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COMPANY PHYSICIANS

ASBESTOS-RELATED CHANGES

The attached article by Sargent provides
pertinent information regarding pleural plaques and
the association with asbestos exposure and may be
helpful to you.

MEDICAL DIVISION

Bruce W. Karrh, M.D.
Director

BWK:ceb

DUP 0937369

BETTER THINGS FOR BETTER LIVING . . . THROUGH CHEMISTRY

DU 038339

Pleural Plaques: A Signpost of Asbestos Dust Inhalation

E. Nicholas Sargent, M.D., George Jacobson, M.D., and Jay S. Gordonson, M.D.

The general contamination of the urban atmosphere by asbestos dust has become a matter of public concern. Industrial exposure of workers to this dust is also widespread. The radiologist and other physicians should be familiar with the occupations in which asbestos dust exposure occurs and should be aware that asbestos is used in more than 3000 commercial items. The major cause for failure of public health agencies to control asbestosis is the long latent period (often 20 years or more) between the beginning of work exposure and the appearance of overt disease. The disease now being discovered was initiated by industrial exposure during or before World War II. Before the 1950s, few precautions were taken to protect workers from this noxious dust. Thus it follows that to prevent asbestos dust disease in the year 2000 we must recognize its characteristic radiographic appearance now and take appropriate measures to reduce exposure wherever evidence of the disease is found.

Although asbestos dust inhalation is not the sole cause of pleural plaques, it is certainly the most common. The association between pleural plaques and occupational or nonoccupational asbestos dust exposure has been amply confirmed by population studies.¹ The prevalence of pleural changes seen on radiographs has been shown to increase in relation to the estimated dose of asbestos, the age of the patient, and the length of time after initial exposure to asbestos.^{2,10,22,24} This is further confirmed by a fairly strong correlation between pleural plaques found on postmortem studies¹³ and a history of previous exposure to commercial types of asbestos.^{12,24} All varieties of asbestos

have been implicated, but the highest rate of association occurs with anthophyllite.⁶

In addition, other factors such as the site and nature of the geologic deposit of the asbestos may be important. For example, in the chrysotile mining areas of Quebec, the occurrence of pleural plaques in miners is common in some mines, rare in others, and absent in one of the largest mines not too distant from the others.¹⁴ In nonoccupationally exposed populations, particularly in Finland, the prevalence of pleural plaques relates to the proximity of residence to certain mining areas, and the air is so polluted that even cattle are contaminated.^{5,16,18} Similarly, plaques have been observed in some agricultural populations¹⁷ and in cattle¹⁹ in areas in which the soil is contaminated with asbestiform materials.

PATHOGENESIS AND PATHOLOGY

To date, no totally satisfactory explanation has been developed to explain how the pleural reaction that results in plaques develops in response to the inhalation of asbestos fibers and particles. Asbestos bodies have rarely been found in plaques,¹³ but asbestos fibers may be present. However, the lack of asbestos fibers in plaques mentioned by previous investigators has largely been due to inadequate techniques.²⁵ By means of recently developed techniques of transmission electron microscopy, selective area electron diffraction, and microchemical analysis of particles, many small submicroscopic asbestos fibers in pleural plaques have been identified unequivocally by morphologic, structural, and chemical studies.^{16,22} It is of interest that these are more concentrated in the calcified zones than in the fibrous zones.¹³ Asbestos fibers are also commonly observed in the lung tissues of patients with plaques.^{7,14}

Inhaled fibers longer than those that can be phagocytosed by histiocytes or giant cells move downward and outward in the lungs. This type of gravitational movement (so unusual in other pneumoconioses) is influenced by the coincidence of three variables: the presence of thin, sharp asbestos fibers; the soft, easily punctured pulmonary structure; and the constant motion of the lung.²⁶

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Inhaled fibers longer than those that can be phagocytosed by histiocytes or giant cells move downward and outward in the lungs. This type of gravitational movement (so unusual in other pneumoconioses) is influenced by the coincidence of three variables: the presence of thin, sharp asbestos fibers; the soft, easily punctured pulmonary structure; and the constant motion of the lung.²⁵

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A proposed theory of pathogenesis of pleural plaques is as follows: Fibers, particularly the long ones, tend to move toward the lung periphery, some leaving the lung and reaching the parietal pleura along the chest wall, diaphragm, and pericardium. Most of the emergent asbestos fibers pass through the soft tissue of the intercostal muscles and muscular portion of the diaphragm. Others are blockaded by the hard tissue of the ribs, the dense fibrous tissue of the tendinous portion of the diaphragm, or the pericardium. The localization of pleural plaques at these sites, especially posteriorly and laterally, is also said to be enhanced by the recumbent position during sleep. The plaques are almost exclusively on the parietal pleura and are practically never encountered in the apices of the lung, below the 11th rib, in the region of the costophrenic angles, or anteriorly near the costal cartilages. Thus the plaques result from mechanical or other reaction to the asbestos fibers. Individual sensitivity is probably also an important factor in their formation.

