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Histopathological study of corpora amylacea pulmonum

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Summary. In this paper, we present a rare disorder which is known as corpora amylacea pulmonum. X-ray CT scanning showed an abnormal focus of the lung as a solitary mass with high density and spicular features around the surface. The resected lung tissue was characterized by the appearance of round, concentrically laminated acellular bodies about 40-80 microns in diameter. The bodies were usually found lying free in the alveolar space and surrounded by the exudate alveolar macrophages or multinuclear giant cells. Some of these macrophages were in a state of progressive degeneration. The bodies showed an affinity for Congo red and exhibited partial birefringence. Moreover, all the bodies had a strong positivity for the PAS reaction and anti lysozyme antibodies. The exudate alveolar macrophages and multinuclear giant cells also displayed reactivity for PAS and lysozyme in a similar manner to that of the bodies. Electron microscopically the bodies were fundamentally composed of fibrillar elements, which bore some resemblance to amyloid fibrils and probably accounted for the partial affinity of the bodies for Congo red. These amyloid-like fibrils were also found in the cytoplasm of the macrophages. This suggested that the concentrically laminated bodies in corpora amylacea pulmonum might be formed by sequential aggregation, fusion, coalescence and compaction of degenerated alveolar macrophages.

Key words: Corpora amylacea, Lung, Human, Ultrastructure, Amyloidosis

Introduction

Pulmonary corpora amylacea is a very rare disease characterized by round, homogeneous or laminated deposits in the alveoli of the lungs, first described by Friedreich (1856) as «*corpora amylacea pulmonum*». In 1957 Michaels and Levine reported on these deposits with a description of histological and histochemical findings as well as electron microscopic appearances. The corpora amylacea are usually found freely in the alveolar space as concentrically laminated bodies. Electron microscopically they are composed of peripheral fibrils and a central core. With regard to their origin, it is suggested that the bodies are formed by the aggregation of alveolar macrophages or of some protein products. However there is no consensus of opinion concerning either the cause and origin of corpora amylacea or the mechanism of their formation.

Recently we got a chance to observe a case of pulmonary corpora amylacea, which was resected under suspicion of lung cancer. In our light and electron microscopic study, we show various specific types of the bodies and attempt to clarify the developmental process of the laminated bodies.

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Materials and methods

Case

The case was a 75-year-old Japanese woman. She was quite well until a periodic checkup, when her chest radiograph revealed an abnormal, high density area in the right lung field (Fig. 1). The patient was admitted to Tendo Onsen Shinoda Hospital for further examination. Her general condition was good. Physical laboratory analysis did not result in any abnormal data. However, X-ray CT scanning showed an abnormal mass lesion with high density area in the lateral segment of the right middle lobe (Fig. 2). The size was about 2 × 2 cm and it had spicular features on the periphery. No pleural effusion was noticed. No tumor cells were observed in cytological examination of bronchoalveolar lavages and repeatedly smeared sputa. Lobectomy of the middle lobule was performed under suspicion of lung cancer,

because of a slight increase in the abnormal region. A tumorous mass was found in the S4 segment of the extirpated lobule.

The first pathological diagnosis of the dissected lung tissue is reported in the following description. The main component of the tumorous mass consisted of granulation tissues, which were composed of epithelioid cells, multinucleated giant cells and lymphocytes. These tissues were similar to tubercles, but tubercle bacili could not be detected in the section stained with Ziehl-Neelsen's method. A characteristic finding was eosinophilic amorphous bodies in the alveoli.

Procedure for light microscopical specimens

For further detailed examinations, various different portions of the dissected lung tissue which had been stored in 10% formalin were routinely re-embedded in paraffin. Newly-prepared specimens were cut to a thickness of approximately 3 microns, deparaffinized in xylene and placed in absolute alcohol. Subsequently, the sections were stained by means of histochemical procedures and immunoperoxidase techniques. Histochemical reactions are summarized in Table 1, which refers to Michaels and Levine's results (1957) for a comparison with our results of histochemical tests of corpora amylacea. In the immunoperoxidase techniques, two methods were applied. One was the PAP method and the other the indirect immunoperoxidase method, using various monoclonal antibodies against human tissue antigens. In the PAP method, the sections were sequentially incubated with rabbit anti-human antiserum, swine anti-rabbit serum IgG antiserum and PAP. In the indirect method, the sections were incubated in monoclonal mouse anti-human antiserum and subsequently peroxidase-labelled goat anti-mouse immunoglobulins. Phospate buffer (0.05 M, pH 7.6) was used for all dilutions and washing. The site of antibody binding was determined with a diaminobenzidine reaction. Immunohistochemical reactions are summarized in Table 2.

