

Exposure to asbestos is not limited to persons who directly handle the material. Cases of asbestos-related diseases have been encountered in individuals with only bystander exposure, such as painters and electricians who worked alongside insulation workers in a shipyard. Community exposure resulted from the use of asbestos-containing mine and mill tailings as landfill, road surface, and playground material (e.g., Libby, MT, the site of a vermiculite mine in which the ore was contaminated with asbestos). Finally, exposure can occur from the disturbance of naturally occurring asbestos (e.g., from increasing residential development in the foothills of the Sierra Mountains in California).

Asbestos has largely been replaced in the developed world with synthetic mineral fibers such as fiberglass and refractory ceramic fibers, but it continues to be used in the developing world. The major health effects from exposure to asbestos are pleural and pulmonary fibrosis, cancers of the respiratory tract, and pleural and peritoneal mesothelioma.

*Asbestosis* is a diffuse interstitial fibrosing disease of the lung that is directly related to the intensity and duration of exposure. The disease resembles other forms of diffuse interstitial fibrosis (Chap. 315). Usually, exposure has taken place for at least 10 years before the disease becomes manifest. The mechanisms by which asbestos fibers induce lung fibrosis are not completely understood but are known to involve oxidative injury due to the generation of reactive oxygen species by the transition metals on the surface of the fibers as well as from cells engaged in phagocytosis.

Past exposure to asbestos is specifically indicated by pleural plaques on chest radiographs, which are characterized by either thickening or calcification along the parietal pleura, particularly along the lower lung fields, the diaphragm, and the cardiac border. Without additional manifestations, pleural plaques imply only exposure, not pulmonary impairment. Benign pleural effusions also may occur. The fluid is typically a serous or bloody exudate. The effusion may be slowly progressive or may resolve spontaneously.

Irregular or linear opacities that usually are first noted in the lower lung fields are the chest radiographic hallmark of asbestosis. An indistinct heart border or a “ground-glass” appearance in the lung fields may be seen. HRCT may show distinct changes of subpleural curvilinear lines 5–10 mm in length that appear to be parallel to the pleural surface (Fig. 311-1).

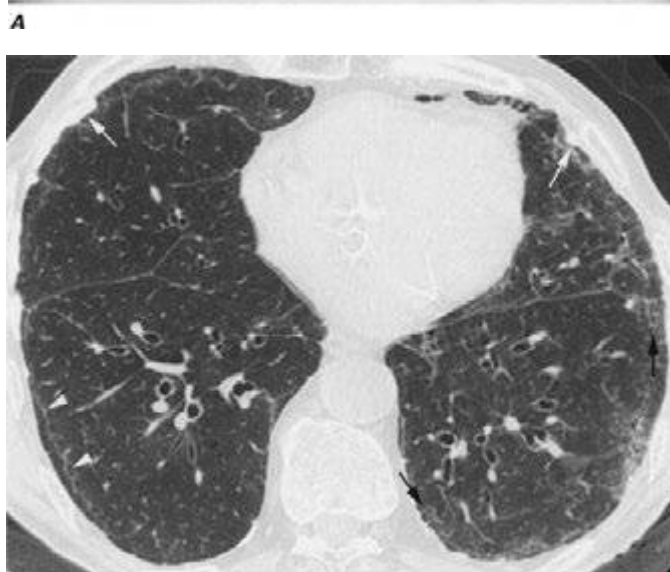
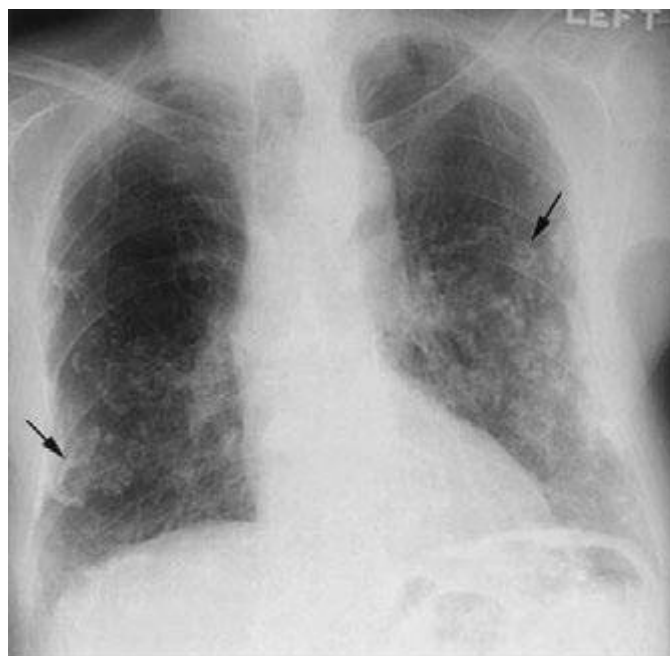
Pulmonary function testing in asbestosis reveals a restrictive pattern with a decrease in both lung volumes and diffusing capacity. There may also be evidence of mild airflow obstruction (due to peribronchiolar fibrosis).

Because no specific therapy is available for asbestosis, supportive care is the same as that given to any patient with diffuse interstitial fibrosis of any cause. In general, newly diagnosed cases will have resulted from exposures that occurred many years before.

*Lung cancer* (Chap. 107) is the most common cancer associated with asbestos exposure. The excess frequency of lung cancer (all histologic types) in asbestos workers is associated with a minimum latency of 15–19 years between first exposure and development of the disease. Persons with more exposure are at greater risk of disease. In addition, there is a significant interactive effect of smoking and asbestos exposure that results in greater risk than what would be expected from the additive effect of each factor.

*Mesotheliomas* (Chap. 316), both pleural and peritoneal, are also associated with asbestos exposure. In contrast to lung cancers, these tumors do not appear to be associated with smoking. Relatively short-term asbestos exposures of  $\leq 1$ –2 years, occurring up to 40 years in the past, have been associated with the development of mesotheliomas (an observation that emphasizes the importance of obtaining a complete environmental exposure history). Although the risk of mesothelioma is much less than that of lung cancer among asbestos-exposed workers, over 2000 cases were reported in the United States per year at the start of the twenty-first century.

Because epidemiologic studies have shown that >80% of mesotheliomas may be associated with asbestos exposure, documented mesothelioma in a patient with occupational or environmental exposure to asbestos may be compensable.



**FIGURE 311-1 Asbestosis.** **A.** Frontal chest radiograph shows bilateral calcified pleural plaques consistent with asbestos-related pleural disease. Poorly defined linear and reticular abnormalities are seen in the lower lobes bilaterally. **B.** Axial high-resolution computed tomography of the thorax obtained through the lung bases shows bilateral, subpleural reticulation (black arrows), representing fibrotic lung disease due to asbestosis. Subpleural lines are also present (arrowheads), characteristic of, though not specific for, asbestosis. Calcified pleural plaques representing asbestos-related pleural disease (white arrows) are also evident.

### SILICOSIS

Despite being one of the oldest known occupational pulmonary hazards, *free silica* ( $\text{SiO}_2$ ), or crystalline quartz, is still a major cause of disease. The major occupational exposures include mining; stonecutting; sand blasting; glass and cement manufacturing; foundry work; packing of silica flour; and quarrying, particularly of granite. Most often, pulmonary fibrosis due to silica exposure (silicosis) occurs in a dose-response fashion after many years of exposure.

Workers heavily exposed through sandblasting in confined spaces, tunneling through rock with a high quartz content (15–25%), or the manufacture of abrasive soaps may develop acute silicosis with as little as 10 months of exposure. The clinical and pathologic features of acute silicosis are similar to those of pulmonary alveolar proteinosis