□ ORIGINAL ARTICLE □

Seven Patients with Plasma Cell Granuloma (Inflammatory Pseudotumor) of the Lung, Including Two with Intrabronchial Growth: An Immunohistochemical and Electron Microscopic Study

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Seven patients (mean age, 50.7 ± 20.4 years; range 21–77) with plasma cell granuloma (PCG) of the lung are reported. Cough and sputum were the most common presenting symptoms, followed by fever. Elevated erythrocyte sedimentation rate and serum C-reactive protein levels were found in all patients tested. Radiologically, five cases presented as solitary, well-circumscribed masses and two as ill-defined, pneumonia-like densities. One showed focal calcification. No predilection of occurrence was observed in either lobe of the lung. Histologically, the lesions consisted of a proliferation of mature plasma cells and reticulo-endothelial cells supported by a stroma of granulation tissue, with varying degrees of myxoid change or collagenization. Angioinvasion within the lesion was observed in 4 of the 7 cases. Immunohistochemical staining revealed the IgG-predominant polyclonal nature of the plasma cells, indicating a reactive inflammatory process rather than a neoplastic one. Electron microscopy confirmed the benign nature of the plasma cells with fibroblast and myofibroblast proliferation admixed with that of other inflammatory cells.

(Internal Medicine 31: 756–765, 1992)

Key words: lung tumor, inflammation, pulmonary infection, immunohistochemistry, histiocytoma, plasmacytoma

Introduction

Plasma cell granuloma (PCG) of the lung is a rare non-neoplastic mass of unknown cause and pathogenesis; it consists of a variety of inflammatory cells with plasma cell dominance, and includes spindle-shaped mesenchymal cells (1–3). It is a frequently forgotten entity that is important in the differential diagnosis of masses of the lung and is often confused with primary or metastatic carcinoma (2). A variety of names, based on different components of the cellular infiltration, have been given to this entity, e.g., inflammatory pseudotumor, histiocytoma, xanthoma, and fibroxanthoma (1–3). The vast majority of PCG are intraparenchymal and only a few cases have presented as obstructive masses in the bronchi or trachea (1–3). Over a period of 20 years we have seen 7 PCG cases, of which two showed intrabrochial exophytic growth. In this report we present the clinicopathologic findings of the 7 cases, together with details of our immunocytochemical and electron microscopic studies.

Materials and Methods

Seven surgical specimens of plasma cell granuloma (PCG), dating from 1968 to 1989, were collected from the files of the Pathology Section, Kanazawa University Hospital, School of Medicine, Kanazawa University. The specimens were fixed in formalin, and routine histologic examination was performed after paraffin embedding and staining with hematoxylin-eosin, Azan-Mallory, elastica Van Gieson, and periodic acid-Schiff (PAS).

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Received for publication May 7, 1991; Accepted for publication February 28, 1992

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Electron microscopy

Fresh samples from two cases were minced into small cubes and fixed in 2.5% glutaraldehyde in phosphate-buffered saline (PBS), postfixed in 1% osmium tetroxide in PBS, and embedded in Epon 812. Ultrathin sections were stained with uranyl acetate-lead citrate, and examined under a transmission electron microscope.

Immunohistochemistry

In all cases, the paraffin sections were stained using the avidin-biotin-peroxidase complex (ABC) method, to investigate immunoglobulin IgA, IgM, IgG, and the kappa and lambda chains, as reported previously (4).

Results

Clinical findings

Clinical findings of the seven patients are shown in Table 1. The mean age of the four male and three female patients was 50.7 ± 20.4 (mean $\pm$ S.D., range 21–77). All patients except one had respiratory symptoms. Cough and sputum, often bloody in nature, were the

Table 1. Clinical Findings of Patients with Plasma Cell Granulomas of the Lung

Case	Age/sex	Symptoms	Past history, and/or associated disease**	Smoking pattern	Laboratory data	Radiographic findings	Location
1	21/F	Non	Pneumonia at 18 years	Non-smoker	ESR 32/67, RBC 430×10^6/cmm WBC 6,400/cmm	Mass lesion	LUL
2	33/F	Hemoptysis cough	Tuberculosis* at 6 years, and pneumonia at 16 years	Non-smoker	ESR 42/78, RBC $386/10^6$/cmm WBC 6,600/cmm	Mass lesion with calcification	LLL
3	41/M	Cough, sputum fever	None	Non-smoker	ESR 112/151, WBC 6,700/cmm CRP 8.7 mg/dl, RBC 456×10^6/cmm	Mass lesion	RLL
4	77/M	Hemosputum cough	Gastrectomy for ulcer at 70 years, pneumonia at 70 years	20 per day for 20 years	CRP 1.4 mg/dl, RBC 334×10^6/cmm WBC 7,400/cmm	Mass lesion	RUL
5	68/M	Sputum, fever	Chronic hepatitis**	40 per day for 10 years	CRP 0.6 mg/dl, RBC 328×10^6/cmm WBC 3,800/cmm	Pneumonia-like density	LLL
6	65/F	Cough, sputum fever, malaise	Tuberculosis* at 25 years	Non-smoker	CRP 1.1 mg/dl, RBC 403×10^6/cmm WBC 7,800/cmm	Mass lesion	RUL
7	50/M	Cough, fever chest pain	Diabetes**	30 per day for 30 years	CRP 1.3 mg/dl, RBC 337×10^6/cmm WBC 5,800/cmm	Pneumonia-like density	RUL

*: pulmonary tuberculosis, **: associated disease, ESR: erythrocyte sedimentation rate, RBC: red blood cells, WBC: white blood cells, CRP: C-reactive protein, LUL: left upper lobe, LLL: left lower lobe, RLL: right lower lobe, RUL: right upper lobe