Procedure for electron microscopical observation

Formalin-fixed lung tissue was cut into small blocks of about 1 mm^3 and washed in cacodylate buffer three times. The materials were sequentially fixed in cacodylate-buffered 2% glutaraldehyde for 4 h and 1% osmium tetroxide for 1 h. The materials were then dehydrated in graded alcohol and embedded in epoxy resin. Ultrathin sections were counterstained with uranyl acetate and lead citrate and examined with a Hitachi HS-9 electron microscope.

Results

The removed lung tissue provided highly varied histopathological findings, which showed evidence of granuloma, remarkable exudation of macrophages, and bleeding. The granuloma was composed of many various-sized nodules which bore a close resemblance to tubercles. Some nodules showed a remarkable presence of multinuclear giant cells in the layer of the epithelioid cells surrounded by lymphocytes. However, no caseous necrosis was found in any nodules nor could tubercle bacili be detected (Fig. 3).

General structure of corpora amylacea

In the present case pulmonary corpora amylacea were usually observed free in the alveolar lumen, in close contact with the alveolar wall or in the interstitial connective tissue. They were eosinophilic in hematoxylin and eosin staining. Occasionally they appeared in clusters or were scattered in the peripheral portion of the granuloma. They were spherical and about 40-80 microns in diameter.

Usually the small corpora were round, acellular bodies which were very similar to multinuclear giant cells in shape and size. They were rather homogeneous and neither concentric layers nor radial stripes could be recognized (Figs. 4, 5). Some corpora included adhesion of a few mononuclear cells on their surfaces and others were completely surrounded by a continuous layer of the cytoplasmic processes of mononuclear cells. These mononuclear cells could be discriminated as alveolar macrophages by their active phagocytosis, deposits of coal dust and the results of histochemical and immunohistochemical staining. The surface demarcation line between the corpora amylacea and the surrounding macrophages was not well defined. There was also considerable resemblance between the small corpora and the aggregated alveolar macrophages which had reached a stage of progressive degeneration (Fig. 6). These findings indicated that the degenerated alveolar macrophages had coalesced to form the bodies. Medium-sized corpora amylacea were often enclosed by multinuclear giant cells, all more or less similar in size to the bodies (Fig. 7). Occasionally the bodies showed nuclear chromatin-like materials or a darker basophilic core in the center. Large corpora involved a darker center with various features and a paler periphery. In some corpora, concentrically laminated structures were noted at the intermediate zone between the center and the periphery. Radial stripes were also observed at the periphery of large corpora (Fig. 8). Other specific findings were concentric deposition of carbon-like particles and thin rods of crystalline inclusions, which often appeared in the center of large bodies (Figs. 9, 10).

Histochemical and immunohistochemical findings

The results of histochemical and immunohistochemical studies are summarized in Table 1 and Table 2 respectively. The main findings were as follows.

In the histochemical investigation of corpora amylacea, noteworthy results were obtained with Congo red and PAS staining. When Congo red stained sections were observed with polarizing microscopy, the peripheral

portion of the bodies, which were composed of radial fibrillar stripes, showed strong birefringence (Figs. 11, 12). Usually, large bodies displayed a Maltese-cross appearance and a fine lamina of rings with special polarizing properties and yellowish green in color. On the other hand, the small- or medium-sized bodies, without fibrillar stripes, did not show any binding affinity for Congo red. Both the multinuclear giant cells and the alveolar macrophages were negative for Congo red. In sections stained with PAS, the location of corpora was easily recognized because of strong positivity. The PAS reactions were rather homogeneous in all types of corpora, not only in the large but also the small bodies (Fig. 13). Additional PAS reactivity was found on the giant cells and macrophages. In their cytoplasm, the reaction products appeared as granular or vesicular deposits (Fig. 14). Coexistence of lipids, nuclear acid, calcium or iron could not be detected in the bodies. The localization of protein components was also obscure.