Grossly, the pleural plaque is characterized by hyalinized discrete elevated gray-white or pearly areas involving the parietal pleura (Fig. 1). Despite varying sizes and shapes, the pattern of the plaques is repetitive. There is a flat or slightly uneven surface, with steep edges rising abruptly from the surrounding normal pleura, and here and there rounded mounds, with somewhat overhanging edges.¹⁴ The thick pleural plaques are typical in appearance, and the classic comparison is to frosting or sugar icing (*Zuckerguss*), as is seen on serous membranes.¹⁵

Microscopically the plaques consist mainly of avascular, acellular, hyalinized, laminated, intermingling collagenous fibers, usually with an orientation parallel to the surface. Elastic staining shows intact lamellae beneath the plaque, in continuity with the surrounding normal parietal pleural connective tissue. The pleural plaque is, in fact, not a pleural plaque at all, but subpleural or extrapleural. Early in its formation, the mesothelial cell lining of the normal parietal pleura takes no part in the process, but merely acts as a passive covering.^{14,16} The growth of the plaque is slow and is of nonexudative type. Thus adhesions between the visceral and parietal pleurae are virtually absent. Plaque



Fig. 1. Postmortem specimen showing white hyalinized plaques on the parietal pleura. Small plaques follow the contours of the surface of the upper ribs. Larger confluent plaques cross the interspaces over the lower ribs posteriorly. Relatively normal parietal pleura is present between the elevated, well-margined borders of the plaques.

formation on the visceral pleura is rare and then only minimal.

Calcium deposition is present microscopically in a high proportion of plaques. It is of the dystrophic type, appearing as granules along the course of the acellular and degenerated collagen fibers and ceasing abruptly where the abnormal connective tissue changes into normal pleural tissue.

RADIOGRAPHIC ASPECTS

The radiographic changes in the pleura following asbestos inhalation result from two types of parietal pleural reaction. Most frequent is the localized, limited, plateaulike smooth or nodular area of pleural thickening that corresponds to the pathologically observed pleural plaque. Much less commonly, a non-specific extensive diffuse pleural thickening may be found.

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Fig. 2. Advanced stage of well-developed bilateral pleural plaque formation crossing intercostal spaces. Note the sharp margin (arrows) adjacent to the ribs, where the plaques are seen in profile, even through the overlying scapula. Tapering of the margins superiorly and inferiorly is evident, particularly on the right. Localized well-developed plaques alter the contour of the left hemidiaphragm and to a lesser extent the right (white arrow head).

Noncalcified Pleural Plaques

Noncalcified hyalinized pleural plaques are not visualized radiographically until the fibrous tissue is sufficiently thick; thus they often go undetected, and their prevalence during life is underestimated. Only a small percentage of early plaques identified at postmortem examination have been detected on routine PA radiographs.⁷ In this projection a well-developed pleural plaque results in either a *profile* presentation or an *en face* presentation. The plaque in profile appears as a sharply marginated white band of water density, roughly

paralleling the inner margin of the lateral thoracic wall. It is usually not obscured by the overlying scapular shadow (Fig. 2).

The very early plaque will show only a thin white line of increased density adjacent to the rib that has been likened to a flange.³ Thus the profile line of early plaque formation is easily overlooked and often detected only in retrospect (Fig. 3), particularly when it resembles the normal inner rib surface. As it enlarges, it becomes somewhat oval and moderately protuberant, with tapering superior and inferior margins typical of an extrapleural lesion (Fig. 4). A plaque rarely extends vertically more than

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Fig. 3. Minimal pleural plaque formation (small arrow). Note also early calcification in the region of right diaphragmatic pleura (large arrow).