Table 2. Pathologic Findings of Plasma Cell Granulomas of the Lung

Case	Age/sex	Location	Size (cm)	Border	Cut surface appearance	Consistency	Plasma cell*
1	21/F	LUL (S-5), periphery	$7.1 \times 6.3 \times 3.7$	Clear	Gray to yellow-whitish with areas of hemorrahge cysts, and gelatinous change	Hard	Polyclonal IgG $\gg$ IgA $\gg$ IgM
2	33/F	LLL (S-6) with intrabronchial growth in subsegmental bronchus (B-6b), periphery	$4.3 \times 2.2 \times 3.0$	Clear	Grayish with calcification	Firm	Polyclonal IgG $\gg$ IgA $\gg$ IgM
3	41/M	RLL segmental bronchi (intrabronchial growth) (B-9), hilar	$4.5 \times 2.7 \times 2.5$	Clear	Whitish yellow with gelatinous areas	Soft and friable	Polyclonal IgG $\gg$ IgA $>$ IgM
4	77/M	RUL (S-1), periphery	$5.0 \times 5.0 \times 4.5$	Relatively unclear	Yellowish white with small xanthomatous foci and patchy anthracosis	Hard	Polyclonal IgG $>$ IgA $\gg$ IgM
5	68/M	LLL (S-10), periphery	$2.5 \times 2.0 \times 1.5$	Unclear	Yellowish and pneumonia-like	Soft	Polyclonal IgG $\gg$ IgA $>$ IgM
6	65/F	RUL (S-3), periphery	$4.5 \times 3.0 \times 3.5$	Slightly unclear	Yellowish	Fleshy	Polyclonal IgG $\gg$ IgA $>$ IgM
7	50/M	RUL (S-1), periphery	$5.0 \times 4.0 \times 3.0$	Unclear	Yellowish	Soft	Polyclonal IgG $>$ IgA $>$ IgM

*: evaluated by immunohistochemical staining.
LUL: left upper lobe, LLL: left lower lobe, RLL: right lower lobe, RUL: right upper lobe

most common symptoms, and these were observed in 5 symptomatic patients, fever was observed in 4, chest pain in 1, and general malaise in 1. There was a history of antecedent pulmonary infection in 4 patients (pneumonia in 2, tuberculosis in 1, and pneumonia and tuberculosis in 1). However, the period prior to discovery of the PCG ranged from three to 40 years. Three patients were cigarette smokers.

Regarding the laboratory data, the erythrocyte sedimentation rate (ESR) was elevated in all three patients tested, and CRP was also above the normal level in all patients tested. Slight anemia was observed in three patients, but no significant leukocytosis was found in any of the patients.

Radiologically, the lesions of five patients were well-circumscribed, round masses within the lung. Focal calcification was observed in one case (case 2). Two patients showed pneumonia-like, ill-defined density. Three lesions were located in the right upper lobe (RUL), two in the left lower lobe (LLL), one in the left upper lobe (LUL), and one in the right lower lobe (RLL).

Pathologic findings

The main macroscopic features of the 7 lesions are shown in Table 2. Five lesions presented as peripheral intrapulmonary masses, ranging from 2.5 to 7.1 cm in diameter. Case 3 presented mainly as an intrabronchial mass (Fig. 1). One case (case 2) showed an intrapulmonary mass with an intrabronchial exophytic growth in the subsegmental bronchus. Three lesions were well-circumscribed (Fig. 2), two were relatively ill-defined (Fig. 3), and two were ill-defined. The cut-surface of the lesions was generally homogeneous, and was grayish, yellowish-white, or yellow (Figs. 1, 2). Areas of hemorrhage, gelatinous or cystic changes (Fig. 4), and anthracosis were noted in some cases. The consistency of the lesions varied from firm to soft.

Histologically, even in the well-circumscribed lesions, there was no fibrous capsule or delimitation, and the cell components infiltrated the surrounding parenchyma to

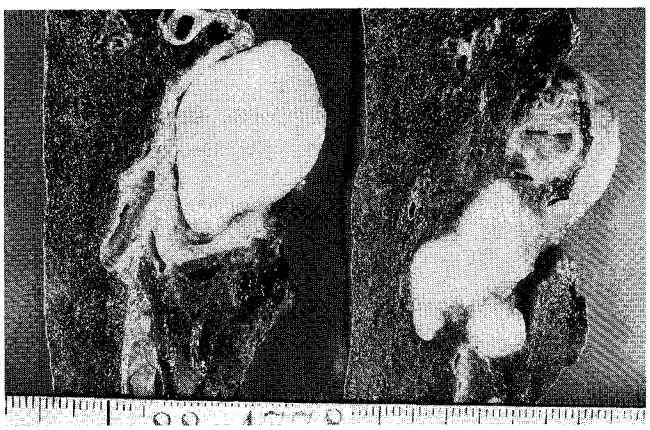

Fig. 1. Sectioned surface of plasma cell granuloma, presenting as an intrabronchial exophytic, obstructed mass (case 3).

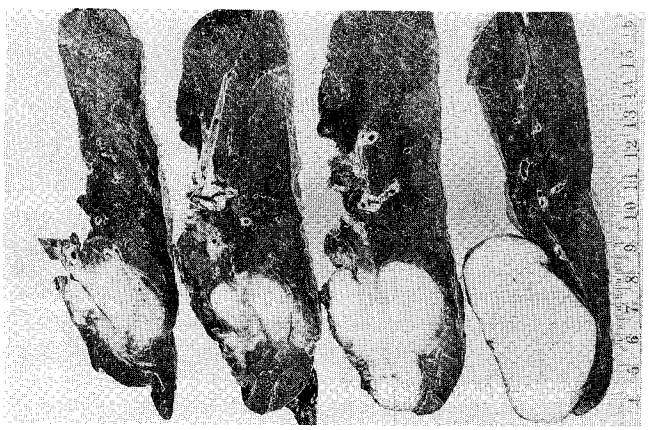

Fig. 2. Plasma cell granuloma presenting as a well circumscribed, firm, pale, gray intrapulmonary mass (case 1).

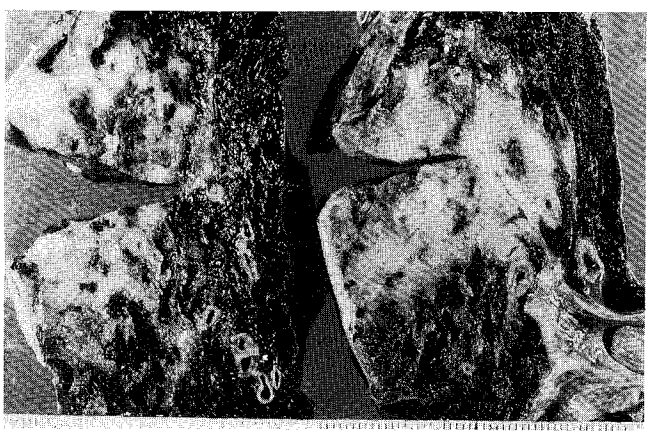

Fig. 3. Macroscopic appearance of plasma cell granuloma presenting as a relatively ill-defined, intrapulmonary mass (case 4).