All tissues examined with the immunoperoxidase technique showed excellent morphologic detail. The reaction products were readily detected as dark brown deposits at the antibody binding sites, which showed sharp contrast as a result of hematoxylin counterstain. Corpora amylacea did not react against anti-AA, a common marker of secondary amyloidosis. P. component (P. comp.), which can usually be detected in amyloid deposits, was also negative. For the immunohistochemical method, lysozyme was the most effective marker for detection of corpora amylacea, because all types of corpora showed a strong, well-defined positivity for anti-lysozyme antibody (Fig. 15). In many cases, small corpora showed amorphous staining without concentric features or radial fibrillar components (Fig. 16). Sometimes large corpora amylacea showed distinct concentric stratification and delicate radial striation of fibrous components (Fig. 17). Lysozyme activities were also found on the macrophages, epithelioid cells, multinuclear giant cells and neutrophils. Most of the macrophages in the alveoli showed lysozyme activity, although their staining varied from strong to weak. The reaction products were localized in the cytoplasm as fine granular deposits. The epithelioid cells and multinuclear giant cells in the granulomas were moderately positive for intracytoplasmic lysozyme. In the cytoplasm of the multinuclear giant cells two types of localization patterns of lysozyme activity were recognized. They could be classified as the diffusely homogenous pattern and the nodular concentrated pattern which was situated at the center of abundant cytoplasm (Fig. 18). The intracytoplasmic distribution pattern of lysozyme activity was very similar to that of the PAS activity in Fig. 14. Except for lysozyme, all other markers of macrophages which included CEA, α_1-AT, α_1-ACT, MT-1, Leu M-1 and ferritin showed negative results.

Kappa (κ) and lambda (λ) light-chains, IgG, IgM, IgD, IgE, IgA, and secretory component (S. comp.) were detected around the surface of the corpora amylacea, but

Table 1. Histochemical reactions of corpora amylacea

Test for	Our results	Michael's results
Congo red	Partially positive	
Ziehl-Neelsen's method for TB	−	
Polysaccharide		
PAS	+	Moderate +
PAS after diastase	+	Moderate +
Best carmine	+	
Acid mucopolysaccharide		
Alcian blue	−	−
Metachromasia with toluidine blue	−	−
Lipids		
Sudan IV	−	−
Sudan black	−	−
Proteins		
Coupled tetrazonium H!	±	+
Nucleic acids		
Feulgen	−	−
Methylgreen-pyronine	−	
Prussian blue	−	−
Calcium		
Von Kossa	−	−
PTAH	−	−
Azan	−	
Von Gieson-elastica		

Table 2. Immunohistochemical reactions of resected lung tissue

anti sera	CA	macrophage	giant cell	PML
amyloid AA	–	–	–	–
P. comp.	–	–	–	–
lysozyme	+	+	+	+
CEA	–	+	–	+
α_1-AT	–	+	–	±
α_1-ACT	–	+	±	–
MT-1	–	membrane +	±	membrane +
Leu M1	–	–	–	+
ferritin	–	±	±	±
osteocalcin	–	–	–	–
κ	surface +	partly +	–	–
λ	surface +	partly +	–	–
IgG	surface +	partly +	–	–
IgM	surface +	partly +	–	–
IgD	surface +	partly +	–	–
IgE	surface +	partly +	–	–
IgA	surface +	partly +	–	–
S. comp.	surface +	–	–	–
J-chain	–	–	–	–

Fig. 1. The chest radiograph shows an abnormal high density area (arrow) in the right lung field.

Fig. 2. The abnormal focus in Fig. 1. is observed by CT scan as a high density mass (arrow) with spicular features at the periphery.

