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Inflammatory Pseudotumor of the Lung in a Child with Mycoplasma Pneumonia

Sung Hye Park, Ghee Young Choe, Chul Woo Kim, Je G. Chi, and Sook Hwan Sung*

Departments of Pathology and Thoracic Surgery Seoul National University Children's Hospital
Seoul National University College of Medicine*

A case of inflammatory pseudotumor of the lung occurring in a six-year-old boy is reported with clinicopathologic findings, including its ultrastructure. The patient had had frequent upper respiratory tract infections, and one and half year before the discovery of the lung mass, he suffered from pneumonia of the right lung, which was serologically proven to be a mycoplasma pneumoniae infection. Exploratory thoracotomy revealed a large mediastinal mass that was removed together with the right middle and lower lobes of the lung. The mass arose from the lung with an endobronchial element. Microscopically, the mass was composed of a variety of inflammatory and mesenchymal cells, including plasma cells, histiocytes, lymphocytes, and fibroblast-like spindle cells. Ultrastructurally, the spindle-shaped mesenchymal cells were either fibroblasts or myofibroblasts. At the time of diagnosis of the inflammatory pseudotumor of the lung, the serum titer of antimycoplasma antibody rose again, and the lung parenchyma adjacent to the mass showed interstitial pneumonia with features of bronchiolitis obliterans. The present case suggests that the inflammatory pseudotumor of the lung could be a postinflammatory lesion associated with mycoplasma pneumoniae infection.

Key Words: *Inflammatory pseudotumor, Lung, Childhood, Mycoplasma pneumoniae*

INTRODUCTION

Inflammatory pseudotumor of the lung is a nonneoplastic pulmonary mass lesion of unknown etiology. It remains as a poorly understood lesion, even though many reports have been published since the initial reports of Brunn (1939) and Umiker, and Iverson (1954) in which clinical characteristics and pathological features are well delineated. Several theories have been suggested, but none has been proven. However, the majority of investigators regard this entity as a variant of an inflammatory repair process rather than a true neoplastic process.

Reprint request to *Dr. Je G. Chi, Department of Pathology Seoul National University children's Hospital, 28 Yongon-dong, Chongno-Gu, Seoul 110-744, Korea*

An inflammatory pseudotumor of the lung is composed of a variety of inflammatory and mesenchymal cells, including plasma cells, histiocytes, mast cells, lymphocytes, and spindle-shaped mesenchymal cells (Carter and Eggleston, 1974). A number of synonyms such as plasma cell granuloma and histiocytoma, depending upon the prominent cell present, have been used to describe the lesion, thus adding confusion to its terminology.

We present a case of inflammatory pseudotumor of the lung occurring in a child, who had previous episodes of mycoplasma pneumoniae infection.

CASE HISTORY

This six-year-old boy was transferred to the Department of Pediatrics of Seoul National University Children's Hospital due to alleged right posterior mediastinal mass. One week before this admission,

cough, high fever of 40°C, and vomiting developed. At a local clinic, he received symptomatic treatment. The symptoms were transiently relieved but reappeared. The chest X-ray revealed a right pulmonary mass.

He was born via normal full term spontaneous delivery with a birth weight of 3kg. He had suffered from frequent upper respiratory tract infections. One-and-a-half years before this admission, he was admitted for five days under the impression of mycoplasma pneumoniae infection. At that time, cold agglutinin against mycoplasma was 1:32. Antimycoplasma antibody was positive at 1:40, and 13 days later it was 1:80. The chest X-ray showed dense parenchymal infiltration in the right lower lung field without pleural effusion or hilar lymphadenopathy. He was managed with Na-penicillin 700,000U intravenously, and the follow-up serum antimycoplasma antibody was negative.

On physical examination at admission on April 20, 1990, he was alert but had a thin, ill-looking appearance. The peripheral blood count revealed Hgb 10.5gm/100ml, Hct 36.6%, and white blood cells 25400/cmm (poly 84.9%, lymph 9.6%). The platelet count was 51,500/cmm. The arterial gas analysis was pH 7.39, pCO2 27.0mmHg, pO2 99mmHg, HCO3- 16mmol/l. C-reactive protein was 5 positive. The serum antimycoplasma antibody was positive (1:40). No infectious agent was found despite several sputum smears and culture study. Other laboratory data were within normal limits.

The chest CT showed a soft tissue-density mass in the right posterior mediastinum with dense irregular-shaped calcification (Fig. 1). A small amount of pleural effusion in the right thorax was seen. Collapse of the right middle and lower lobes and anterior deviation of the inferior vena cava and right atrium were also noted. Abdominal CT revealed no abnormality. Aspiration cytology of the mass showed numerous neutrophils and bland-looking fibroblast-like spindle cells. The pathologic report of cytology suggested inflammatory lesion.

