Radiology of Asbestos Disease

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ALTHOUGH the hazards of asbestos exposure were first reported in the early 1900s, the full extent of its effects is still being evaluated. Persons with occupational exposure to asbestos have a higher incidence of pulmonary fibrosis (asbestosis), various pleural disorders (plaque, thickening, and effusion), and neoplasms (lung, pleura, and gastrointestinal). In recent years, attention has turned to asbestos as an environmental pollutant. Household contacts of asbestos workers, persons living near asbestos plants, and urban dwellers frequently have asbestos fibers in the lungs at autopsy.

The chest roentgenogram often offers the first evidence of asbestos exposure, because a history of asbestos exposure is difficult to obtain or is not sought. Therefore, it is important for both the radiologist and clinician to understand both the radiological findings of asbestos exposure and the significance of each change.

The final diagnosis of asbestosis (pulmonary fibrosis) rests on a combination of pulmonary lesions on the roentgenogram, bibasilar rales, and reduced lung function in a patient with the appropriate exposure history. Microscopic verification is usually not obtained.[1,2] The diagnosis of asbestos-related pleural disease relies more heavily on the radiological changes (see below). Roentgenograms demonstrating the typical lateral pleural changes in the absence of old trauma or empyema have a 0.81 correlation with prior exposure to asbestos.[3]

A rough correlation exists between the degree of asbestos exposure and the type of disease produced. Pulmonary fibrosis (asbestosis) and malignant neoplasms of the lung and gastrointestinal tract are most likely to develop in those individuals with the heaviest exposure. Those persons with mild to moderate exposure are more likely to contract benign or malignant pleural disease. These relationships are complex and depend on the type of fiber, pattern of exposure, and time since initial exposure. Usually, a 15- to 20-year latency period occurs between the initial exposure and the first radiological or clinical evidence of disease. Cigarette smoking potentiates both the fibrogenic and carcinogenic effects of asbestos.[3]

For both epidemiologic studies and the individual patient, good-quality 120-kV posteroanterior and lateral roentgenograms usually provide the necessary radiological information. Oblique roentgenograms may accentuate questionable pleural changes. Although computed tomography (CT) will demonstrate lesser degrees of fibrosis, pleural plaques, and pleural calcification, routine use of it hardly seems justified. The major value of CT appears to be in distinguishing focal plaques (benign) from intraparenchymal nodules (presumably cancer) and determining whether large lesions are lung masses, solid pleural lesions (presumably mesotheliomas), or focal pleural fluid collections. In patients with proven lung cancer and mesothelioma, CT is a valuable aid in determining the extent of the lesion both for planning of treatment and determining prognosis.

Asbestosis (Pulmonary Fibrosis)

The radiological changes of pulmonary fibrosis may precede, be associated with, or follow the onset of pulmonary symptoms. Unlike most other pneumoconioses (with the exception of talcosis), the predominant radiological lesions involve the lower half of the lungs. These are usually small, irregular, poorly defined linear opacities. Pinpoint opacities, thickened septal lines, gross linear stranding, and honeycombing are less frequent (Figs 1 and 2, left). (Their severity is scored on a 12-point scale adopted by the International Labor Office.) Lung volumes tend to be normal or diminished. Associated obstructive lung disease is more likely caused by smoking than by asbestos. Conglomerate masses (progressive massive fibrosis), a well-known complication of silicosis and coal workers pneumoconiosis, are extremely rare in asbestosis. When it does occur in asbestosis, it tends to be in the lower lobes, the area of predominant pulmonary fibrosis. Therefore, any focal lung density appearing in a patient with exposure to asbestos should suggest a carcinoma rather than a conglomerate mass.[1,4]

A recently recognized entity that may mimic lung cancer or the rare

Fig 1.—In this posteroanterior film of right hemithorax, reticular nodular interstitial infiltrate is present throughout lungs (asbestosis) in this patient with emphysema. Observe mild pleural thickening along lateral chest wall and minor fissure. Small calcified plaque is noted as well (black arrow). Spheroid, pleural-based mass (white arrow) was not present on previous films, proving at surgery to be adenocarcinoma involving lung, pleura, and chest wall.