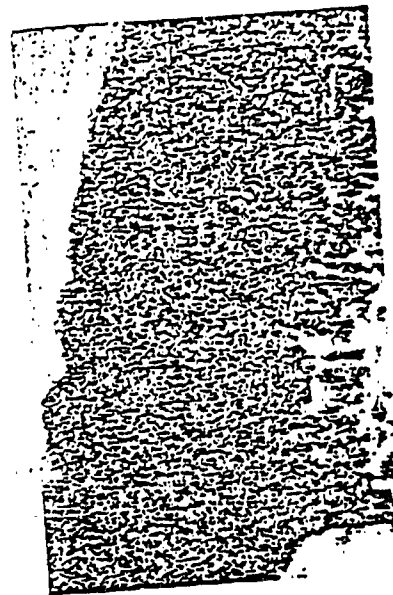


Fig. 4. Oval, slightly protuberant pleural plaque seen in profile (lower arrows). The superior and inferior tapering margins are characteristic of a subpleural lesion. Note the adjacent normal pleural line (upper arrow).

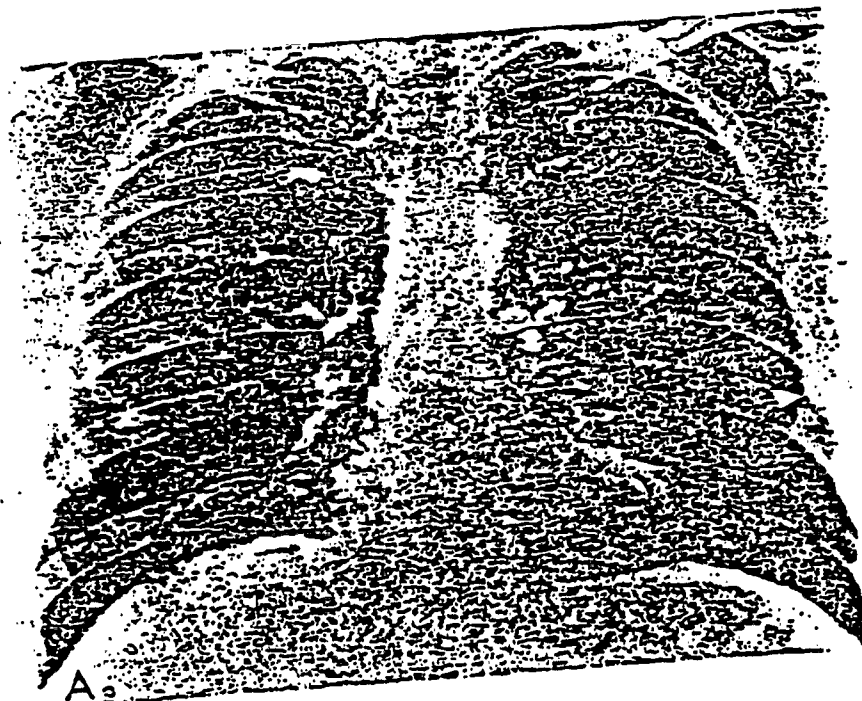


Fig. 5A.