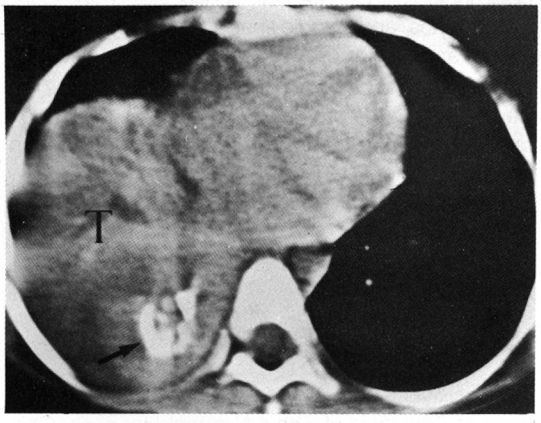

Fig. 1. Chest Ct shows a large tumor mass (T) in the right posterior mediastinum with an area of dense irregular calcification (arrow).

Exploratory thoracotomy was done, and a large solitary mass was found located in the lung parenchyma and protruding to the mediastinum. The middle and lower lobes showed consolidation and atelectatic change. The mass was adhering to the esophagus and pericardium. The mass, together with the right middle and lower lobes, was resected. The middle main bronchial lumen was filled with a whitish solid mass.

Postoperatively, the patient remains in good condition.

PATHOLOGY

The lobectomized lung including mass was handled as follow. Multiple sections from the lung mass and adjacent parenchyma were fixed in 10% formalin, processed routinely, and embedded in paraffin. The hematoxylin eosin stain and several special stains, such as Masson trichrome, reticulin, elastic, toluidine blue, Congo red, and iron hematoxylin stains, were performed. The pieces from the lung mass were fixed for four hours in 2.5% glutaraldehyde solution buffered with 0.1M phosphate. These parts were postfixed for two hours in 1% osmium tetroxide, dehydrated, and embedded in Epon. Thin sections of lung mass were stained with uranyl acetate followed by lead citrate and were examined under the electron microscope (Hitachi H-600).

Grossly, the resected mass was located in the right middle lobe of the lung, approximately three-fourths of which grew outside the lung. The mediastinal aspect of the mass showed a smooth glistening appearance covered with visceral pleura (Fig. 2). On section, a gritty sensation was felt. The mass was connected to the right middle bronchus with a polypoid endobronchial lesion occluding one-third of the lumen. It was blandly cirumscribed to the lung (Fig. 3) and was firm-to-hard in consistency. The cut surface was gray-tan and slightly trabeculated with multifocal calcification.

Microscopically, the mass was composed of interlacing bundles of bland-looking spindle-shaped mesenchymal cells, focally resembling storiform pattern, hyalinizing stroma, along with collections of plasma cells, lymphocytes, and histiocytes (Fig. 4, 5, 6).

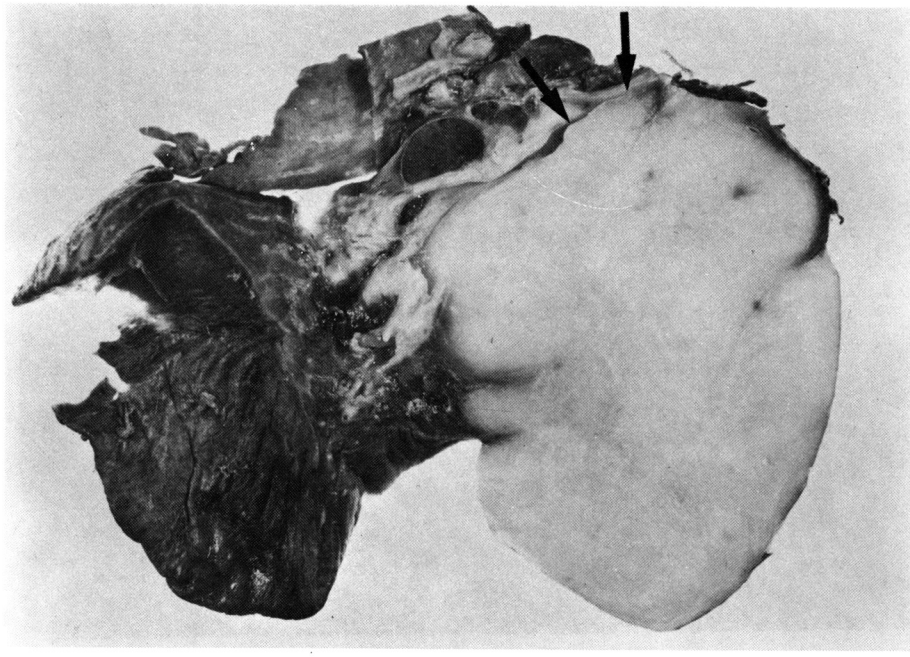

Fig. 2. The mass in the right middle lobe of the lung grew out of the lung. The mass was connected to the right middle lobe bronchus forming a endobronchial lesion (arrows). The cut surface of the mass is gray-tan and slightly trabeculated with calcified speckles.