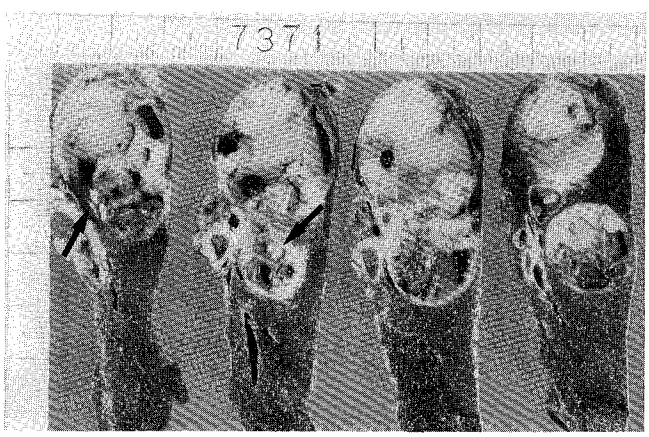

Fig. 4. Sectioned surface of a well-circumscribed mass of plasma cell granuloma (case 2), showing areas of hemorrhage, cyst formation, gelatinous change, and calcification (arrows).

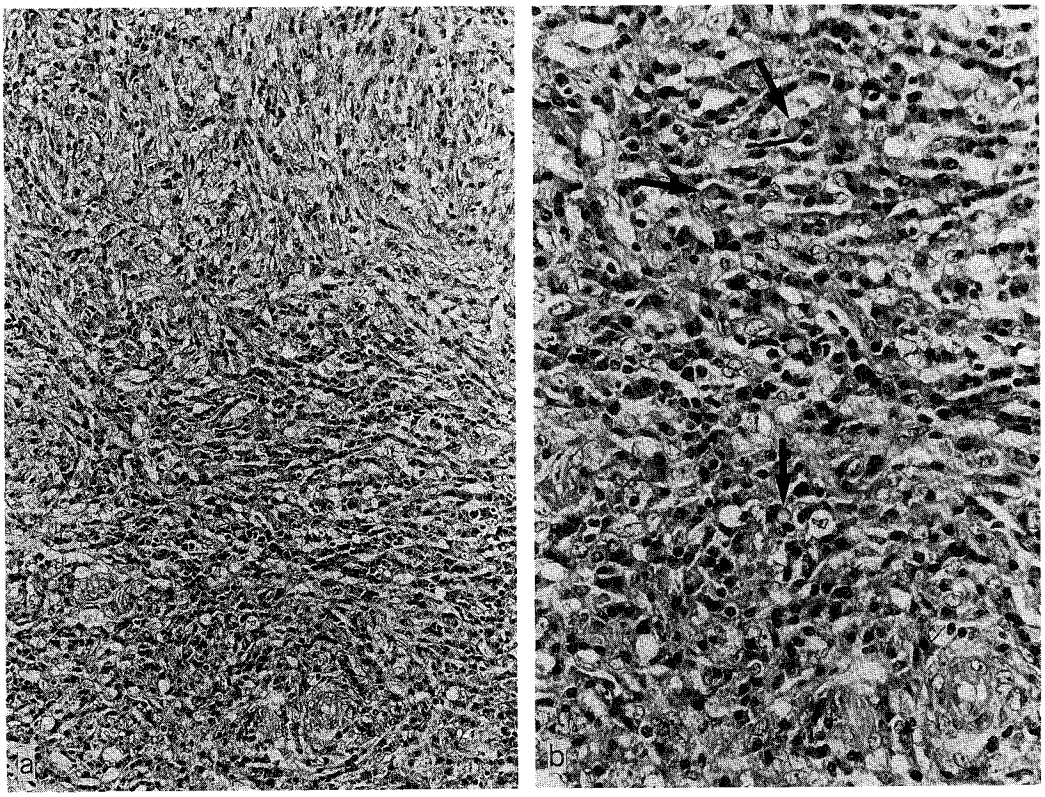

Fig. 5. Microscopic appearance of the plasma cell granuloma from case 1, showing interlacing fibroblastic proliferation with a vague storiform pattern (a), admixed with abundant plasma cells with occasional Russell bodies (arrows, b). H & E stain. a) ×600, b) ×1,200.

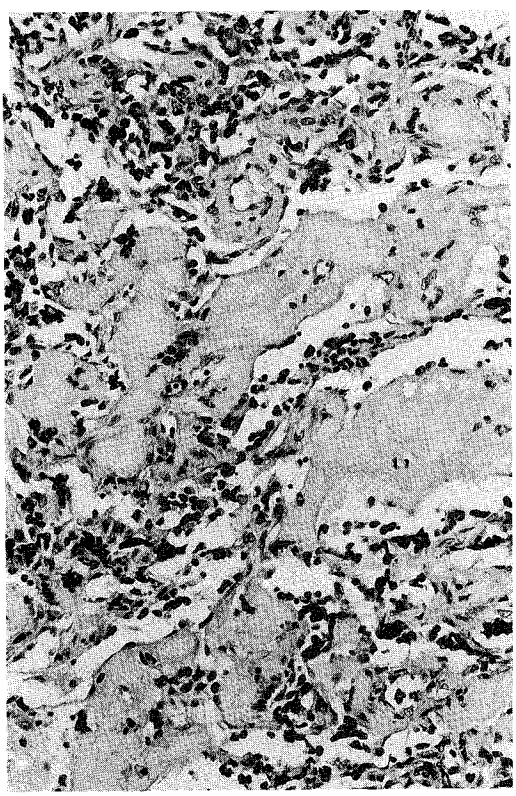

Fig. 6. Hyaline fibrosis and residual chronic inflammatory cell infiltration (case 3). H & E stain, ×600.