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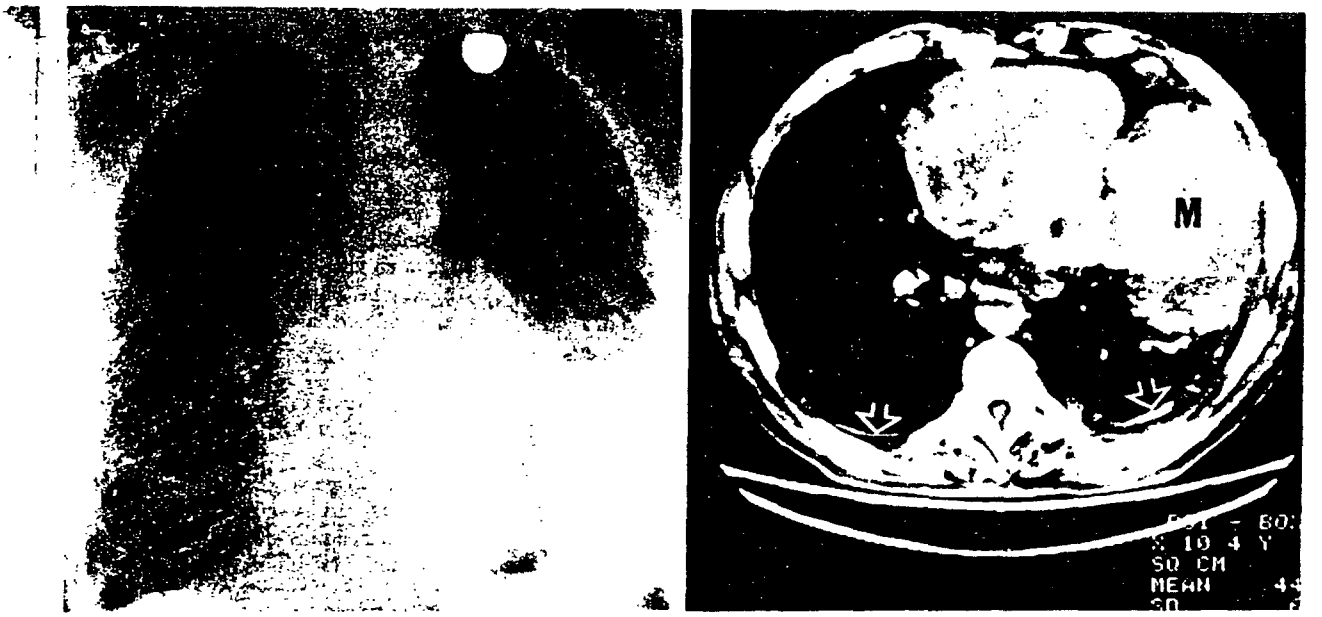

Fig 2.—Left, In this posteroanterior roentgenogram of chest, considerable dense, linear, interstitial thickening is noted in lower half of right lung (asbestosis). Costophrenic angle is blunted and pleura is thickened to level of posterior segment of third rib. On left side, upper lobe nodule as well as homogeneous opacification of lung base is present. Differential diagnosis included loculated effusion, mesothelioma, and lung cancer. Right, Computed tomographic scan through lung base demonstrates large, left lower lobe lung mass (M), bilateral pleural thickening, and calcification (arrows) (oat cell carcinoma, left lower lobe).

lower lobe progressive massive fibrosis is the pulmonary pseudotumor (rounded atelectasis). This is usually a swirl of atelectatic parenchyma adjacent to thickened pleura. Tomograms or oblique films may demonstrate a "tail" to the medial aspect of the mass, and CT may show clearly the atelectatic nature of the lesion. Thus, one can often distinguish between round atelectasis and a tumor.[13]

Lung Cancer

Asbestos and cigarette smoking are cocarcinogens. Lung cancer is 70 times more likely to develop in a smoking asbestos worker than in a nonsmoker without asbestos exposure. The malignant neoplasms are most frequently peripheral and at the bases of the lungs as opposed to the more frequent upper lobe predominance. The tumor frequently arises in a background of interstitial fibrosis or pleural disease, making detection of a small neoplasm difficult (Fig 1). Any change identified on serial films should be viewed with great suspicion, because fibrosis and plaques often take years to demonstrate radiological progression. Tomograms or CT may be helpful in distinguishing overlapping shadows, a lung nodule, or a pleural mass[1,2] (Fig 2).

