muscle disorders of the lung into three large groups: leiomyomatosis in women, metastatic leiomyoma in men and children, and multiple pulmonary fibroleiomyomatous hamartomas. Our patient had a history of hysterectomy for uterine myoma and thus is considered pulmonary leiomyomatosis.

Bronchial fibroleiomyoma may extend along a bronchial lumen for a considerable distance as a tongue of soft, grayish tumor tissue. There have been no reports to our knowledge of endobronchial extension in pulmonary leiomyomatosis. Some of the tumors in the present case showed endobronchial extension and caused respiratory failure. Polypoid lesions were covered with squamous cells or stratified columnar epithelium. Uterine myoma originating from endometrium occasionally showed a polypoid appearance and is called fibroid. The endobronchial extension seen in the present case may mimic growth of the earlier uterine myoma.

Fibroleiomyomatous tissues were observed in the wall of a large cyst in the right lower lobe. Multiple nodules of the right lung showed a histologic pattern similar to those of the tumors in the left lung. Some nodules formed small cysts that were histologically identical with the large cyst. Small cysts are usually seen microscopically in pulmonary leiomyomatosis. Such a large cyst of more than 10 cm has not been reported in this disorder. During thoracotomy, deflated small cysts were inflated on ventilation. That suggested that those cysts communicated with the airway. Large cysts may be formed under a check valve mechanism of the communicating airway.

Becker et al. recommended regular follow-up examinations rather than surgical intervention after establishment of the diagnosis. Hormone therapy—e.g., oophorectomy, progestrone, tamoxifen—is effective and seems to be the treatment of choice. However, as in the present case, giant cyst formation may occur and compress the neighboring healthy lung, and endobronchial extension of the tumor may also obstruct a major airway, causing respiratory failure. In such circumstances, surgical treatment may be necessary.

REFERENCES


Chronic Constrictive Pericarditis Associated with Asbestosis*

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Observation of chronic constrictive pericarditis associated with pulmonary asbestosis is reported here. Heart catheterization revealed typical patterns of cardiac constriction. The diagnosis of asbestosis was based on pathologic features associated with a long history of asbestos dust exposure without evidence for tuberculosis. The etiology of this constrictive pericarditis was related to an asbestosis pericarditis involvement. (Chest 1988; 94:646-47)

Exposure to asbestos dust is associated with an increased incidence of pulmonary complications such as asbestosis, pleural mesothelioma and bronchogenic carcinoma. Other localizations are exceptional, even though asbestos bodies may easily be found outside the respiratory area. This article describes calcified pericarditis which led to diastolic constriction in a patient suffering from asbestosis.

CASE REPORT

An 82-year-old man was hospitalized in 1984 with symptoms of cardiac insufficiency. One year previously, carcinoma of the left upper lobe of the lung had been discovered but was not treated due to the patient's advanced age. The patient's past history revealed occupational exposure to asbestos dust (the patient worked in boiler construction for 38 years). Exposure to asbestos stopped in 1962.

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FIGURE 1. The pulmonary radiography reveals calcifications of pericardial and diaphragmatic pleurae.
No history of tuberculosis was found.

On admission to the hospital, clinical examination revealed arterial pressure of 130/80 mm Hg, protodiastolic vibration, peripheral edema of the lower limbs, pulsatile hepatomegaly and rales in the lower part of the lungs. Electrocardiogram showed global microvoltage, diffuse ventricular repolarization troubles and complete arrhythmia due to atrial fibrillation. Pulmonary radiography showed heterogenous opacity of the left lung and, above all, calcifications of pericardial and diaphragmatic pleurae (Fig 1). Tomodensitometric scanning showed extension of the calcifications (Fig 2). Echocardiography confirmed pericardial thickening and showed dilatation of the inferior vena cava.

Heart catheterization documented adiastolia ("dip-plateau") extending to the left side of the heart, right atrial pressure 20 mm Hg, right ventricular pressure 49/12 to 22 mm Hg, pulmonary artery pressure 42/25 to 32 mm Hg and pulmonary wedge pressure 21 mm Hg. PPD skin test and sputum culture results of mycobacteria were negative. Due to the high surgery risk because of the patient’s age, no pericardiectomy was carried out and diuretic treatment was started. In 1966, the patient was hospitalized again with a sudden hemiplegia which was followed by coma, rapidly leading to death.

Autopsy revealed, on one hand, an adenocarcinoma of the left upper lobe of lung and, on the other hand, fibrohyaline plaques covering the diaphragmatic pleura and extending to the anterior side of the liver and pericardium (the pericardium was thick and calcified). Moderate interstitial fibrosis was found only in the lower lobes of the lungs. There was no mesothelioma. Coronary arteries were atheromatous but without any significant stenosis. Asbestos bodies were present in the pulmonary parenchyma and absent in pericardial plaques. No sign of tuberculosis was found.

**DISCUSSION**

The association of a high level of asbestos dust exposure to pathologic features such as pulmonary interstitial fibrosis, pleural calcification and presence of asbestos bodies is sufficient to establish the diagnosis of pulmonary asbestosis.\(^3\)\(^4\) During exposure to asbestos dust, secondary pericardial symptoms have been described. These are either of an occasionally calcified (but not compressive) fibrosis of the pericardium\(^5\)\(^6\) or of a pericardial mesothelioma.\(^7\) Pericardial calcification is rare (1.7 percent of Yazicioglu’s asbestosis cases\(^8\)) but no constrictive pericarditis has, to our knowledge, been reported.\(^9\)

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**Thymoma Displaying Endobronchial Polypoid Growth**

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Two rare cases of invasive thymoma with prominent endobronchial polypoid growth are presented. Chief complaints of both patients, women aged 56 and 62 years, were blood streaked sputum and cough. A large mass shadow located in the mediastinum and lung field was seen on chest x-ray film and an endobronchial polyp was found by bronchoscopy in each patient. Histologic features of the bronchial biopsy specimens were highly suggestive of thymoma. The tumors resected surgically exhibited a unique manner of extension, in which the lumina of bronchi of various sizes were occupied and obliterated by a part of the tumor contiguous to the mediastinum. These tumors were diagnosed histologically as invasive thymoma, predominantly epithelial type, and mixed lymphoeytic and epithelial type. (Chest 1988; 94:647-49)

The manners of extension of thymomas are direct invasion to neighboring structures in the thorax (invasive thymoma) and distant metastasis (malignant thymoma), the latter being rare.\(^10\) Endobronchial invasion with polypoid growth, however, is a rare mode of extension of thymoma.

We experienced two cases of invasive thymoma with an...