□ CASE REPORT □

Abscessing Bronchioloectasia with Elements of Plasma Cell Granuloma

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A 79-year-old male was admitted to the hospital with the complaint of bloody sputum. Chest X-ray revealed an abnormal shadow in the right upper lobe. Macroscopically, the lesion measured about 3.5×2.5×2.0 cm with a central cavity containing pus. Histologically, the lesion was composed of interlacing fibroblastic proliferation with abundant plasma cell infiltration and central cavitation. The inner surface of the cavity wall was partially covered by bronchial epithelial cells; there was no cartilage found, suggesting that the lesion had developed from chronic inflammatory processes in relation to ectatic bronchioles. Since the pathogenesis of plasma cell granuloma (PCG) has not been well established, it is probable that this case represents one stage in the development of classic PCG.
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Introduction

Plasma cell granuloma (PCG) of the lung is a relatively rare tumor-like lesion composed of mature plasma cells with a varying number of reticuloendothelial cells, lymphocytes and intermediate "plasmacytoid" cells in a spindle cell stromal proliferation (1). The lesion may be confused radiologically and clinically with primary or metastatic neoplasms of the lung. The cause of PCG is unclear, though localized injury with subsequent chronic inflammation is suspected. Bahadori and Liebow first coined the term "plasma cell granuloma" (1), referring to a group of lesions that were previously designated by a variety of terms, such as xanthoma, fibroxanthoma, xanthogranuloma, histiocytoma, and post-inflammatory pseudotumor, based on different components of the cellular infiltration. The presence of a number of designations indicats the broad histologic spectrum of this condition. We recently analyzed 7 cases of PCG pathologically and reported the findings (2). Here, we present an unusual case of cavitary lung abscess containing discrete foci of PCG, a constellation of findings that, to our knowledge, has not been reported previously.

Case Report

A 79-year-old Japanese male was admitted to Kanazawa University Hospital on February 2, 1993 with the complaint of bloody sputum. He was a non-smoker and had no fever, cough or other symptoms related to respiratory-tract infections. There was no cyanosis or clubbing of the digits. Laboratory data were all within normal limits, including tumor markers. Acid-fast smear and culture of sputum and bronchial lavage samples were negative. Routine bacterial cultures of sputum and bronchial lavage samples were also negative. There were no eosinophils and no malignant cells on Papanicolaou-stained sputum or nasal secretions, nor on transbronchial brushing specimens. Chest x-rays showed an ill-defined abnormal shadow in the right upper lobe (S-2) (Fig. 1). Computed tomography (CT) revealed a mass with a central low density area (Fig. 2).

Since chest X-ray and CT findings could not exclude the possibility of malignancy; exploratory thoracotomy and excisional biopsy of the lesion were performed on February 17, 1993. Frozen section of the surgical specimen revealed benign nonspecific chronic inflammatory change. Macroscopic examination of the resected specimen revealed an ill-defined, white-yellow mass measuring 3.5×2.5×2.9 cm with a central cavity containing pus (Fig. 3). Postoperative histologic examination

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revealed that the lesion was composed of a central cavity and fibrous inflammatory granulation tissue with abundant plasma cell infiltration (Fig. 4), similar to plasma cell granuloma, together with fibrinous and polymorphonuclear cell exudate in the cavity. The outer area of the lesion merged with the organizing pneumonia of the surrounding parenchyma. A small part of the inner surface of the cavity was covered with respiratory columnar epithelium, but there was no cartilage detected (Fig. 5). This appearance is consistent with the theory that the lesion originated from repeated inflammation in a bronchioloectatic area. Examination of the adjacent lung tissue disclosed mild bronchopneumonia and focal bronchioloectasis. Bacterial culture of the lesion revealed the identification of two types of anaerobic Gram-negative bacteria, according to the sensitivity to antibiotics.

The immunoperoxidase technique on formalin-fixed, paraffin-embedded tissue, as reported previously (2), for immunoglobulins demonstrated that the plasma cells in the lesion were stained with kappa and lambda light chains, indicating the polyclonal nature of these cells.