Fig. 3. In this low-power picture, the mass (lower portion) blends into the surrounding lung tissue without forming a capsule. (H&E, ×1).

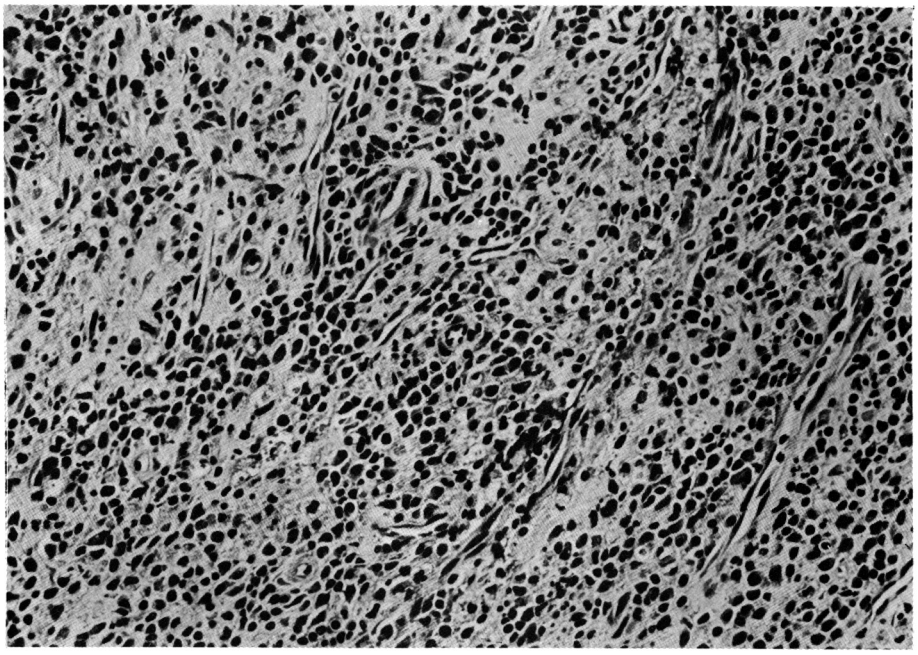

Fig. 4. Microscopically, the cellular area of the mass shows mixed inflammatory cell infiltration, especially plasma cells with capillary proliferation resembling granulation tissue (H&E, x200).

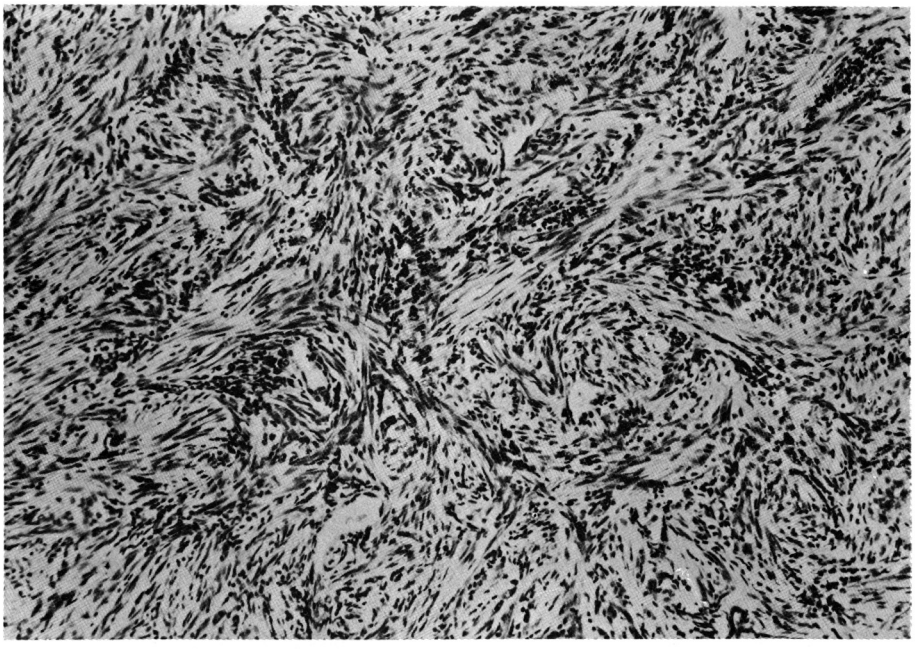

Fig. 5. Some area of the mass shows interlacing bundles of spindles cells, resembling storiform pattern with scattered and patchy collections of plasma cells, lymphocytes, and histiocytes (H&E, ×100).

