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Pulmonary Asbestosis : Radiologic-Pathologic Brief Report

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Pulmonary asbestosis is defined as bilateral diffuse interstitial fibrosis of the lungs caused by exposure to asbestos. Many occupations are at risk for asbestos exposure, particularly in the mining, milling, manufacturing, construction, shipbuilding, and automotive industries. Therefore, the prevalance of asbestosis should be fairly widespead. The diagnosis of asbestosis can be made on either clinical or pathological grounds. We recently encountered one case of asbestosis which was confirmed histologically. On HRCT, there was ground- glass opacity with irregular linear shadows, subpleural curvilinear lines and parenchymal bands. Neither plaque nor calcification were noted. The histologic findings observed on open- lung biopsy specimen were well in accord with those in HRCT. Many asbestos-coated bodies were present along with black dust.

Key Words: Asbestosis, computed tomography(CT), high-resolution

Asbestosis is defined as bilateral diffuse interstitial fibrosis of the lungs as a result of the accumulation of airborne asbestos in the lungs (Churg and Green., 1988). There are a variety of workers who may be occupationally exposed to asbestos, for example in the mining, milling, manufacturing, construction, shipbuilding, and automotive industries. Therefore, the prevalance of asbestosis should be fairly widespread. Asbestosis is diagnosed pathologically by the detection of ferruginous bodies or asbestos fibers, but their detection is difficult. The diagnosis of asbestosis is then achieved clinically, based on criteria adopted by the American Thoracic Society in 1986 (American Thoracic Society, 1986).

To our knowledge, there has been no case study of histologically-proven asbestosis in Korea. We recently experienced a case of asbestosis which was confirmed by open-lung biopsy in a patient with a past history of occupational exposure to asbestos.

CASE REPORT

A 60 year old Korean man was admitted to hospital because of exertional dyspnea and productive cough. The patient had been fine until five years before, when dyspnea developed along with a cough that produced small amounts of sputum. Medical treatment had been given without benefit. He had smoked one pack of cigarettes a day for 25 years.

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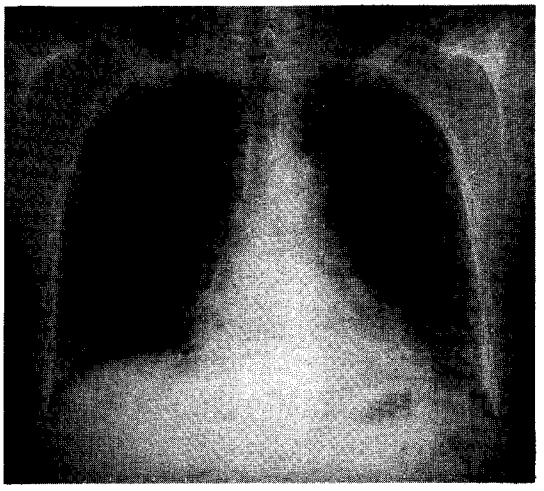


Fig. 1. Chest PA. There are patchy increased opacities and linear shadows at both lower peripheral lung zones.

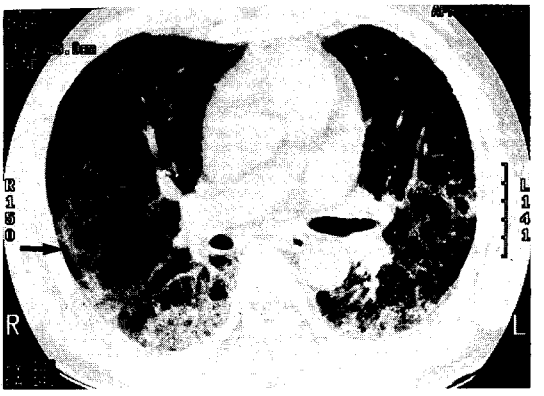
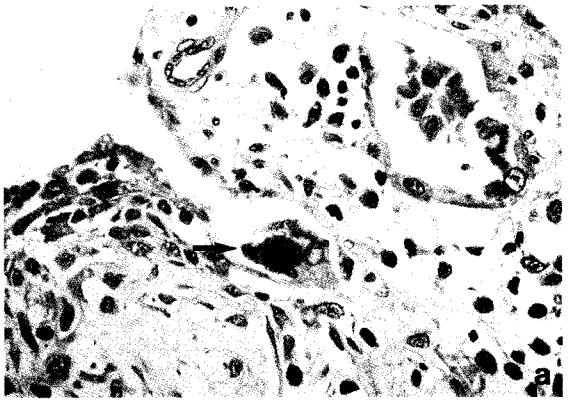
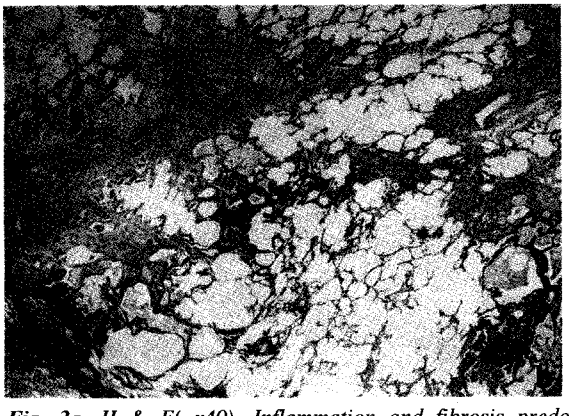


