient died from peritoneal mesothelioma.

these cases a certain degree of correlation has been reported between the degrees of parenchymal and pleural change (531). But in the last twenty years many cases of pleural change have been reported in persons with minimal or radiologically undetectable parenchymal disease and the etiology has sometimes been the subject of dispute.

In 1960 Kiviluoto published a report of finding radiological evidence of pleural plaques in farm dwellers, sometimes many kilometers away from an asbestos mine (401). Experience had already established the close association between plaques of this nature and asbestotic changes in the lung, but there seemed to be no possibility of occupational exposure in most of these cases. Later investigations (402,426) showed that the occurrences were largely confined to an area of East Finland in which numerous deposits of asbestos occur and in which asbestos had been worked on a small scale or used in the homes intermittently from prehistoric times (see Chapter 1, Historical Background). Samples of dust collected at various points showed a distribution of asbestos in concentrations inversely proportional to the distance from the asbestos mine opened at Paakkila in 1918 (see Chapter 5). However, since the frequency of the plaques was markedly higher among older people born before the mine opened, it was concluded that at least some of the exposure must have come from incidental use of asbestos. The percent distribution of calcified pleural plaques by age is given by Raunio (599) as follows:

30–34	35–39	40–44	45–49	50–54	55–59	60–64	65–69	70–74	75–79	80+
1	2	8	10	18	21	29	29	32	24	32

In his extensive report, Meurman found bilateral thickening of the parietal pleura common (39% of cases) over all of Finland, more so in urban than in rural populations (512). Asbestos bodies were present in 57.6% of the 264 lungs studied and again more frequently in urban dwellers. They were also more frequent in the lungs of workers in building and associated trades. However, both calcified plaques and asbestos bodies were more often found in the remote "calcified" areas of the country reported by Kiviluoto. Asbestos bodies were not detected in those plaques examined, but they were found in the lungs of 87.5% of those with bilateral plaques. On the other hand, 40% of those whose lungs showed asbestos bodies were free from pleural plaques which is not surprising in the light of studies of metropolitan dwellers discussed in the preceding chapter.

A somewhat similar situation is reported from Bulgaria (810). A small mine opened in 1942, but the age distribution of calcified plaques is very similar to that reported by Raunio, rising to 60% in the 70–79 age

group. Out of 155 persons examined 132 had no direct contact with asbestos production. The authors attribute the finding to tilling of soil naturally contaminated with asbestos since the plaques tend to occur in certain families working certain areas but not in others. In a later account (120) the position is summarized as follows:

> The development of endemic, nonoccupational pleural plaques is due not only to air pollution by asbestos mining or manufacturing. The natural erosion of an asbestos-containing soil and the dust formation from it, as well as its cultivation can also and independently produce pleural calcification.
>
> a high incidence of persons with pleural plaques is established in regions with anthophyllite deposits, whereas typical bilateral pleural calcifications are seldom detected in areas with deposits of tremolite asbestos . . .
>
> some mineralogical and epidemiological data suggest a possible participation of the fibrous mineral sepiolite in the etiology of endemic pleural plaques.

A fairly high frequency of calcifications was found by Anspach in Dresden (46) and by Manns *et al.* in Munich (480,481). The latter authors attributed them to causes other than asbestos, particularly to histoplasmosis, and this point will come up in the following chapter (Chapter 9) in connection with differential diagnosis. Bohlig (91) challenges this attribution and considers an underestimate of the effects of asbestos dangerous because the prevalence of pleural plaques could be an index of the liability of the population to mesothelioma.

A 1970 report from Poland noted that bilateral pleural calcifications were found in 11 out of 3,826 persons radiologically examined in the district of Kilarovo, where there are circumscribed deposits of amphibole asbestos but no mining or production of asbestos goods (121). Occupational histories, however, were not provided; the ages of the affected individuals ranged from 62–92.

Czechoslovak experience provides a parallel example (358). Hromek in 1962 reported that, in a mass survey of some 10,000 persons in the Pacov district during the period 1956–1960, 339 cases of pleural calcification were found which appeared to be identical with those described in asbestos workers, talc workers or those working with mica. It appeared predominantly in older persons, rising from 0.3% in the fifth decade to 17.9% in the eighth and 19.5% in the ninth (77 persons survived into their nineties). Males were more affected than females. The people affected were primarily farmers. There is no industry of any consequence in the district. The geology is similar to that of other parts of the Bohemian–Moravian highlands. There was no radiographic evidence of parenchymal change in any of the affected persons. Navratil and Trippe, however, also working in Czechoslovakia, question whether pleural calcifications develop apart from some, albeit minimal, expo-

sure to asbestos or the operation of some other well recognized cause such as tuberculous infection (541).

A similar situation has recently been reported from Turkey (806). In a mountainous area where chrysotile deposits occur, there is no mining, but the material is used indiscriminately to paint walls and floors of houses. In three localities studied, the prevalence of pleural calcification averaged 2.6%, with the highest rate of 14% in one village with 589 people. As in other studies cited above, the prevalence increased with age, from 0 in the 6–20 age group, to 19 in those over 70.

Of 59 cases found by the Pneumoconiosis Research Unit in South Africa to have only calcified plaques, only 32 had occupational exposure, and one of these for only two months (562). Asbestos fibers were found in one-half of the sputa examined from these persons, and asbestos bodies in one-tenth, indicating exposure even in those without occupational opportunity. No correlation was found between the appearance of pleural plaques and intensity or duration of exposure or with age.