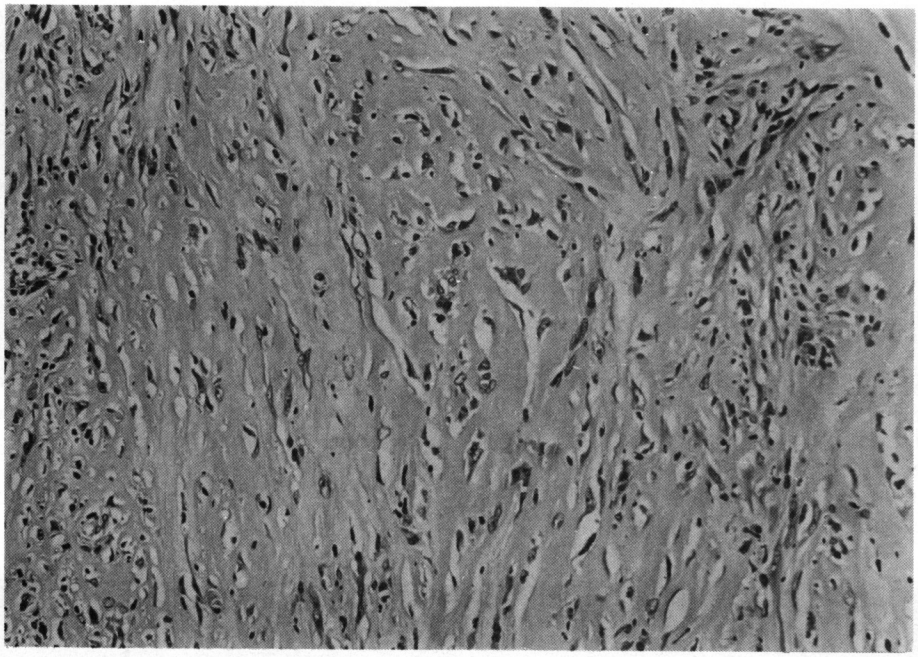

Fig. 6. Another area of the mass shows hyalinization of the stroma (H&E, ×100).

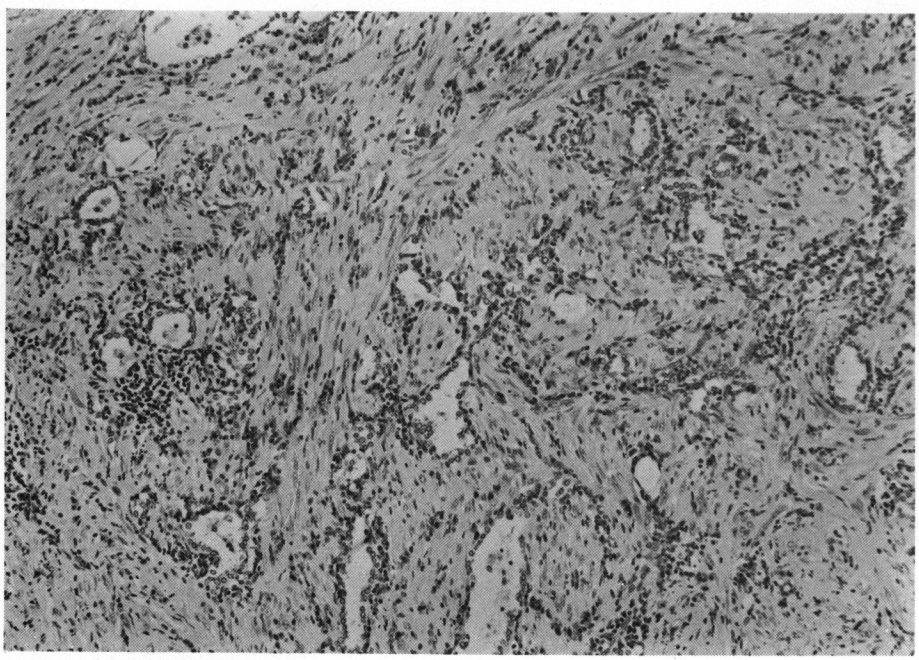

Fig. 7. Some area of the mass shows entrapped alveioli lined by type II pneumocytes. There is perialveolar plasma cell infiltration (H&E, x100).