Fig. 2. HRCT at the level of the intermediate bronchus. Parenchymal band (arrow) and ground glass opacities with irregular linear shadows are seen in both lungs.





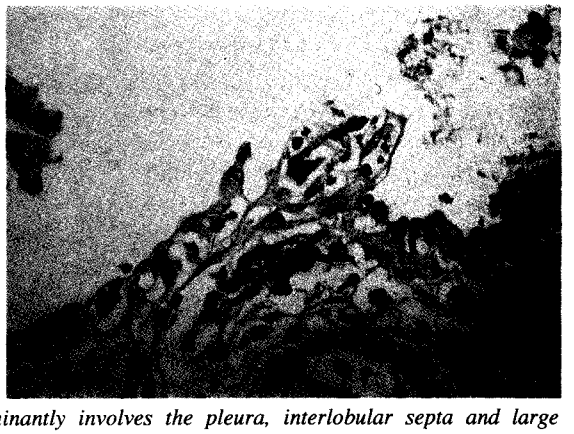


Fig. 3a. *H & E(x40). Inflammation and fibrosis predominantly involves the pleura, interlobular septa and large bronchovascular structures.*

Fig. 3b. *H & E (x400). Asbestos bodies (arrow) with clear, straight and colorless cores are seen in fibrotic tissue associated with black mineral dust.*

Fig. 3c. *H & E (x400). Hyperplastic pneumocytes are seen containing flocculent hyaline material in the cytoplasm (arrow).*

He had worked at a brake-lining company that manufactured brake linings using chrysotile, a type of asbestos fiber, for 20 years. On admission, dry rales were heard in both lower lung fields on auscultation. On physical examination, there were regular heart beats with no murmurs. On arterial blood gas analysis, pH was 7.432, PO2 was 80 mmHg, PCO2 was 42.7 mmHg, HCO3 was 28 mmol/L and O2 saturation was 96%. On pulmonary function test, FVC was 2.8 L (78% predicted), FEV1 was 2.20L (84% predicted), FEV1/FVC was 79% (108% predicted), DLco was 19.0 ml/min/mmHg (101% predicted). Plain chest radiograph showed a patchy distribution of irregular lines and ground-glass haziness predominantly in both lower peripheral lung zones (Fig. 1). On HRCT, ground-glass opacity with irregular linear shadows, subpleural curvilinear lines, and parenchymal bands were noted (Fig. 2). But pleural plaque or calcification could not be ascertained. A thoracoscopic wedge biopsy of the lateral basal segment of the left lower lobe was performed.

The biopsied lung measured 4 cm × 5 cm × 2 cm. Cobblestoning of pleura was present to a slight degree. On section, there was subpleural honeycombing in the background of the fibrotic parenchyme. Microscopically, the lung showed a peripheral acinar pattern of interstitial fibrosis and inflammation alternating with relatively normal alveoli. The inflammation and fibrosis predominantly involved the pleura, interlobular septa and larger bronchovascular structures, and therefore resembled idiopathic pulmonary fibrosis (Fig. 3a). Peribronchiolar fibrosis was not so obvious. Asbestos bodies with a clear, straight and colorless core were seen in fibrotic tissue mixed with black mineral dust (Fig. 3b). The honeycombed area showed much more severe inflammation, as well as extensive fibrosis. Although the focal extension of visceral pleural fibrosis involved subpleural peripheral lung tissue, the visceral pleura was thickened diffusely enough to be diagnosed as visceral pleural fibrosis. A small number of hyperplastic pneumocytes in the areas of fibroblast foci were seen to contain flocculent hyaline material in the cytoplasm, which was initially described as cytoplasmic hyaline in asbestosis (Fig. 3c).