Fig. 3. The removed lung shows granulomas composed of tubercle-like nodules together with multinuclear giant cells (arrows). × 30

Fig. 4. In the alveolar spaces, the exudation of macrophages (arrowheads) and the appearance of giant cells (arrow) are remarkable. × 480

Fig. 5. Small corpora amylacea (CA) are round, acellular bodies composed of homogeneous materials. They are very similar to giant cells in size. × 480

Fig. 6. The ghost of a degenerated giant cell with the remnant of the nucleus. × 300

Fig. 7. A medium-sized corpora amylacea (CA) is enclosed by giant cells. It can be gathered from this photograph that fibrillar structures have appeared in the corpora. × 360

Fig. 8. Large corpora amylacea shows a basophilic dark center and a paler periphery. The concentrically laminated structures and the radial stripes are also remarkable. × 400

Fig. 9. A large corpora amylacea which is tightly enclosed by alveolar macrophages. The corpora contains concentric deposits of carbon-like particles. × 400

Fig. 10. The crystalline-like inclusion (arrow) can be recognized at the darker center of a large corpora. × 400

Fig. 11. The peripheral radial stripes of large corpora amylacea show the strong affinity for Congo red. × 360

Fig. 12. The birefringence of the corpora amylacea in Fig. 11. The radial fibrillar stripes show strong birefringence. The Maltese-cross appearance can also be seen. × 360

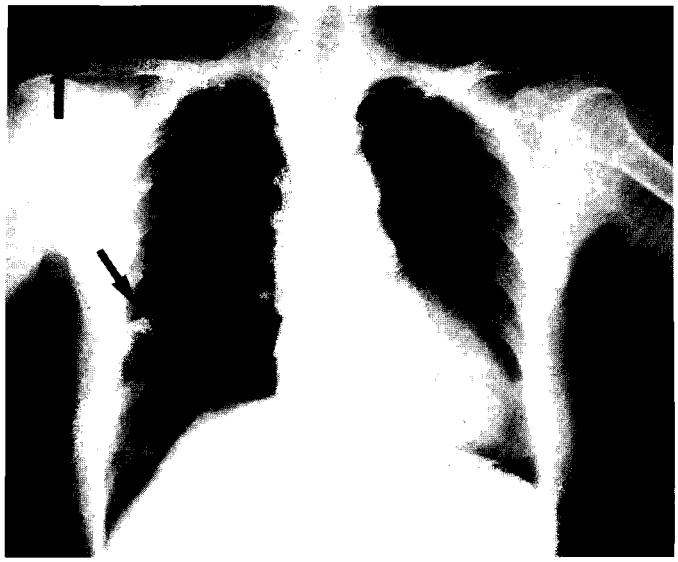
Fig. 13. Generally all corpora amylacea show homogenous PAS-reactivity. However, the PAS-positivity seems to be rather stronger in the small corpora. × 150

Fig. 14. PAS-reactivity is found in the cytoplasm of the giant cells and macrophages. As compared to the corpora amylacea in Fig. 13, reaction products in the giant cells show some granular or vesicular features. × 300

Fig. 15. The activity of lysozyme as detected with the PAP method. All corpora amylacea (arrows and arrowheads) show a strong, well-defined positivity for lysozyme. × 30

Fig. 16. An enlarged view of the bodies indicated by an arrowhead in Fig. 15. × 120