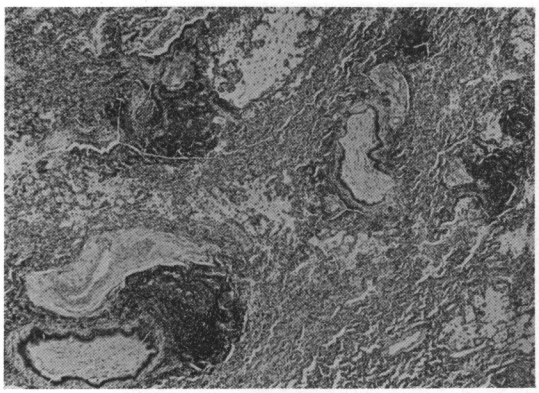

Fig. 8. The lung parenchyme of the middle lobe shows a lymphoid aggregation with germinal center formation along the bronchial trees and perivascular area. The bronchioles are dilated and filled with mucinous material (H&E. x40).

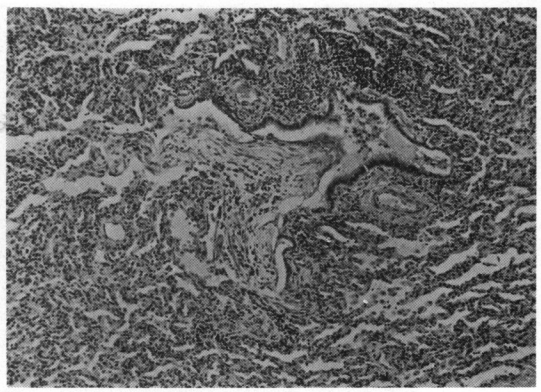

Fig. 9. The features of bronchiolitis obliterans are seen in the remaining portion (H&E x100).

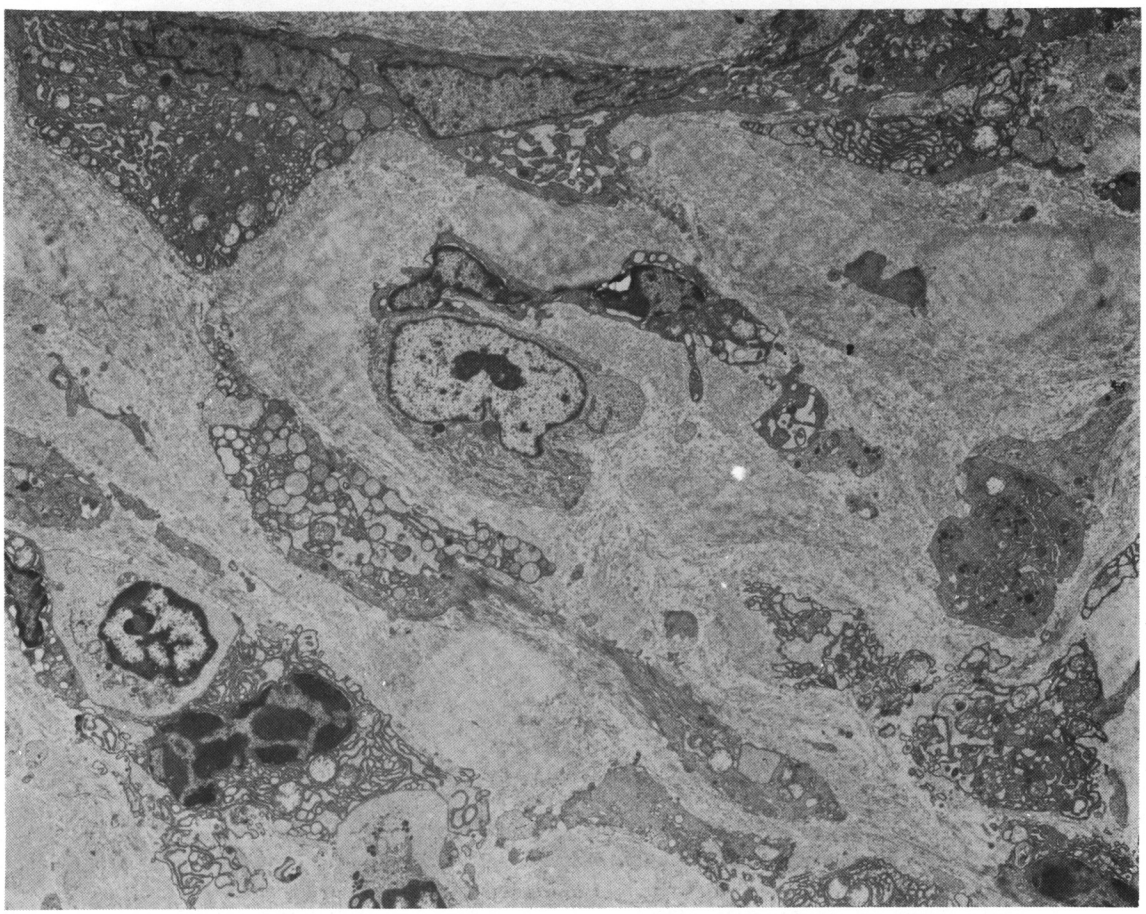

Fig. 10. Ultrastructurally, the mass is composed of fibroblasts, myofibroblasts, plasma cells, histiocytes, and lymphoid cells in collagenous background. (Uranyl acetate and lead citrate, ×3750).