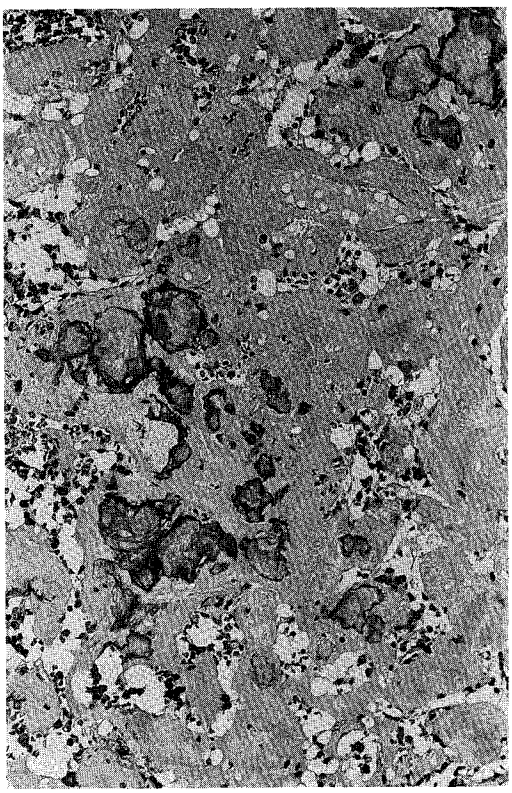

Fig. 7. Marked stromal fibrosis with areas of calcification (case 2). H & E stain, ×600.

some extent. In cases where the border was ill-defined macroscopically, inflammatory cells infiltrated much more abundantly into the surrounding parenchyma and the interstitial tissue was thickened. The histologic features showed only a few variations from case to case. The principal components of the lesion were spindle-shaped or fusiform cells and plasma cells (Fig. 5). However, the density of the plasma cells varied from area to area and from case to case. The plasma cells appeared mature, and intracytoplasmic and extracellular Russell bodies were occasionally found (Fig. 5). Occasional plasma cells were binucleated, but mitotic figures were not observed. Fusiform or spindle-shaped cells were arranged in a whorled, interlacing or storiform pattern (Fig. 5). No cellular atypia or mitotic figures were found in these cells. Lymphocytes were present in small clumps, sometimes in a follicular arrangement with germinal centers. Histiocytes, occasionally with a foamy appearance, and polymorphonuclear leukocytes were also observed in small clumps. Eosinophils and mast cells were present in some lesions but were never numerous. Intercellular eosinophilic amorphous hyaline materials resembling amyloid were found in four cases (cases 1–3, and 5) (Fig. 6). These materials, however, were negative for Congo red stain. In two cases (cases 2 and 3), these materials were abundant, and in one of these cases (case 2) they were associated with focal calcification (Fig. 7).

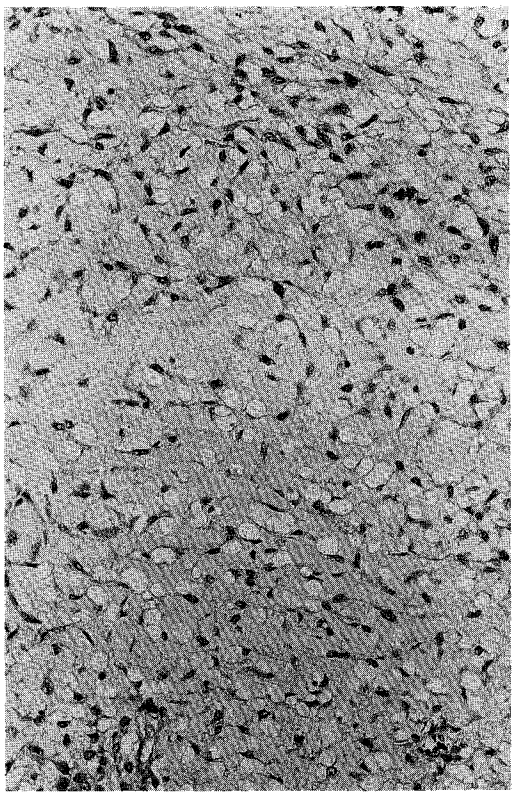

Fig. 8. Marked stromal myxoid change with fusiform fibroblastic cell growth and infiltration of a few inflammatory cells (case 3). H & E stain, ×600.

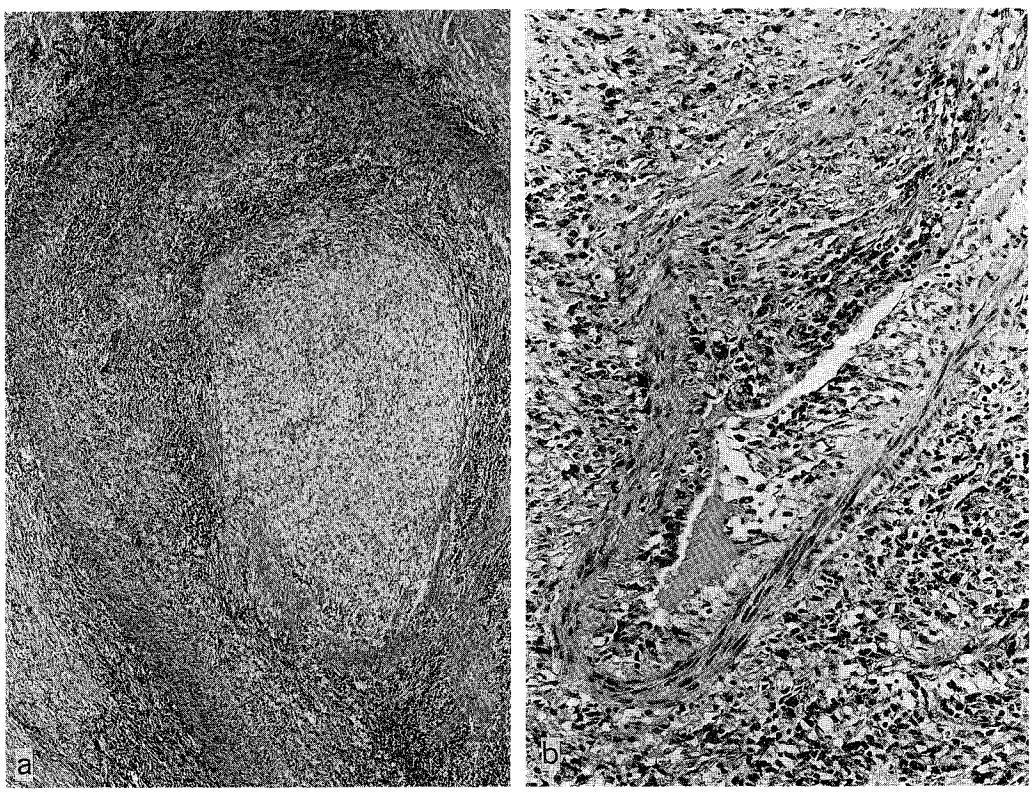

Fig. 9. Histologic appearance of vascular invasions at the periphery of plasma cell granulomas, seen in case 3 (a) and in case 2 (b). H & E stain. a) ×120, b) ×600.