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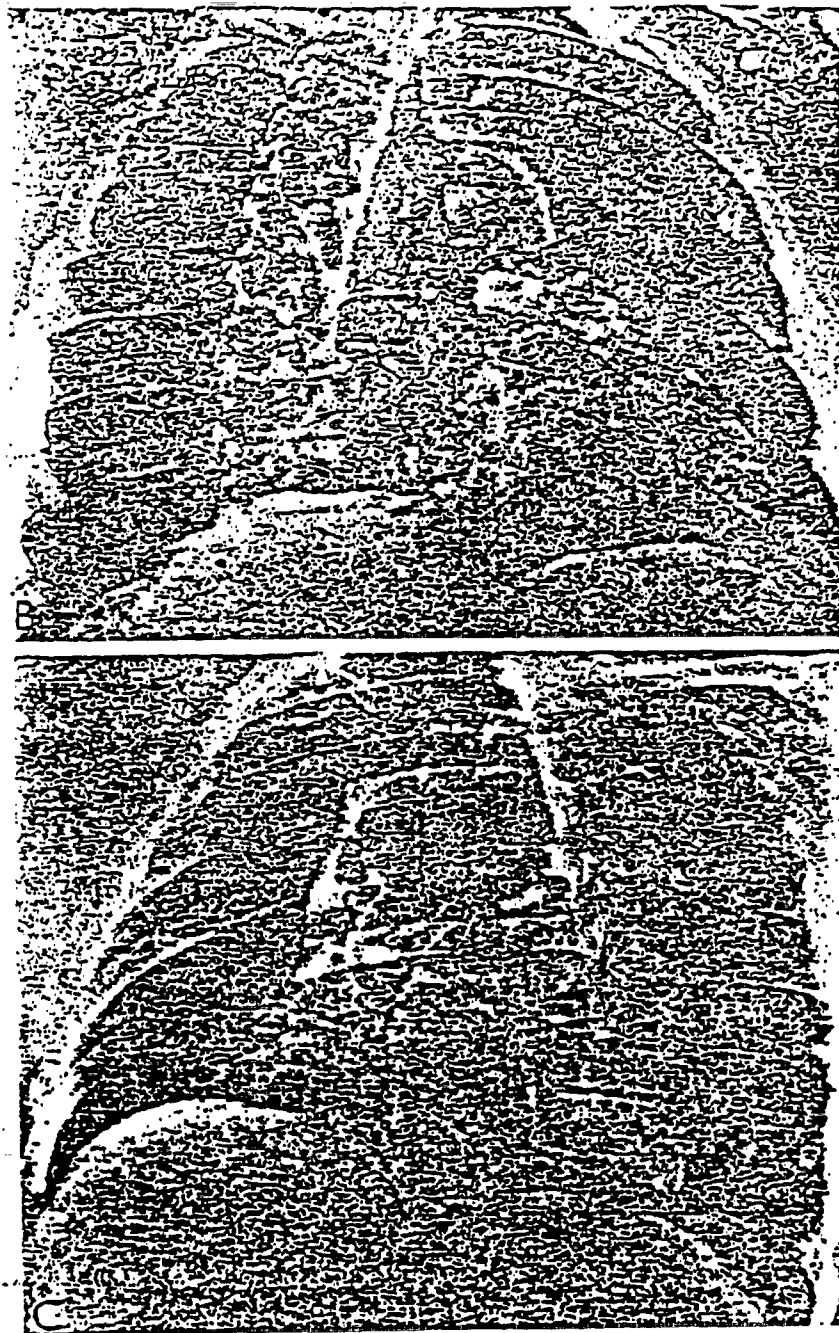


Fig. 5. Pleural plaques (black arrows), bilateral noncalcified on/fac. (A) PA film. The faint veil-like opacities along the lateral halves of the lung fields spare the apices and costophrenic angles. A few small plaques are seen in profile (white arrows). (B) Right oblique view. The on/fac plaques are projected in profile, posteriorly on the right and anteriorly on the left (arrows). (C) Left oblique view. The converse is noted. The costophrenic angles are spared on all projections.

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four interspaces.^{20,26} The thickness of the plaques varies from 1 mm to more than 10 mm, but it is usually in the range of 1-5 mm. Plaques are usually bilateral and often symmetric. Most commonly they lie adjacent to the 5th, 6th, 7th, and 8th ribs and interspaces.²⁰

As stated earlier, involvement of the apices or costophrenic angles is extremely rare. Early, the plaque may be solitary, but later several distinct nonconfluent plaques are often found. Less than 25% of patients with plaques have evidence of accompanying interstitial lung disease.²⁰ Diffuse pleural thickening is infrequent and is characterized by uniform homogeneous density, by a lack of nodularity of contour, and particularly by accompanying costophrenic angle obliteration. Pleural effusion is quite rare.

Plaques in profile are found less commonly on the diaphragm and rarely on the pericardial surface, where they appear as one or more localized elevations on the normal convex surface of these structures. These are usually accompanied by additional plaques along the lateral thoracic wall; they are seldom isolated lesions. On the diaphragm they are found at some distance from the angles, i.e., close to the central tendon. When uncalcified, they must be differentiated from normal (scalloping) or abnormal variations in diaphragmatic contour (Figs. 2 and 7).

The second type of radiographic presentation, the *en face* projection, is that of a faint, ill-defined veil-like opacity or shadow.¹ These also occur most commonly in the region of the 5th to 8th ribs (Fig. 5A-C). They are rarely detected when in the medial half of a thorax, but they are often obvious when they overlie the lateral aspect, where they are more oblique to the x-ray beam and hence absorb more radiation. The *en face* plaque rarely appears uniformly rounded; it shows a peripheral irregularity of contour that has been likened to the fringe of a map or holly leaf. Because of the faintness in outline, it is often dismissed as an artifact on a single radiograph; persistence on repeat examination will prevent this error. Because it is flat, it is less radiopaque than an intrapulmonary nodule of the same diameter. Careful observation shows that the *en face* shadow is often less dense centrally, unlike the more uniform homogeneous density of the round or oval intrapulmonary nodule. When multiple, the indi-

vidual *en face* plaques are often separated by a band of normal radiolucent lung.