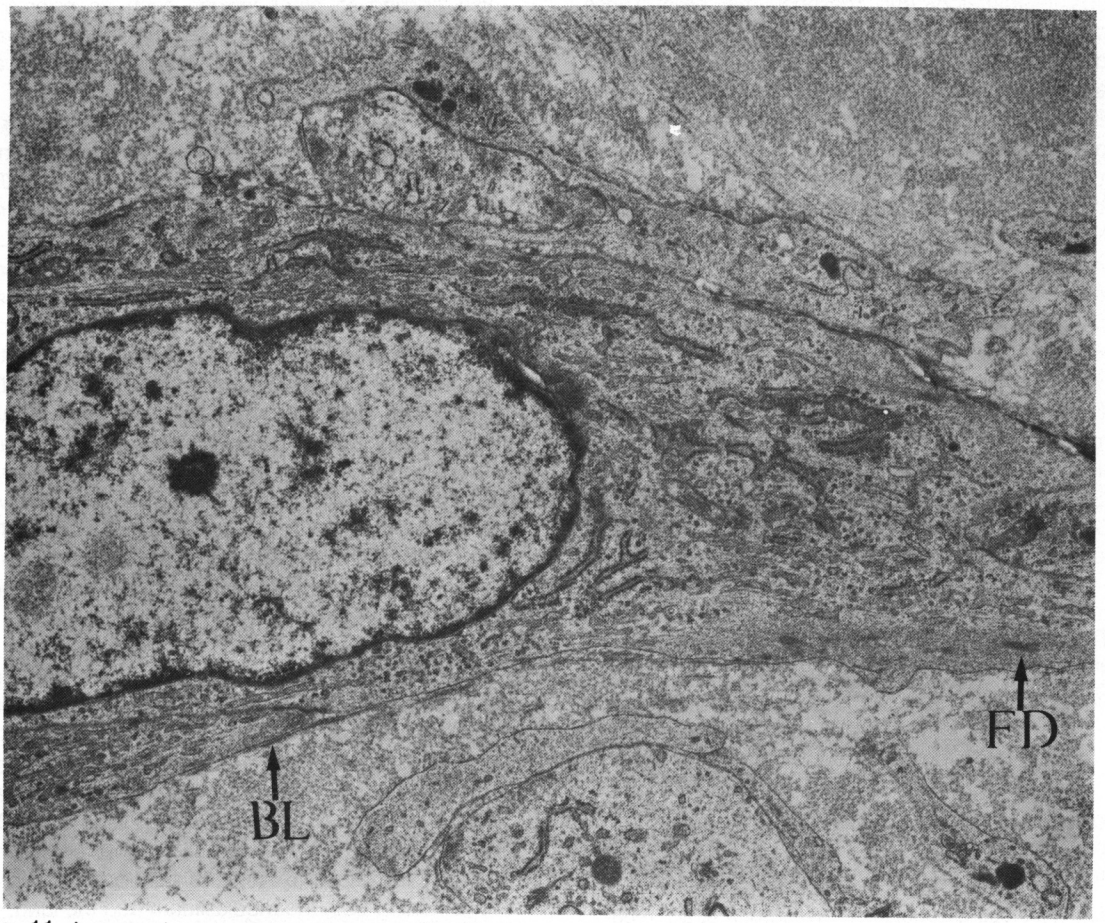

Fig. 11. A mesenchymal spindle cell shows a fibroblastic appearance with submembranous cytoplasmic microfilaments and focal density, along with basal lamina, characteristic of myofibroblasts. (Uranyl acetate and lead citrate, ×10,000) BA: basal lamina, FD: focal density.

Numerous entrapped abortive bronchioles and alveoli were seen, epithelial nature of which were well delineated by elastic stain and reticulin stain. The plasma cells were scattered individually or collected around the vessels and entrapped alveoli, and they showed occasional binucleation and Russell bodies. There were neither atypical cells nor mitoses.