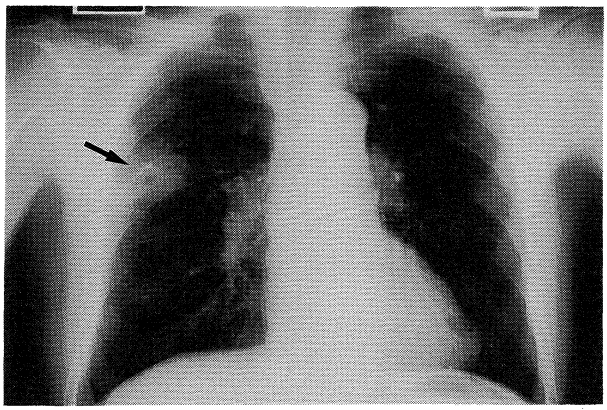

Fig. 1. Chest X-ray showing an ill-defined density in the right upper lobe (arrow).

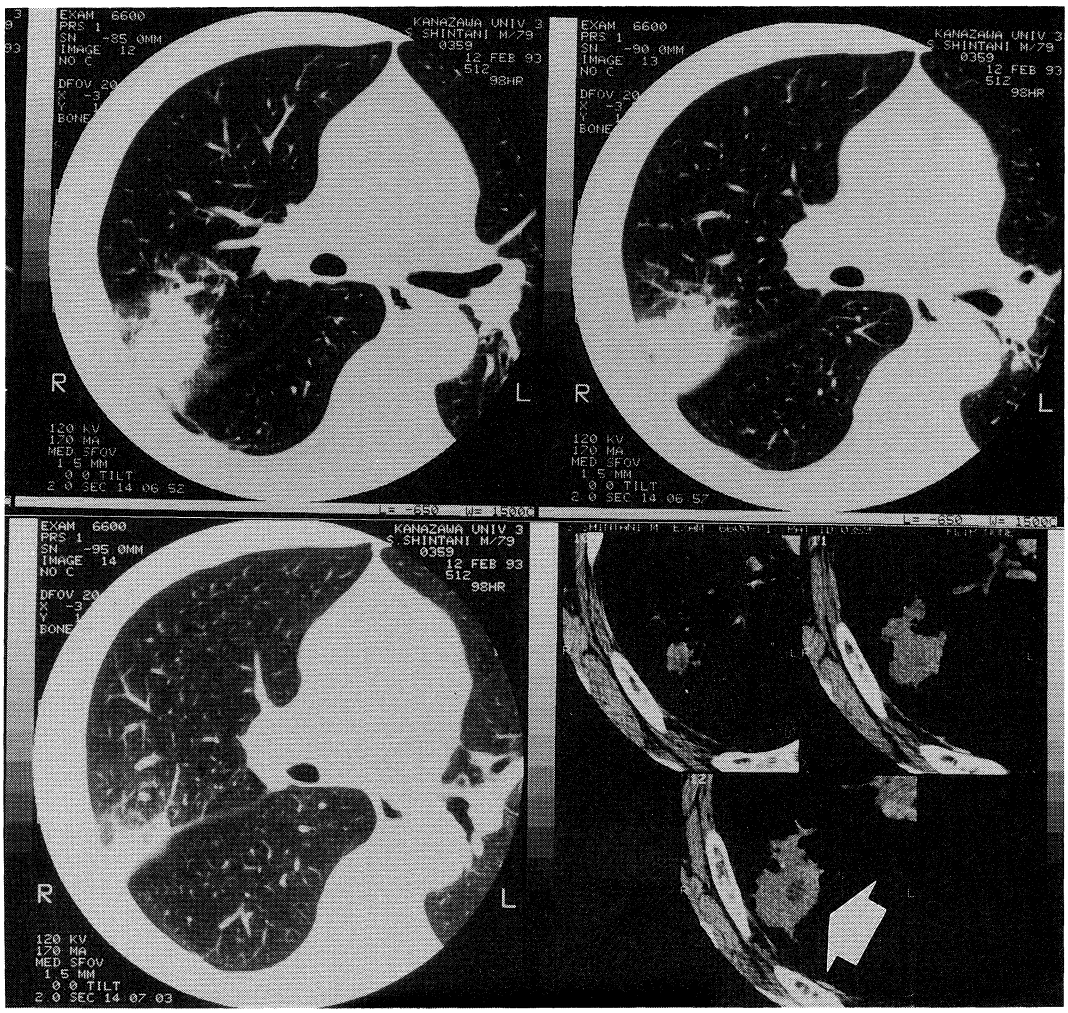

Fig. 2. Computed tomography of the lesion showing central cavitation (arrow).