The use of oblique views is strongly recommended, not only for confirmation of suspected pleural plaque formation but also for detection of additional plaques or pleural thickening unsuspected in the PA projection (Fig. 5A-C). With these views, the *en face* plaque will often present a profile appearance. The use of fluoroscopy and spot filming to determine the proper obliquity for demonstrating a plaque in profile is of great value.

Oblique views have confirmed the fact that plaques spare the apices and the costophrenic angles and are most frequently found along the posterior reflection of the costal pleura. They may extend laterally and anteriorly as far as the costal cartilage junction, but they rarely overlie the rib cartilages. Pneumothorax has confirmed the parietal location of a plaque and has demonstrated the absence of adhesions between the visceral and parietal pleura. Adhesions, if present, do not occur in areas of plaque formation. Tomography is of little assistance in demonstrating noncalcified plaques, as the composite shadows of adjacent ribs tend to blur out the ill-defined shadows of the plaques. Computerized axial tomography has demonstrated the plaques.¹¹ Ultrasonic examination has been offered as a more sensitive modality for detection of pleural plaques. It has been suggested that plaques less than 2-3 mm thick, which are missed by radiographic examination, can be detected by ultrasound.²⁰

Plaques versus normal anatomic shadows. The combined thickness of the tissues comprising the visceral and parietal pleural layers that surround the normal lung is not ordinarily measurable on the radiograph. The normal pleural tissue consists of a single layer of mesothelial cells covering the surface of a small amount of fibroareolar tissue measuring 10-50 μ in thickness. On the routine PA projection a vertical line of water density may be seen, sometimes paralleling the medial surface of the first three or four ribs along the lateral thoracic wall. This vertical line has variously been called the companion shadow, the pleural shadow, the pleural line, the pleural margin, and the pleural stripe. It is contributed to by a combination of muscles (subcostal and intercostal), the areolar tissue of the endothoracic fascia, and, to a

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greater or lesser extent, deposition of fat. The sharp medial margin of the shadow is best seen when the x-ray beam is tangential to the surface of the tissues causing this shadow. This normal pleural shadow increases in width as it is traced superiorly toward the first rib; it usually tapers rapidly inferiorly, becoming imperceptible below the level of the 4th intercostal space. In the PA projection the thickness of the shadow is never greater than 5 mm; however, in the oblique projection this normal pleural shadow is wider, varying from 0 to 10 mm in the men and somewhat less in women.¹⁰ Occasionally, a very thin, barely perceptible line may be seen medial to the lower half of the lateral thoracic wall; 1 mm has arbitrarily been designated as the maximum acceptable thickness of this normal pleural shadow below the level of the 4th intercostal interspace.

The costal slips of origin of the serratus anterior and external abdominal oblique muscles have a characteristic radiographic pattern: a rhythmic sequence of shadows between successive intercostal spaces (Fig. 6). They are most commonly visible over the 8th rib, but the 7th rib is also affected, and occasionally all of the ribs from the 5th to the 9th are involved. They appear as one or two distinct triangular shadows or a combination of two opacities superimposed. They should not be mistaken for multifocal plaques, which may also show a series of shadows. Multiple plaques are more likely to differ from each other in size and shape, unlike muscle shadows, which are usually more uniform in size. The muscle shadow usually has one sharply defined border and elsewhere fades into the surrounding soft tissues. A slightly oblique projection will often resolve this difficult problem. A plaque that is well defined in profile becomes ill defined when rotated to an *en face* position. Additional plaques are often seen on the rotated film. Plaques may become more distinct as rotation moves the overlying muscle shadows, whereas muscle shadows will alter their shape or disappear.

Pleural thickening should not be confused with extrapleural deposits of fat. The oblique view is again helpful, the fat producing a wavy margin most prominent on the posterolateral surface of the ribs, usually sparing the anterior and paravertebral areas.¹⁷ Tomography may be



Fig. 6. Normal costal slips of origin of serratus anterior and external abdominal oblique muscles (arrows). These are bilateral symmetric shadows, not to be confused with pleural plaques. The sharp medial margins fade inferiorly. The shadows disappear completely on oblique views.

helpful in revealing fat density in contrast to the water density of pleural thickening.