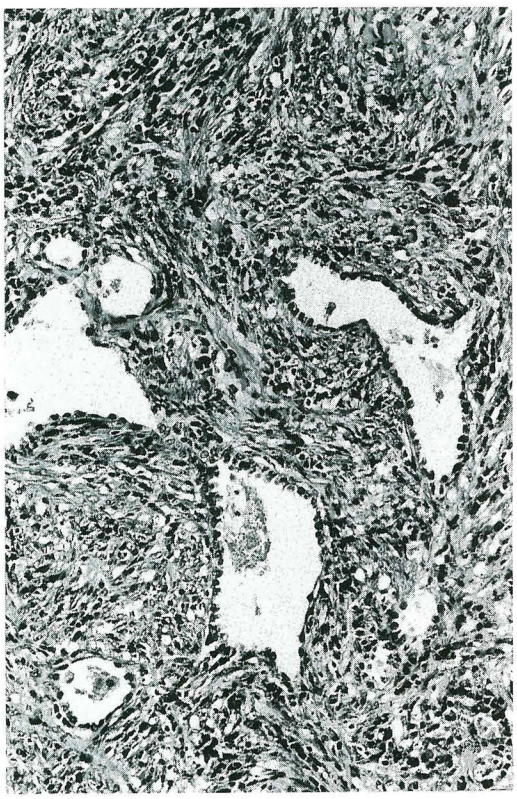

Fig. 10. Plasma cell granuloma with residual epithelial structures (case 1). H & E stain, ×600.

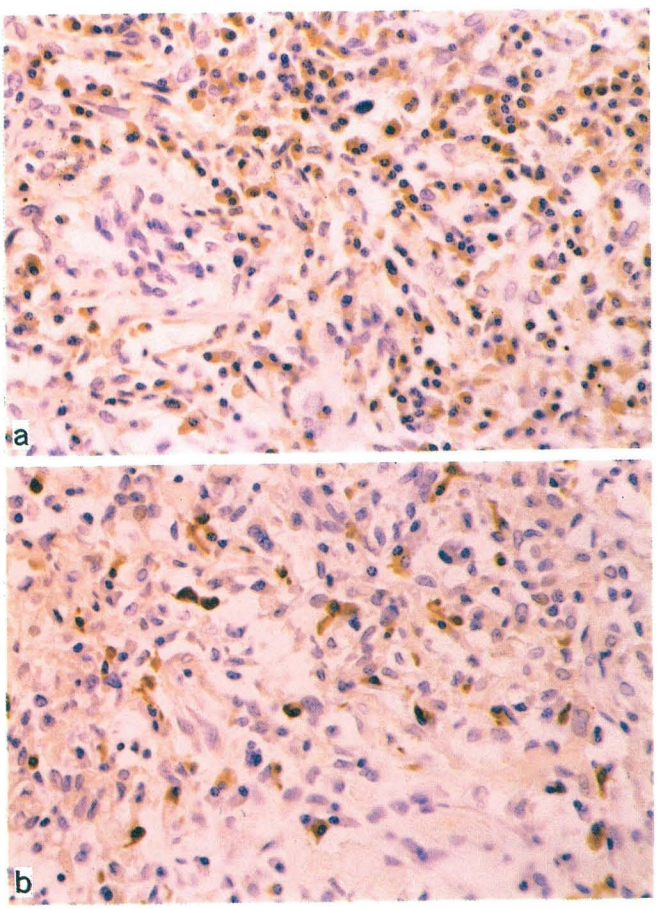

Fig. 11. Immunohistochemical staining of plasma cell granuloma plasma cells for immunoglobulin kappa (a) and lambda (b) in case 3. Plasma cells containing both types of immunoglobulin are observed. Immunoperoxidase staining. Both a) and b), ×1,400.

