

weighed 1,000 gm together and their pleural surfaces were smooth, gray, and emphysematous. Blebs were not present. In the lateral superior region of the upper lobe of the left lung was a subpleural spherical mass measuring 1.5 cm in diameter. This was covered by umbilicated pleural surface.

Microscopically, the pulmonary alveolar spaces as well as the bronchial lumina contained scattered clusters of pigment-laden macrophages and a few asbestosis bodies. Emphysema was manifest by stretched, thinned, and broken alveolar septa creating small cystic spaces. There was perivascular, peribronchial, and nodular interstitial fibrosis. The fibrotic areas contained a moderate amount of anthracotic pigment and numerous typical asbestosis bodies.

Microscopically, the mass in the upper lobe of the left lung was a tumor which originated in an ectatic bronchus. It extended in a haphazard and stellate fashion into the adjacent pulmonary parenchyma. The architecture was comprised of immature and atypical squamous cells among which keratin and intracellular bridges were not demonstrable. The nuclei were large and vesicular; some were of relatively gigantic proportions with irregularly clumped chromatin and distinct, wrinkled nuclear membranes. The cytoplasm ranged from colorless to finely granular and amphophilic. Typical asbestosis bodies were numerous both in and about the tumor mass (Fig 3).

Pathology of Asbestosis

The crystalline asbestos fibers become wedged in the lumens of the respiratory bronchioles, and with the inspiratory narrowing-elongation and expiratory widening-shortening of these tubes through each respiratory cycle the fibers are worked into the walls of the bronchioles. This mechanical irritation accounts for the fibrosis, which at first is peribronchiolar. As the disease progresses, irregular diffuse fibrosis occurs throughout the pulmonary tissues with effacement of alveoli in the areas of fully developed disease. Ultimately, the reactive tissue interferes with lymphatic drainage of the lung and stasis becomes an additional factor predisposing fibroplasia. Increasing flow of lymph toward the subpleural lymphatic network tends to produce irritation of the pleura with attendant pleural thickening and adhesions to the parietal pleura.

The "ground glass" appearance of the lung shadows in films of patients with asbestosis may in part be due to diffuse parenchymal

fibrosis and in part to the pleural thickening; in advanced cases pleural-pericardial adhesions and fibrosis may become so pronounced that a characteristic "porcupine-heart shadow" results.²

Concomitant with the development of fibrosis, distortion and emphysematous enlargement of many alveoli become prominent. Bronchitis and bronchiolitis become chronic and often lead to bronchiectasis. The dependent portions of the lungs are usually more profoundly affected than the upper halves. This serves as a gross diagnostic point in differentiating asbestosis from the more widespread and nodular alterations of silicosis.

Asbestosis bodies are essential to the microscopic diagnosis of the condition. Some of these structures may lie free in alveoli, but most of them are embedded in fibrous tissues of the walls of alveoli or bronchioles. A few may be surrounded by giant cells of foreign body type. These bodies are comprised of asbestos fibers surrounded by albuminous material, calcium salts, and iron salts. The ends are usually knobbed, while the central regions may be notched. The color is yellow to pinkish red in the usual histologic preparations. A few are as small as 1μ in length but the majority are 10μ to 60μ long.

Clinical Findings

Dyspnea is usually the first sign of disease, and it seldom occurs with an industrial exposure to asbestos dusts of less than five years. Loss of weight is often marked. Dry cough is common, and more than half of the patients expectorate small amounts of blood-streaked sputum sometime during their illness. Asbestosis bodies may be found in the sputum of most patients by microscopic examination.

The chest becomes emphysematous, and fine crackling rales are present over the lower lobes. Cyanosis and clubbing of the nails are frequently present in patients with moderately advanced disease, and cor pulmonale is often a late complication. Asbestosis does not predispose to tuberculosis, differing in this respect from silicosis.³