Calcified Pleural Plaques

Pleural calcification usually occurs 20 years or more following exposure to asbestos. The incidence increases with the time interval.¹² The exposure to asbestos does not have to be prolonged; it can be on the order of only a few weeks or months, depending on the concentration of the dust. However, calcification in pleural plaques is a relatively uncommon radiographic finding. It is much more common histologically, occurring in approximately 87% of plaques.¹⁸ Radiographically demonstrable evidence of interstitial pulmonary disease has been reported in only one-third of patients with pleural plaque calcification, but conversely more than 50% of patients with extensive



Fig. 7. Development of costal and diaphragmatic pleural plaques (arrows). Calcification, beginning in 1973, is well visualized in 1978. The very early plaque formation in 1967 was overlooked and found retrospectively. Diaphragmatic pleural plaques rarely occur without additional plaques along the ribs.

pulmonary fibrosis eventually end up with pleural calcifications.²⁵

As a rule, calcified plaques are more obvious and more striking than uncalcified plaques on the roentgenogram. When the x-ray beam strikes a plaque margin tangentially, the calcification in the plaque is seen as a dense white line, usually somewhat discontinuous, paralleling the chest wall, diaphragm, or cardiac border. Since the calcium is deposited near the center of the typical subpleural hyalinized plaque, it is separated from the inner surface of the rib by a line of water density produced by the surrounding pleural thickening. The calcifications are often small and (especially in films of low contrast) easily overlooked unless systematically sought after (Figs. 3 and 7). They are rare in the upper zones.

If the x-ray beam strikes the surface of a calcified plaque *en face*, it presents an irregular and unevenly dense pattern, again often likened to the fringe of a holly leaf or map. Oblique views are extremely helpful in detecting calcifications that may be unsuspected in the PA projection, in confirming *en face* calcifications by bringing them into profile, in localizing calcifications in the region of the diaphragmatic pleura or the pericardium, and in excluding costal cartilage calcification (Fig. 8).

When calcified plaques are small or when they are superimposed on the relative denser ribs or diaphragm, but particularly when the film is underexposed or poorly processed, they are often overlooked. They are well seen on grid films. Unlike the uncalcified plaque, the calcified plaque may be better outlined and better localized by tomography. Pneumothorax has demonstrated that the calcified plaque is in the parietal pleura and is not associated with visceral pleural involvement. The location of these plaques in the parietal pleura can also be confirmed during life by fluoroscopy or cineradiography, which reveals that on respiration the lung moves independently of the plaque and the plaque moves synchronously with the chest wall.²⁶

DIFFERENTIAL DIAGNOSIS

The rare basilar pleural effusion of asbestosis resolves slowly and may leave signs of residual diffuse pleural thickening, particularly in the costophrenic angles. Pleural plaques may also

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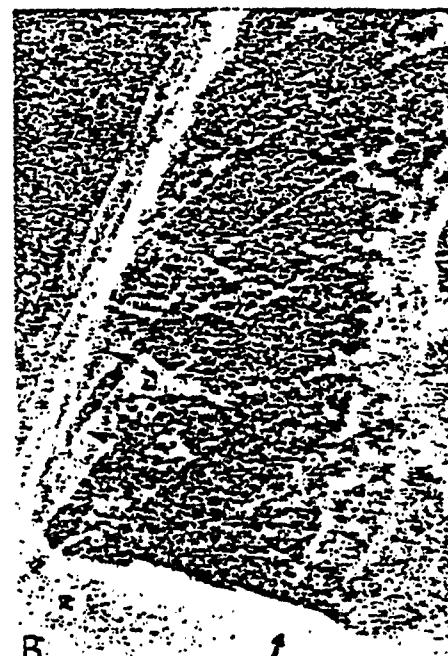
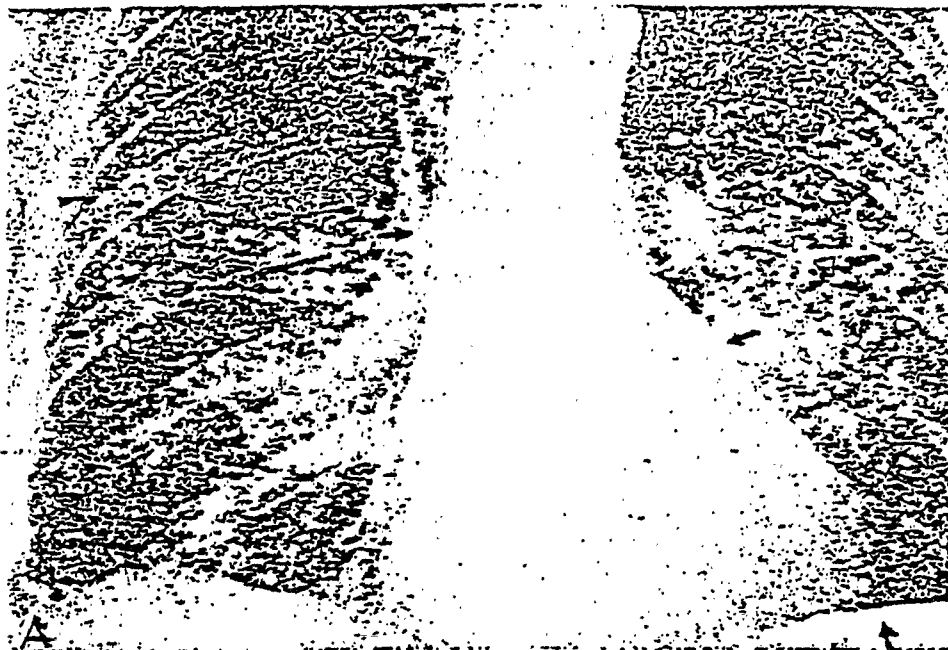