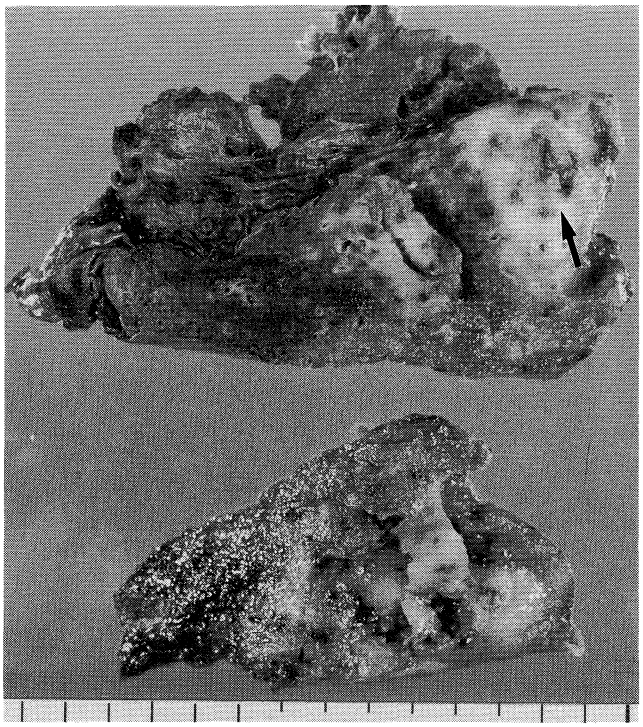

Fig. 3. Macroscopic appearance of the cut surface of the lesion, showing an ill-defined white-yellow mass with a central cavity (arrow).

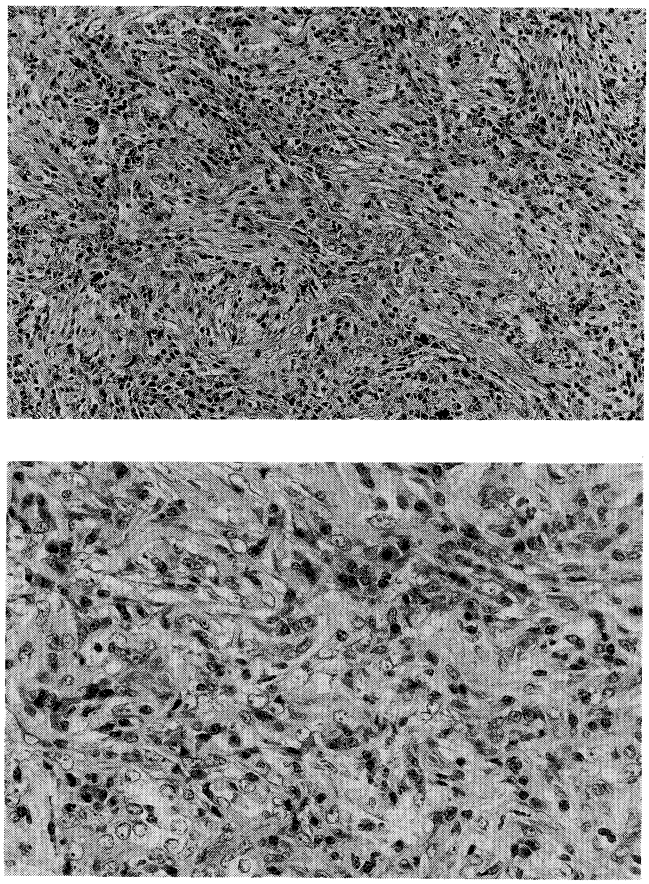

Fig. 4. Microphotograph of the lesion, showing interlacing fusiform fibroblastic proliferation with a vague storiform pattern (upper), and admixed with abundant plasma cells (lower) (HE stain, a: ×200, b: ×400).