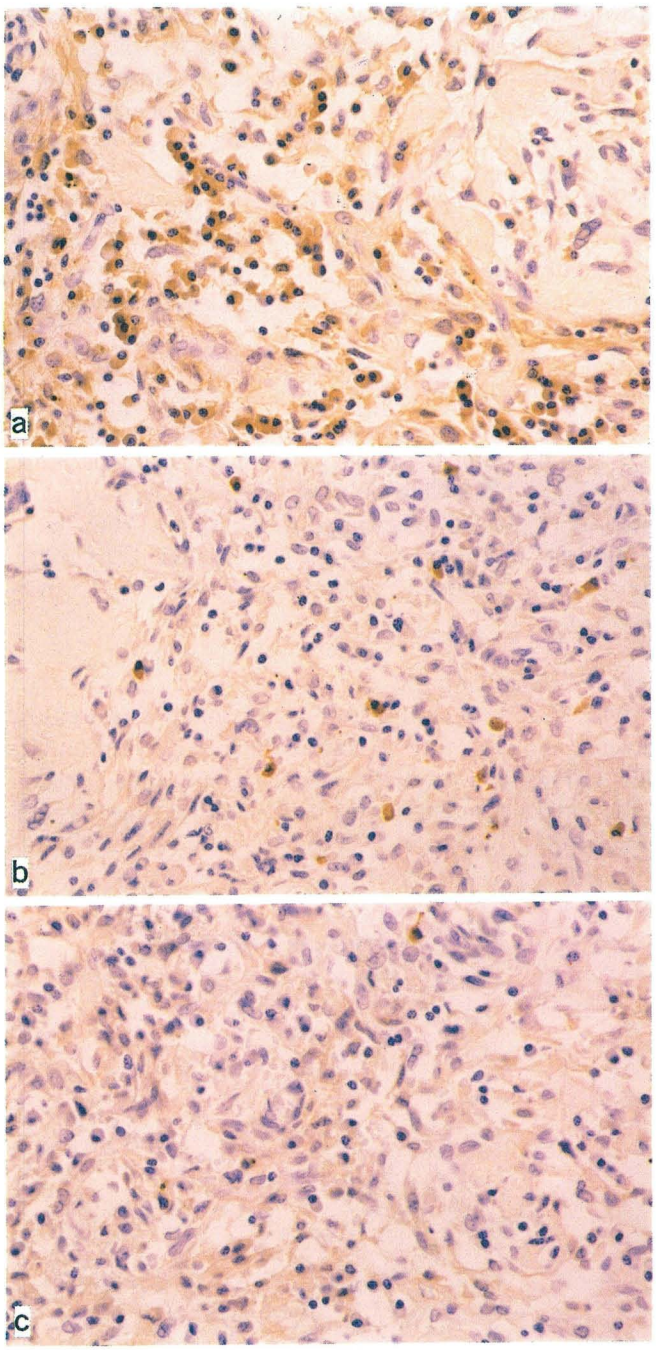

Fig. 12. Immunohistochemical staining of plasma cell granuloma plasma cells for IgG (a), IgA (b), and IgM (c) in case 3. The majority of the plasma cells are positive for IgG (a). Immunoperoxidase staining. a), b), and c), ×1,400.

Myxoid areas were observed in one case (case 3) (Fig. 8). In four instances (cases 1, 2, 3 and 7), invasion of medium-sized vessels and frequent obliteration of their lumina were observed within the lesion, especially at the peripheral part (Fig. 9). In two cases (cases 2 and 4) there were small necrotic foci. Focal cystic change was observed in one case (case 2). At the periphery of the lesions, epithelial-lined glandular structures, probably representing the entrapped alveolar spaces, were sometimes found, and these were occasionally quite numerous (Fig. 10).

Immunohistochemical findings

Plasma cells were stained with kappa and lambda light chains, indicating the polyclonal nature of these cells (Fig. 11). In all cases in the present study, the majority of plasma cells were positive for IgG (Fig. 12a); a minority of them were positive for IgA (Fig. 12b). However, IgM-positive plasma cells were only rarely found (Fig. 12c).

Ultrastructural findings

Ultrastructural studies were performed in two cases, in which the basic ultrastructure was found to be similar. The lesions consisted of spindle-shaped cells, plasma cells, and other inflammatory cells, with varying amounts of fibrillary intercellular matrix. The spindle-shaped or fusiform cells were predominantly fibroblastic in type and were arranged to form parallel streams or concentric whorls (Fig. 13). The majority of the nuclei of these cells was characteristically indented. No intercellular junctions were demonstrated. The cytoplasm was large in area and had conspicuous rough endoplasmic reticulum and dilated cisternae. Some of these cells occasionally contained bundles of cytoplasmic fibrils with occasional dense bodies, mostly arranged parallel to the cell axis in a perinuclear distribution (Fig. 14), a feature of myofibroblasts. Pinocytic vesicles were rarely noted. Smooth muscle cells were not observed. Plasma cells displayed characteristic peripheral nuclear chromatin clumping and abundant loosely packed rough endoplasmic reticulum (Fig. 15). The mitochondria were not abundant and the Golgi apparatus appeared to be normal. Atypical or immature plasma cells with less abundant endoplasmic reticulum were not observed.

Discussion

It is widely believed that plasma cell granulomas (PCG) (inflammatory pseudotumors) of the lung arise as postinflammatory, reactive lesions most commonly in young persons under the age of 30 (1–3). According to

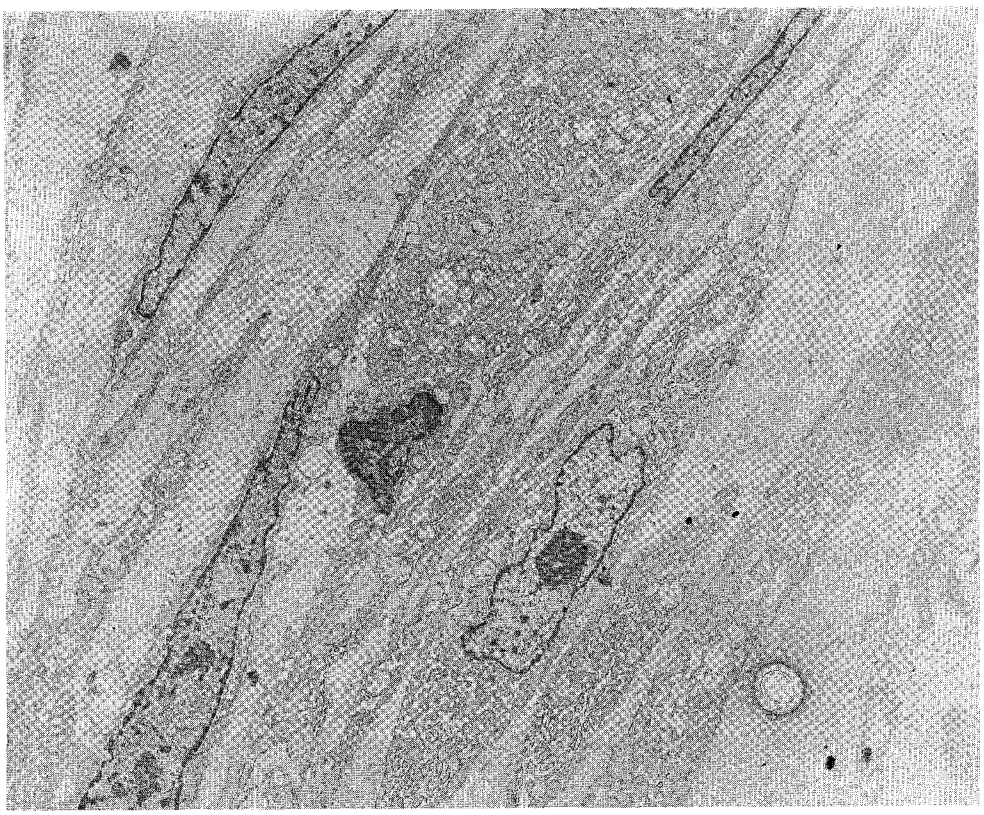

Fig. 13. Electron microscopic feature of a plasma cell granuloma from case 4; numerous spindle-shaped or fusiform cells are seen lying in a fibrillary matrix. (×3,000)