Fig. 2. Calcified pleural plaques. (A) PA film. *In face* calcifications on right; linear calcifications in profile in mediastinal reflection of pleura on right and in pericardium on left (transverse arrow); linear calcification in vertical profile in left diaphragmatic pleura. (B) Left oblique film. A linear pleural calcification is seen in profile in the area of the central tendon of the right hemidiaphragm (arrow). The *en face* plaques of (A) now appear in profile as extensive linear calcifications (arrow heads) adjacent to anterior ribs.

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be associated with bilateral obliteration of the costophrenic angles, but only uncommonly. Pleural thickening secondary to hemorrhage or infection usually involves the visceral pleura as well, particularly extending into the costophrenic angles.

Calcification of the pleura secondary to hemorrhage or infection is usually unilateral and often extensive and sheetlike. Fluoroscopy of the chest or cineradiography may be of value in demonstrating that the calcification is not confined to the parietal pleura. The calcification does not move synchronously with the chest wall as does a calcified pleural plaque. Pneumothorax will show adhesions between the visceral and parietal pleurae along the area of calcification.

Localized pleural thickening and callus, simulating asbestotic plaques, may occur from rib fracture. These are generally unilateral, and the overlying rib deformity is helpful in differential diagnosis. Other rare causes of plaque-like pleural calcifications include radiation, scleroderma,¹⁷ and chronic mineral oil aspiration.⁴ It should also be noted that residual barium in the peritoneal cavity from a ruptured viscus in the past can simulate diaphragmatic pleural calcification. Calcified or metallic pleural plaques occur in other pneumoconioses, including those caused by tremolite talc, mica, Bakelite, calcimine, tin, and barite.⁴ Even more rarely, inhalation of silica and coal dust has been mentioned. However, more likely an unknown exposure to asbestos dust is responsible, not necessarily in the mines but in other occupations associated with the mining process.

Discrete, localized noncalcified pleural

thickening in the pleura may occur with localized mesothelioma, metastatic disease, lymphoma, or myeloma. These are usually unilateral and may mimic pleural plaques. Conversely, pleural thickening due to asbestosis may simulate multiple pulmonary nodules or even healing rib fractures. Differentiation between multiple nodular pleural plaques and pulmonary neoplasia can be made with oblique views, which confirm the pleural location of the plaques.¹¹

Bilateral pleural plaques are signposts indicating pleural asbestosis. They will be detected with greater frequency if particularly looked for on the radiograph. They should be considered to be caused by asbestos dust inhalation until proven otherwise, and a careful lifetime occupational history must be obtained.

Epidemiologic studies have amply confirmed the association between asbestos dust exposure and bronchial carcinoma, and the risk is even greater if there is a history of heavy smoking. A recent study showed that bronchial carcinoma was 2¼ times more frequent in men with pleural plaques.⁸ In addition to its association with lung cancer, there is increasing evidence of an association between asbestos dust exposure and cancer of the larynx and cancer of the gastrointestinal tract. Furthermore, there is an increased incidence of primary malignant mesothelioma of the pleura, pericardium, and peritoneum. Since the occurrence of malignancy is usually on the order of 24-52 years after the initial exposure to asbestos, any patient with bilateral pleural plaques should have lifetime surveillance.