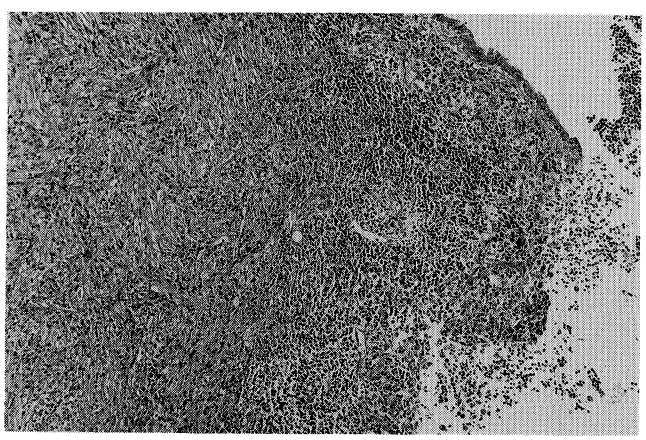

Fig. 5. Microphotograph of the central cavity of the lesion, showing polymorphonuclear leukocyte exudation in the cavity and partial lining of the cavity wall by bronchial epithelium (HE stain, ×100).

Discussion

Plasma cell granuloma (PCG) is a rare non-neoplastic tumor-like lesion occurring most frequently in the lungs of children (1–6). However, PCGs have also been reported in a variety of extrapulmonary sites (7). A solitary circumscribed round or oval mass within the lung is the most common radiological presentation of pulmonary PCG and is often confused with primary or secondary lung carcinoma (1–3). In a minority of cases, the lesion arises endobronchially (1). It may undergo liquefaction, cavitation, or calcification, but if a cavity is present, it is usually small and is clearly related to the remnants of a small bronchus (1), as seen in the present case.

The cause and pathogenesis of PCG remains obscure. Numerous theories have been proposed, but none have been proven (3). However, the majority of investigators regard this entity as a variant of an inflammatory repair process rather than a true neoplastic process (1, 3, 6), because of the frequent history of prior respiratory tract infections and the histologic composition of mature plasma cells, and reticuloendothelial cells, together with varying amounts of other mature inflammatory cells among the background granulation tissue. Recently, in a study of 32 cases of PCG, Matsubara et al concluded that most or all cases of PCG originate as organizing pneumonia (6). Furthermore, it is possible to induce PCG in animal models by intrabronchial instillation of Freund's complete adjuvant (8). These findings indicate that PCG can develop rather rapidly in the wake of a pulmonary infection.

The case reported here suggests a pathogenic relationship between PCG and lung abscess, most probably related to the anaerobic Gram-negative bacterial infection. Mohsenifar et al

reported a case of cystic organizing pneumonia with elements of plasma cell granuloma and Aspergillus fumigatus infection (9). They considered that the presence of Aspergillus fumigatus in the cystic cavity represents superinfection of the cystic organizing pneumonia and indicates an intimate relationship between PCG and organizing inflammatory process (9). Since the natural history of PCG is unclear, it is possible that the present case and the case reported by Mohsenifar et al represent aborted forms or one of the stages in the development of classic PCG.

Although the inhalation of aerosols containing bacteria can result in many of the micro-organisms reaching the lung parenchyma, the majority of the inhaled bacteria under normal conditions removed from the upper air passages proximal to the larynx and the lower reaches of the respiratory tree were sterile as judged by the failure to stain and culture micro-organisms from material aspirated through a bronchoscope (10). A number of conditions which adversely affect the efficiency of these normal physiological means for ridding the lung of infection render the organ more prone to bacterial invasion, including alcoholism, general anesthesia, neurological disease, and a variety of pathologic conditions of the lung, such as lung carcinoma and bronchiectasis. Anaerobic bacteria are normal inhabitants of many mucosal surfaces. They are plentiful in the gastrointestinal tract, genital tract, certain areas of skin, and the oral cavity. In the mouth, they particularly thrive in specific sites such as the gingivo-dental sulcus and the tonsilar crypts where the redox potential is low (11). The lower respiratory tract is normally free of anaerobes. However, it is well known that anaerobes often colonize bronchiectatic bronchi (12). Therefore, we considered that the pulmonary lesion of the present case developed from chronic inflammatory process due to anaerobic bacterial infection of the bronchiectatic bronchioles, according to the pathologic findings of the resected pulmonary lesion. Since precise pathogenesis of PCG is still unclear, our case probably represents one stage of the development of classic PCG.

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