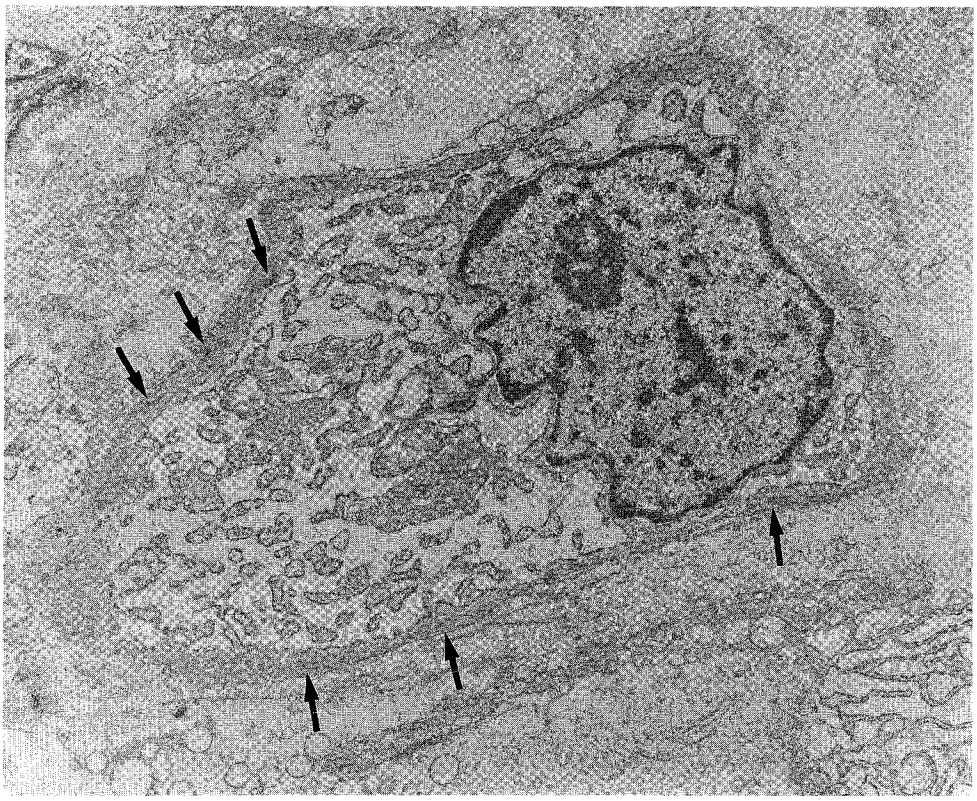

Fig. 14. Electron microphotograph of a fibroblastic mesenchymal cell with well-developed rough endoplasmic reticulum and peripheral cytoplasmic microfilaments and dense bodies (arrows), characteristic of a myofibroblast. Case 4. (×6,000)

an analysis of 40 cases of pulmonary PCG reported by Bahadori and Liebow, more than two-thirds of the lesions were in patients under the age of 30 years, and one-third were in patients under the age of 20 (1). Similarly, a recent analytical review of 181 cases of pulmonary PCG by Berardi et al (2) revealed that the mean patient age was 29.5 years, that 59.2% of the patients were under the age of 40, and that 48.9% of the patients were in the first four decades of life. However, in the present study, six of seven patients were over the age of 30, and five of the seven were older than 40 years of age. Recently, Shirakawa et al (5), in their collective review of forty-eight Japanese patients with pulmonary PCG, reported that thirty-four (71%) were more than 30 years old, and that 10 (21%) were under the age of 20. The mean age was 41.2 years (range, 5 to 71). Thus, PCG seems to be much more frequently observed in older persons in Japan than in the United States or Europe. Furthermore, although PCG affects males and females almost equally in the United States and in Europe (2), 32 of 48 (67%) Japanese cases reviewed were males (5).

The usual presenting symptoms in the present series were cough, followed by sputum, which was occasionally bloody in nature, and fever; these were similar to those reported previously (2), but in many cases there were no symptoms (1–3). PCG is occasionally preceded by infection (1–3). Four of the present seven cases were associated with antecedent pulmonary infections. However, the time period to the discovery of PCG ranged from three to 40 years. In many cases PCG presented as solitary, circumscribed tumor-like masses, nodules or coin-like lesions. However, ill-defined, pneumonia-like density was observed in some cases. Calcification of cavitation within the lesion has been reported in a minority of cases (1–3), while the majority of the lesions were located in the pulmonary parenchyma with almost equal frequency in either lung (1–3). Endobronchial growth of PCG is uncommon and has been reported only rarely. Mehta et al found only nine cases with PCG of endobronchial location in previously reported cases in the literature (6). Of the seven cases presented herein, two showed endobronchial location. One was within the segmental bronchus and the other had intrabronchial, tongue-like extensions of the parenchymal mass to the subsegmental bronchus, where they presented as obstructive polypoid masses.

Histologically, PCG is a type of benign tissue proliferative response; it consists of a variety of inflammatory and mesenchymal cells, including fibroblasts, histiocytes, lymphocytes, plasma cells and other inflammatory cells