REFERENCES

1. Aantos HC: Multiple pleural plaques. Part II. *Br J Radiol* 41:341-348, 1968
2. Becklake MR: Asbestos-related diseases of the lung and other organs: Their epidemiology and implications for clinical practice. *Am Rev Respir Dis* 114:187-227, 1976
3. Edge JP: Asbestos related lung disease in a British ship building population with particular regard to the incidence of bronchial carcinoma in men with pleural plaques. A mortality study. *Am Rev Respir Dis* 115:211, 1977 (abstract)
4. Felson B: *Chest Roentgenology*, Philadelphia, WB Saunders, 1973, pp 489-492
5. Fletcher DE, Edge JR: The early radiological changes in pulmonary and pleural asbestosis. *Clin Radiol* 21:355-363, 1970
6. Harries PG, MacKenzie FA, Sheers G, et al: Radiological survey of men exposed to asbestos in naval dockyards. *Br J Ind Med* 29:274-279, 1972
7. Hourihane DO, Lessof L, Richardson PC: Hyaline and calcified pleural plaques as an index to exposure to asbestos. A study of radiological and pathological features of 100 cases with a consideration of epidemiology. *Br Med J* 1:1069-1074, 1966
8. Jones JSP, Sheers G: Pleural plaques. Biological effects of asbestos. In Bogovski P, Gilson JC, Timbrell V, et al (eds): *Biological Effects of Asbestos*. Proceedings of a Working Conference, International Agency for Research on Cancer (1972). Lyon, France, IARC Scientific Publication 8, 1973, pp 243-248
9. Kivimäki R: Pleural calcification as a roentgenologic

PLEURAL PLAQUES

9. of non-occupational endemic anthophyllite-asbestosis. *U Radiol (Suppl) (Stockh)* 194:1-67, 1960
10. Kivluoto R: Pleural plaques and asbestos: Further observations on endemic and other nonoccupational asbestosis. *Ann NY Acad Sci* 132:235-239, 1965
11. Krod L: Computerized tomography using the E.M.I. general purpose scanner. *Br J Radiol* 50:2-14, 1977
12. Langlands JH, Wallace WFM, Simpson MJ: Insulation workers in Belfast. Morbidity of men still at work. *Br J Ind Med* 28:217-223, 1971
13. Le Bouffant L, Martin JC, Durif S, et al: Structure and composition of pleural plaques, in Bogovski P, Gilon JC, Tumbrell V et al (eds): *Biological Effects of Asbestos. Proceedings of a Working Conference, International Agency for Research on Cancer (1972), Lyon, France, IARC Scientific Publication 8, 1973, pp 249-257*
14. Mattsson SB, Ringqvist T: Pleural plaques and exposure to asbestos. A clinical material from a Swedish lung clinic. *Scand J Respir Dis (Suppl)* 75:1-41, 1970
15. Meurman L: Asbestos bodies and pleural plaques in a Finnish series of autopsy cases. *Acta Pathol Microbiol Scand (Suppl)* 181:1966
16. Ochs CW, Smith JP: Chronic pleural thickening: Some observations on cause and pathogenesis. *Milit Med* 141:77-81, 1976
17. Romey R, Moskowitz M: Scleroderma, pleural calcification and reticulum cell sarcoma of the lungs. *Chest* 64:371-373, 1973
18. Rossiter CE, Bristol LJ, Cartier PH, et al: Radiographic changes in chrysotile asbestos mine and mill workers of Quebec. *Arch Environ Health* 24:388-400, 1972
19. Roux V, Studený J: Actiology of pleural plaques. *Thorax* 25:270-284, 1970
20. Sargent EN, Jacobson G, Gordonson J: The significance of bilateral pleural thickening in the diagnosis of asbestosis. (in press)
21. Sciammas FD, Sherry S, Navani S: Multiple pleural nodules. *Chest* 59:673-674, 1971
22. Selikoff IJ: The occurrence of pleural calcification among asbestos insulation workers. *Ann NY Acad Sci* 132:351-367, 1965
23. Selikoff IJ: Personal communication.
24. Sheers R, Templeton AR: Effects of asbestos in dockyard workers. *Br Med J* 3:574-579, 1968
25. Thompson JC: The pathogenesis of pleural plaques, in Shapiro, HA (ed): *Proceedings of the International Conference of Pneumoconiosis, Johannesburg, South Africa (1969), Cape Town, Oxford University Press, 1970, pp 71-174*
26. Viikari M: Ultrasound examination of pleural plaques. *Acta Radiol (Suppl)* 301:1970, pp 1-42
27. Vix VA: Extrapleural costal fat. *Radiology* 112:563-565, 1974
28. Zolev C, Burukov T, Babadjov L: Pleural asbestosis in agricultural workers. *Environ Res* 1:287-292, 1967

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DU 038351