Fig. 17. Lysozyme activity in a large corpora amylacea and macrophages surrounding the corpora. The central core of the corpora and the cytoplasm of the macrophages show moderately positive. The delicate radial striation of the corpora shows weakly positive. × 400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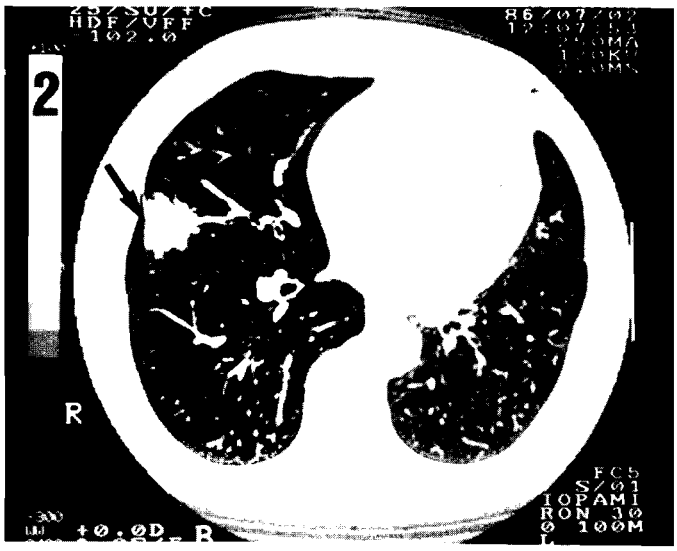
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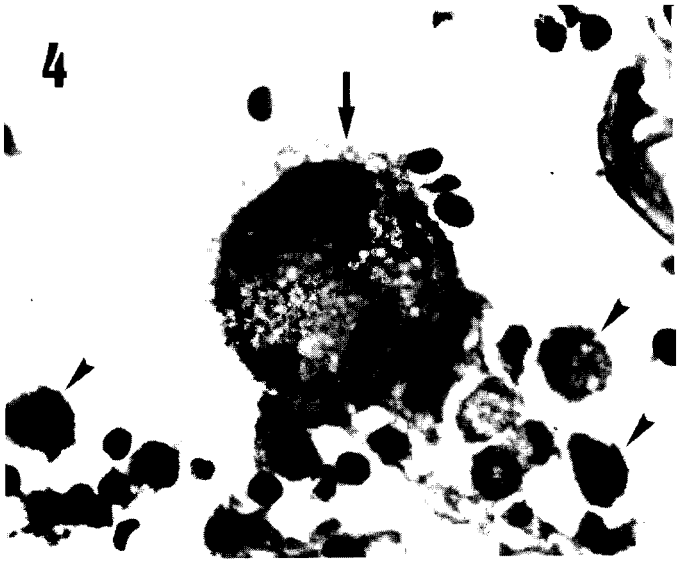