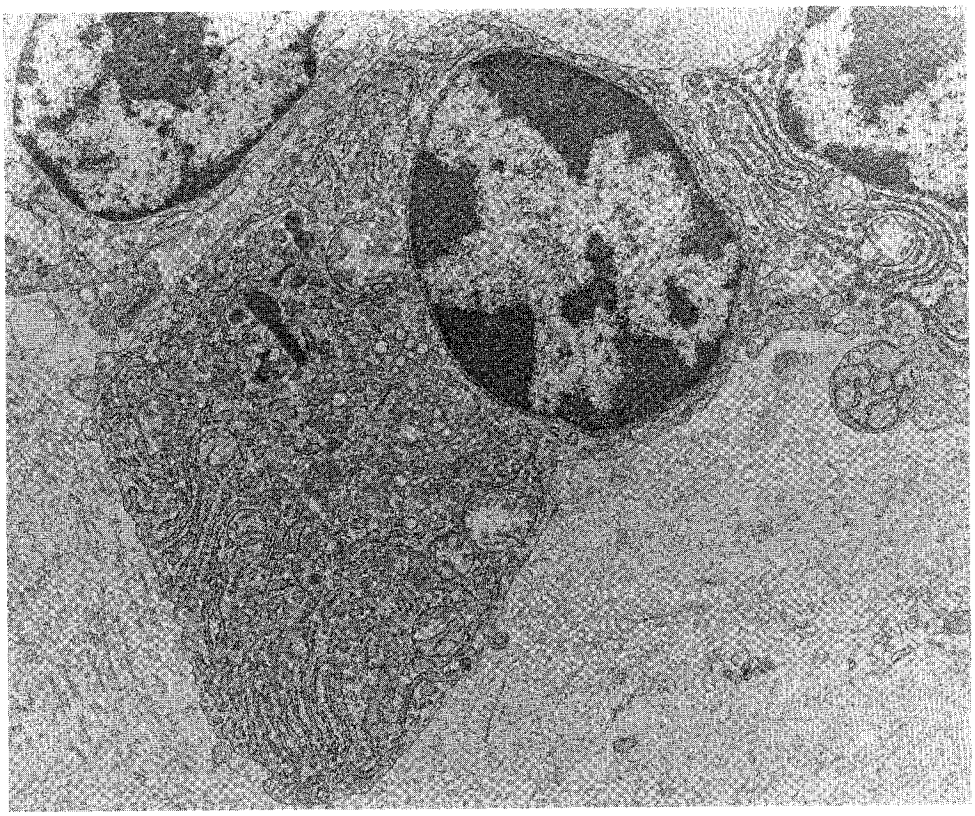

Fig. 15. Electron microphotograh of a plasma cell, showing peripheral clumping of nuclear chromatin, closely packed rough endoplasmic reticulum, prominent Golgi apparatus, and dense bodies. Case 3. (×6,000)

of varying density. Reflecting the variety of cellular components, many designations have been used in the literature, including xanthoma, xanthofibroma, fibroxanthoma, xanthogranulmoa, and histiocytoma (1–3). Mature plasma cells are the predominant and most consistent cellular component, thus the term "plasma cell granuloma" appears to be appropriate (1). However, since most cases have been reported to consist of inflammatory cells and mesenchymal stromal tissue in a circumscribed nodule, the term "inflammatory pseudotumor" is preferred by some authors (2). Histologic appearance varies markedly from area to area in the same case, varying from vascular granulation tissue heavily infiltrated with both plasma cells and lymphocytes to a storiform pattern of fibroblasts and young fibrocytes (a feature frequently reported as pulmonary histiocytoma); it can vary further to complete hyaline fibrosis with or without calcification. Plasma cells are mature in appearance and may, rarely, be multinucleated or contain Russell bodies. Mitoses are not observed. Immunohistochemical staining in the present study revealed the polyclonal nature of the plasma cell proliferation, implying a reactive inflammatory process rather than a neoplastic one. Similar immunohistochemical findings have been reported by others (7, 8).

Mild to moderate cellular pleomorphism is occasionally found in fusiform fibroblastic or fibrocytic cells in PCG, but mitoses are never observed. Although PCG is benign and does not metastasize, and most reported cases have been confined to the lung, a few cases have exhibited infiltrative features that, extending to neighboring organs, produce esophageal and superior vena-cava obstruction (1, 8–10). Further, as recently reported by Warter et al, some of these lesions have an angioinfiltrative nature (11). In the present series of 7 PCG cases, four showed vascular invasion of medium-sized blood vessels in the peripheral parts of the lesion, although all cases were histologically benign. Thus, an angioinvasive growth pattern may not be an infrequent occurrence in PCG, as was indicated by Warter et al (11). Malignant changes, however, may rarely occur in some cases (12). Spencer found two cases with malignant counterparts (malignant histiocytoma) among 27 PCG cases (12). Malignant histiocytoma can only be differentiated from PCG by presence of cellular hyperchromatism, large polymorphic tumor cells, atypical giant cells, and increased mitotic activity (12).

Speckled calcification within the lesion is rarely identified on plain radiography; it was noted in eight of the 181 cases reviewed by Berardi et al (2). Bahadori and Liebow reported two instances of calcification among 40 cases in their series (1). Ultrastructurally, we found

that the PCG was composed predominantly of spindle-shaped or fusiform mesenchymal cells with mature plasma cells and a varying number of other inflammatory cells. Although the spindle-shaped or fusiform cells showed a broad morphologic spectrum of mesenchymal cell nature, including undifferentiated mesenchymal cells, fibroblasts, myofibroblasts, and fibrocytes, myofibroblasts were the most characteristic cells among these mesenchymal cells of PCG. Myofibroblasts, which have the ultrastructural features and many functional properties of both fibroblasts and smooth muscle cells, were suggested by Gabbiani et al (13) and by Majno et al (14), in 1971, to be the specific cells that are present in granulation tissue during wound repair. Since then, myofibroblasts have been demonstrated in various reactive proliferations of mesenchymal tissue, including fibromatosis, a variety of fibrous tumors and granulation tissues, liver cirrhosis, sarcoidosis of the lung, and in the stroma of invasive and metastatic carcinoma (15, 16). Although the histogenesis of myofibroblasts is not well established, recent studies indicate that they derive from at least three different cell types, namely fibroblasts, smooth muscle cells, and pericytes (17). In granulation tissues, myofibroblasts are expressed transiently, but fibroblastic cells which display at the ultrastructural level, similar morphologic features to smooth muscle cells, have been described in diverse normal organs [in the external theca of the rat ovary, the murine adrenal gland capsule, the pulmonary septa of various mammalian species, in rat, rabbit, and human intestinal mucosa, and in the periodontal ligament of the rat (17)]. Although the precise function of these mesenchymal cells is not yet fully understood, they might have contractile properties in these organs and tissues (17). Irrespective of the precise histogenesis, it is suggested that the myofibroblast is an integral part of inflammatory granulation tissue and reactive connective tissue proliferation, and it may play an important role in wound healing by providing the force for contraction (13, 14, 17).

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