The lung parenchyma adjacent to the mass showed diffuse interstitial mononuclear cell infiltration, peribronchial lymphoplasma cell infiltration with follicle formation, and dilated bronchial trees with fibromucinous intraluminal material with features of bronchiolitis obliterans (Fig. 8, 9). The alveoli contained a mononuclear cell infiltrate with type II alveolar cell proliferation but no hyaline membrane formation. In pleural and subpleural areas, lymphohistiocytic cell infiltration was also seen. Toluidin blue stain revealed scattered mast cells in the perivascular area of the lung parenchyma, but they were not present in the mass. The Congo red stain failed to reveal amyloid deposits in the tumor. The iron hematoxylin stain revealed no intra- or extracytoplasmic deposits of iron.

Ultrastructurally, the lesion was composed of elongated and plump fibroblasts, myofibroblasts, and infiltrated plasma cells, some of which contained Russell bodies inside the rough endoplasmic reticulum, lymphocytes and histiocytes in the background of collagen (Fig. 10, 11)

DISCUSSION

Inflammatory pseudotumor of the lung is a non-

neoplastic pulmonary mass lesion composed of various inflammatory and mesenchymal cells. These different types of cells occur in varying numbers in different lesions and in some instances in different areas of the same lesion (Carter and Eggleston, 1974). Because of this, the varying terminology has been applied to describe inflammatory pseudotumors of the lung. It has caused a great deal of a confusion in gaining a better understanding of their natural history. Names applied to such lesions in the past were as in table 1. The lesion have been confused with sclerosing hemangioma because both may show collections of foam cells and increasing fibrosis as they age. However, the sclerosing hemangioma has predominently epithelial component, i.e., proliferation of type 2 pneumocytes (Chan et al., 1982). Now, the two pulmonary conditions are considered as unrelated ones and almost certainly have a different pathogenesis (Spencer, 1984). For a time, Saldana et al. (1983) and Spencer (1985) proposed that it would be desirable to avoid the designation of "postinflammatory pseudotumor" because the term lacked specificity and was also applied to other lesions such as sclerosing hemangioma. Recently, however, the term "inflammatory pseudotumor of the lung" seems to be widely accepted.

Table 1. Names applied to "inflammatory pseudotumor of the lung" in the past.

Postinflammatory tumor	Brown & Johnson, 1951 Umiker & Iverson, 1954 Fisher & Beyer, 1959 Lal & Thompson, 1974
Xanthomatous & inflammatory pseudotumor	Titus et al., 1962
Xathomatous tumor	Madani et al., 1967
Xanthomatous pseudotumor	Wentworth et al., 1968
Inflammatory pseudotumor	Pearl, 1972
Xanthoma	Grossman et al., 1969
Plasma cell tumor	Childress & Adie, 1950 Cotton & Penido, 1952
Plasma cell granuloma	Tchertokoff et al., 1963 Bahadori & Liebow, 1973 Hoover et al., 1977
Pulmonary plasmacytoma	Mazumdar et al., 1969
Histiocytoma	Bates & Hull, 1958 Dabilier et al., 1968
Fibrous histiocytoma	Grossman et al., 1973 Hakimi et al., 1975
Sclerosing hemangioma	Arean & Wheat, 1962
Solitary mast cell tumor	Cherrette et al., 1966
Pseudoneoplastic pneumonitis	Carter & Eggleston, 1974

A true incidence of inflammatory pseudotumor of the lung is difficult to establish. Golbert and Plentnev (1967) noted an incidence rate of 0.7% among 1,075 tumors of the lung and bronchi. Children are not spared from this disease. In fact, Bahadori and Liebow (1973) noted that inflammatory pseudotumors represented the most common isolated, primary tumor-like lesions of the lung among children under 16 years. Berardi et al. (1983), in their review of the 181 instances of inflammatory pseudotumor of the lung, found that 29 percent of the patients were under 20 years, and 8.1 percent were between one and 10 years old. The youngest patient was a one-year-old (Bahadori and Liebow, 1973), and the oldest was 73 years old, the average age being 29.5 years. No sex predilection was noted. In Korea, three cases of inflammatory pseudotumor of the lung were reported in 1988, all of which occurred in adult males (Kim et al., 1988).

The mass may occur in any lobe of the lung, and on chest X-ray it usually appears as a solitary, circumscribed round or oval mass (Carter and Eggleston, 1974). Calcification and cavitation have been reported. According to a collective review by Berardi et al., (1983) the right lung was involved in 61.2% and the left lung was 39.8%. Endobronchial involvement was noted in 10 patients (6.6%), and eight patients had involvement of both the mediastinum and the lung, as in our case. In three patients, both lungs were involved, and in two, only the trachea was involved. Six patients had multiple lesions involving the same lung.

Inflammatory pseudotumor of the lung tends to be clinically silent and it is most often detected on routine chest radiography (Schwartz, 1980). Of 145 patients, 107 (73.8%) were asymptomatic, and 38 (26.%) were symptomatic at the time of admission to a hospital for evaluation of pulmonary pseudotumor (Berardi el al., 1983). The most common symptom was coughing. Sputum, fever, pain, hemoptysis, shortness of breath and clubbing were also reported. Laboratory data were nonspecific.