DISCUSSION

Asbestosis is one of the non-neoplastic asbestos-related disorders which also include pleural plaque, pleural effusion, diffuse pleural thickening and fibrotic masses (Staples, 1992). Asbestosis can be defined as pneumoconiosis characterized by more-or-less diffuse parenchymal interstitial fibrosis secondary to the inhalation of asbestos fibers Fraser *et al.* 1990. Churg stated that diffuse interstitial fibrosis is the only process to which the term asbestosis should be applied (Churg and Green., 1988). Asbestos is a group of minerals that are fibrous in nature and are resistant to high temperatures and various chemical insults. They are divided into two major groups, the serpentines, which include chrysotile, and the amphiboles, which include amosite, crocidolite, anthophyllite and actinolite. Chrysotile is the most important form commercially, accounting for about 90% of the total asbestos marketed worldwide (Craighead *et al.* 1982). Chrysotile tends to curl and possibly to dissolve slowly, whereas amphiboles are relatively straight fibers that are extremely stable in the lung. There are differences which appear to influence both the deposition pattern and propensity to cause specific diseases. Asbestos minerals are valuable because of their high tensile strength, high heat resistance, relative chemical resistance and ability to be woven into cloth. These properties vary with different types of asbestos, and the fiber type is to some extent selected for commercial use by these properties. The patient in this report worked for 20 years at a brake-lining production company that used chrysotile. After inhalation, asbestos fibers are first deposited in the respiratory bronchioles and alveolar ducts, and with prolonged and extensive exposure, they also accumulate subpleurally (Aberle *et al.* 1988). Fibrosis occurs in the peribronchiolar region of the lobular core. As fibrosis progresses, it involves the alveolar walls throughout the lobule and, eventually, the interlobular septa. Honeycombing can be seen in advanced cases (Craighead *et al.* 1982; Akira *et al.* 1990). HRCT findings are well correlated pathologically (Akira *et al.* 1990; Webb

et al. 1996). Thickened intralobular lines on HRCT are peribronchiolar fibrosis. Thickened interlobular lines on HRCT are interlobular fibrotic thickening or edematous thickening. The pleural-based opacities on HRCT are subpleural fibrosis. The parenchymal bands on HRCT are fibrosis along the bronchovascular sheath or interlobular septa, with distortion of the parenchyma. The ground glass appearance on HRCT is mild alveolar-wall and interlobular thickening by fibrosis or edema. And subpleural curvilinear lines are peribronchiolar fibrotic thickening combined with flattening and collapse of the alveoli due to fibrosis (Akira *et al.* 1990). However, none of these findings are specific for asbestosis (Bergin *et al.* 1994). The general microscopic requirements for diagnosis are diffuse interstitial fibrosis and the presence of asbestos bodies in ordinary 5-micron sections. In well-established disease, the interstitial process appears as dense interstitial fibrosis with a chronic inflammatory infiltrate (Churg and Green., 1988). The interstitial process is typically patchy, similar to the pattern of UIP (usual interstitial pneumonia)

In this case, ground-glass opacity, irregular linear shadows, subpleural and septal lines, and parenchymal bands on HRCT were seen, but these findings were similar to HRCT features of usual interstitial pneumonia (UIP). Therefore, the differential diagnosis between asbestosis and UIP could not be done. But fibrosis around the respiratory bronchioles, alveolar ducts and alveolar walls, as well as microcystic honeycombing, were all noted histologically. Although pleural plaque was not seen in this case on HRCT, its absence does not necessarily preclude asbestosis (Bergin *et al.* 1994). Asbestos bodies were demonstrated in this case and therefore the diagnosis of asbestosis was not problematic. Asbestosis can be diagnosed when interstitial fibrosis is associated with intrapulmonary asbestos bodies or astestos fibers in asbestos-exposed patients (Craighead, 1982).

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