Benign Pleural Disease

Pleural disease may take several guises. Asymptomatic, focal, bilateral pleural thickening along the midlateral thoracic wall are characteristic of pleural plaque due to moderate asbestos exposure. Plaques may also be discerned along the mediastinal pleura, the pericardium, and the diaphragm. They tend to spare the lung apexes and costophrenic angles. The majority are not calcified, and most are noted in the absence of pulmonary fibrosis. When calcification occurs, it is in the parietal pleura in a characteristic distribution along the surface of the diaphragm and the lateral chest wall (Figs 1 and 3). Plaques do not undergo malignant degeneration, but are an indicator of significant asbestos exposure. When these characteristic plaques are present, the vast majority of patients have a history of asbestos exposure.[3] Asbestos workers with pleural plaques, but no fibrosis, have a 2½-fold increase in lung cancer.[14]

Diffuse pleural thickening with involvement from the apex to the base may also be due to asbestos exposure. Unlike pleural plaques, this radiological appearance may be observed in a variety of diseases (Fig 2, left).

Pleural effusions are frequently harbingers of pleural or pulmonary malignant neoplasms. Any patient with a notable asbestos history and at least ten years since initial exposure should be presumed to have a malignant neoplasm until proved otherwise. However, effusions may be benign in nature. Epler et al[4] have recently shown that benign effusion was the most common asbestos-related abnormality during the *first twenty years* after exposure.

Mesothelioma

Mesothelioma is approximately three to five times more frequent in asbestos workers than in nonasbestos workers. Because it is a relatively rare tumor, however, its incidence is considerably less than lung cancer. Exposure to asbestos is often mild to moderate, and frequently a 30- to 40-year latency period ensues between initial exposure and development of the mesothelioma.[1,2]

Mesotheliomas may arise from any mesothelial surface, but the vast majority are located in the pleura or pericardium. Symptoms include the insidious onset of pain, dyspnea, and weight loss. The most frequent radiological manifestation is that of pleural effusion. The effusion is often large and may obscure the pleural

Fig 3.—Posteroanterior chest roentgenogram shows lobular pleural plaques along each lateral chest wall (arrows). Lung apexes and costophrenic angles are spared. Calcified pleural plaque is noted on left diaphragm (arrowhead). Little or no interstitial fibrosis is present (asbestosis-pleural plaques).

Fig 4.—In this posteroanterior roentgenogram of chest, large right pleural effusion obscures extensive malignant mesothelioma. Lateral portion of right third rib (upper arrows) is destroyed, and upper segment of trachea is deviated to left. Two nodules observed over left upper lobe represent calcified pleural plaques en face. Diaphragmatic, mediastinal pleura and lateral pleural calcifications are also present (lower arrows).

umor. Large effusions may cause the mediastinum to shift to the contralateral side or mediastinal pleural involvement may "freeze" the mediastinum and prevent mediastinal shift (Fig 4).

When the mesothelioma is visible, it usually appears as a lobular mass between the lung and the chest wall. It may be localized in a small area or may encase a large portion of the lung. The volume of the involved hemithorax may be diminished. Secondary invasion of the chest wall, lung invasion, or lung metastasis are frequently observed late in the course of the disease (Fig 4). Computed tomographic examination frequently discloses the tumor to be more extensive than initially appreciated. Mediastinal invasion and contralateral or subdiaphragmatic spread are frequently demonstrated by CT examination.

Nonoccupational Lung Disease

Concern for the effects of asbestos on the general population arises from multiple sources. Families of asbestos workers have a higher incidence of asbestos-related lung disease. The air in most urban areas contains small numbers of asbestos particles. Autopsies of adults in urban centers frequently show asbestos bodies in the lung, although at a much lower concentration than in asbestos workers.[1] The disease-producing potential of inhalation of asbestos in a low concentration in the general population is still unknown. It has been suggested that the lateral pleural thickening occasionally observed in nonasbestos workers may be related to low levels of asbestos exposure.[1]

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