The cause and pathogenesis of inflammatory pseudotumors remain obscure. Various theories have been suggested, but none has been proved. It have been regarded as a metabolic disturbance (Scott 1948), a response to an immunologic process (Bahadori and Liebow, 1973; Childress and Adie 1950), an organized cellular growth developing in association with pulmo-

nary infection (Brown and Johnson, 1951), a variant of a postinflammatory repair process (Alegre and Denst, 1958; Wentworth et al., 1968; Umiker and Iver son, 1954; Spyker and Kay, 1956) possibly of viral origin (Pearl, 1972; McCully et al., 1973; Spencer, 1984), an antigen-antibody interaction in relation to an agent which is no longer identifiable, or to aspirated material (Nyman, 1963).

Recently, a majority of investigatiors regards this entity as a variant of an inflammatory repair process rather than a true neoplastic process. One reason is that a pulmonary pseudotumor demonstrates a mixed inflammatory infiltrate with a preponderance of plasma cells. Throughout the lesion are bands and sheets of fibrous tissue, which in some areas are densely hyalinized and in other areas quite cellular. These features allow one to differentiate a pseudotumor from several other biologically different lesions, including malignant lymphoma, plasmacytoma, hamartoma, xanthoma, histiocytoma, and a variety of masses. The other reason is the electronmicroscopic features of pulmonary inflammatory pseudotumor as described by Wentworth (1968), Kuzela (1975), Buell et al. (1976), Shirakusa et al. (1979) and Sajjad et al. (1981). The inflammatory pseudotumors contain spindle-shaped fibroblastic cells, some containing myofilaments in the cytoplasm. Ever since the name of myofibroblast was proposed by Gerami et al. (1968) and Soga et al. (1970) for the cells noted in the granulation tissue during wound repair, it has been demonstrated in various reactive proliferations of mesenchymal tissue, including hypertrophic scar, carpal tunnel syndrome, cirrhosis of the liver, nodular fasciitis, and in the stroma of invasive and metastatic carcinoma (Helwig and Ranier, 1953; Fisch and Brodey, 1976; Benjamin et al., 1977). Myofibroblasts have also been noted in obviously neoplastic conditions, such as malignant fibrous histiocytoma and fibrosarcoma. Although the histogenesis of the my-ofibroblast is not known, it appears that the myofibroblast is an integral part of the inflammatory granulation tissue and reactive connective tissue. The third reason is that positive histories of pulmonary disease have been encountered in some patients with pulmonary inflammatory pseudotumors. Of the previously reported 130 patients, 39 instances (30.0%) had positive histories (Berardi et al., 1983). The length of the time elapsed prior to discovery of the pulmonary inflammatory pseudotumor range from one month to 34 years, one month to one year being the most common.

The present case discloses typical clinicopathologic features in several aspects: (1) an occurrence in children, (2) an indolent course despite large tumor-like mass formation, (3) a positive history of pulmonary disease, i.e., mycoplasma pneumoniae infection involving the same lobe of the inflammatory pseudotumor, (4) a tumor-like large mass involving the right lung with endobronchial and mediastinal involvement, and (5) characteristic histopathology.

Definite diagnosis of mycoplasma pneumoniae infection is made by either culture or serology (Murray et al., 1975). The organism is a slowly growing bacterium which requires complex media and a long incubation time for successful culture. The usual serologic test is complement fixation with paired acute and convalescent sera, and a four-fold or higher titer rise indicates infection. With convalescent sera only, a titer of 1:64 or higher is strongly suggestive of infection. Our patient failed to culture the organism when mycoplasma pneumoniae infection was first suspected and a pulmonary inflammatory pseudotumor was found. However, in 1988, the serum titer of antimycoplasma antibody by complement fixation test, initially 1:16 and later 1:80, was significant and strongly suggestive of mycoplasma infection. Thereafter, the antimycoplasma antibody titer became negative, and the patient was in good health without any further problem. One-and-a-half years later, cough and fever developed and the titer was elevated again to 1:40. In addition, the pathology of the lung parenchyma adjacent to the mass showed diffuse interstitial and peribronchial mononuclear cell infiltration and multifocal features of bronchiolitis obliterans. Although these findings might only be an organizing pneumonia induced by obliteration of major bronchus, they might also be the lesion associated with mycoplasma pneumoniae infection. It seems important in our case that the involved lobes were the same for both the inflammatory pseudotumor and previous pneumonia, increased serum antimycoplasma antibody titer, and the mycoplasma pneumonia-resembling histologic features of the lung parenchyma.

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