Over the last ten years there have been many other reports of pleural changes and particularly of calcification of the parietal pleura in persons who have had low, intermittent or vicarious exposure to asbestos dust (212,248,257,263,355,470,471). Parenchymal changes, at least as revealed by radiography, are often minimal or undetectable. As early as 1943, Siegal *et al.* described 5 cases of pleural plaques without apparent parenchymal changes in talc workers (681). In most cases a long period of time, usually more than 20 years, has elapsed since initial exposure.

Harries *et al.* found pleural abnormalities to be ten times more frequent than parenchymal disease in dockyard workers (319). Of 36 cases found by Hourihane *et al.* in a systematic review of autopsies at the London Hospital, 29 were without any history of exposure (355). They went on to say "it would appear that there is a dose–response relationship with regard to the development of these plaques and their detection. Thus, on the one hand, subjects with pleural plaques which are radiologically invisible in life have small plaques with little calcification, rarely have histological asbestosis, have scanty asbestos bodies in the lungs, and have not lived close to a known major source of asbestos. On the other hand, patients with radiologically detectable plaques have large pleural lesions with plentiful calcification, have histological asbestosis with readily detectable asbestos bodies, many live close to an industrial source of asbestos, and most give a history of exposure to asbestos."

It would seem, therefore, that the relationships between pleural response and exposure to asbestos dust constitute a matrix. Along one

axis lie the various manifestations in descending order of severity—marked pleural thickening with or without effusion, extensive calcified plaques, minor calcified plaques, and hyaline plaques. Along the other axis are ranged various degrees of exposure—by persons handling asbestos, by persons associated with but not actually engaged in the handling of asbestos, by members of workers' families and persons living in the neighborhood of asbestos plants, through persons living on and working soil containing asbestos, to trivial or unrecognized exposure. The higher the opportunity for exposure, the more likely is it that the more severe forms of reaction will occur; the smaller the opportunity, the less likely it is that anything but a minor response will develop. Associated with the developments is a strong time element. Those persons with a heavy exposure will tend to develop grossly thickened pleura, with or without superadded effusion, within a moderate period (20 years or more); those with slight exposure will show calcified plaques if at all only after a longer period (50 years or more for the agricultural workers of central Europe). At its upper corner (more severe response and higher exposure) the matrix merges into that for parenchymal asbestosis, which coexists with the pleural in many persons. At its lower corner (mild response to trivial exposure) it passes insensibly into the terra incognita of unrecognizable changes in the face of undetermined exposure. The matrix constitutes a two-dimensional continuum with few, if any, real lines of demarcation.

With this concept in mind, we can return to our five etiological criteria and summarize the extent to which they are satisfied by what we have defined as pleural asbestosis.

Association between exposure and syndrome. As just mentioned, there is a continuous spectrum of relationship between exposure to asbestos dust and development of pleural responses.

Dose–response relationships. As the dose decreases, pleural response decreases less rapidly than does parenchymal response so that we have many instances where low, vicarious, or even apparently trivial doses have been found to have resulted in plaque formation, after a long interval, with minimal or virtually no corresponding parenchymal involvement.

Concentration of agent in tissues. Asbestos fibers and asbestos bodies are found in the vicinity of thickened visceral pleura, sometimes in greater concentration than in the underlying parenchyma. Fibers are not conspicuous in the fluid of pleural effusion, but asbestos bodies and fibers have been repeatedly demonstrated in the parenchyma of the person so affected and occasionally in the fluid. Fibers have been several times demonstrated in the decalcified collagenous matrix of

calcified plaques but are usually quite small and visible only by electron microscopy.

Animal experimentation. The pleura of most experimental animals will show granulomatous response when asbestos is injected, but similar reactions are produced by other mineral dusts, so that specificity is wanting (175). The cellular reactions to asbestos, however, closely resemble those that occur in the parenchyma.

Influential factors. Many authors suggest that some sensitivity reaction is involved in at least plaque formation, and others [including the IARC Advisory Committee (274)] take pains to leave the door open for the cooperation of some other factor not yet recognized, but no details are given. The importance of age has been repeatedly demonstrated, particularly in agricultural workers not exposed to any industrial source of asbestos, but this may simply mean that the individuals have to live long enough for the very slow process to produce recognizable results. The question as why only a certain proportion of the population apparently exposed to very low doses develop the disease remains here, as with other responses to asbestos, entirely open. To ascribe the phenomenon to variations in susceptibility is simply to dress ignorance in a transparent cloak.

9

Prevalence, Diagnosis, and Course of the Asbestoses

With the nature of parenchymal and pleural asbestosis established, this chapter will turn to practical questions that confront the medical practitioner who has to deal with the spectrum of asbestotic disease that may be encountered in his patients. Parenchymal and pleural effects can appear in almost any combination and will be considered together. As in preceding chapters, the account given here combines the experience gained in the course of extensive studies and practice at Mount Sinai with that reported by others (153,215,333,531,562,677,801,803).

PREVALENCE

The medical practitioner needs to have an estimate of how often he is likely to see cases of asbestosis in his practice, or what the epidemiologist calls the prevalence in a specified population group. The medical literature is not devoid of data; numerous reports appear of the number of cases seen, the frequency with which asbestosis is found at autopsy, mortality figures, etc. But many of these data have limited usefulness because they do not indicate the size of the exposed population from which the cases were drawn, or what is called the population at risk, so that the reader cannot try applying the reported experience to the group of patients that he is likely to see. One trouble is that the disease that is seen today had its origin in the past, 20 or more years ago, and too little is known about the number of people who were employed in various occupations at that time. This is particularly true

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