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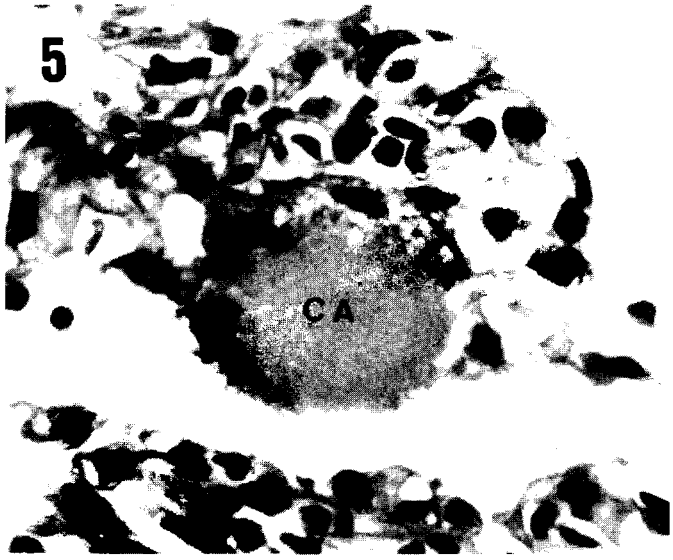
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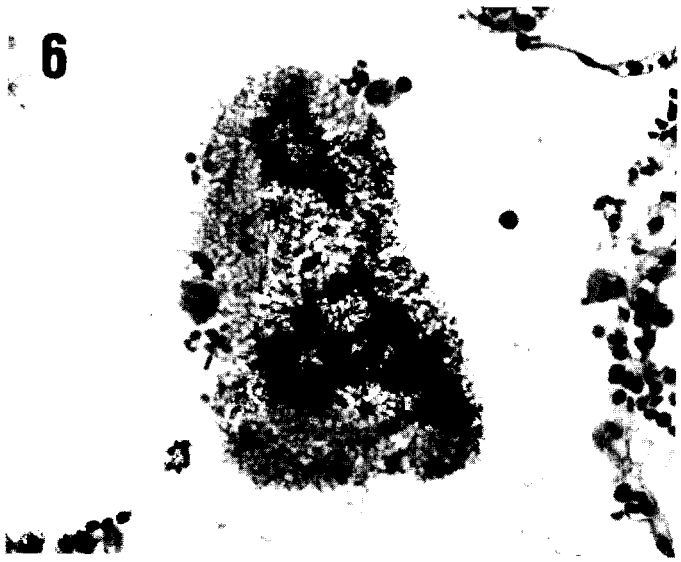
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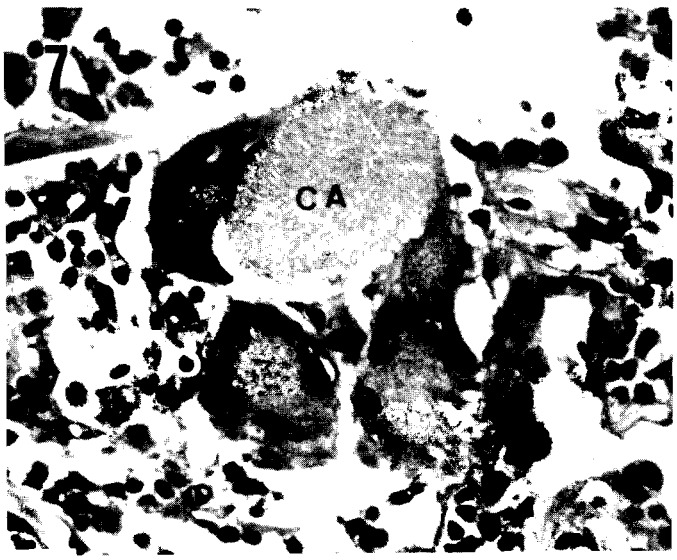
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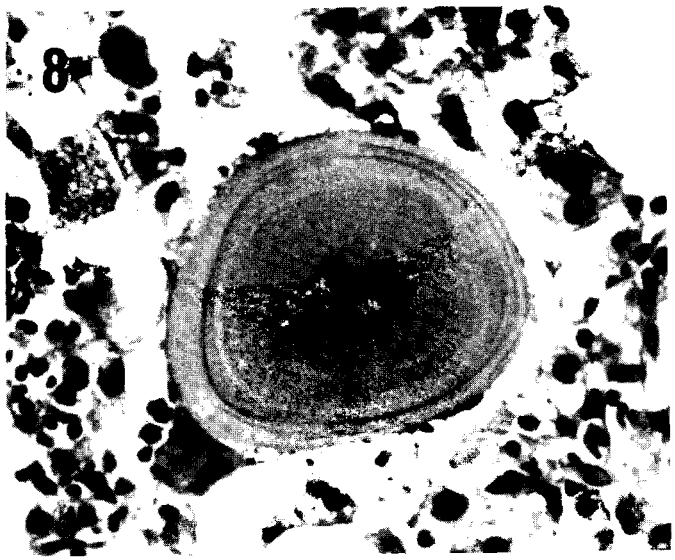
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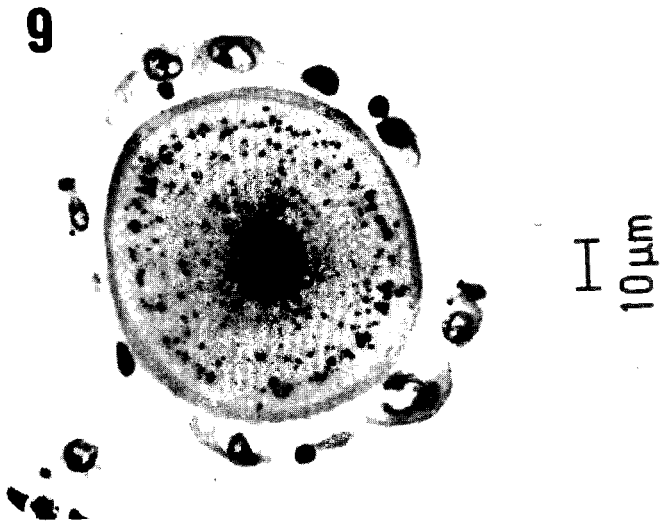
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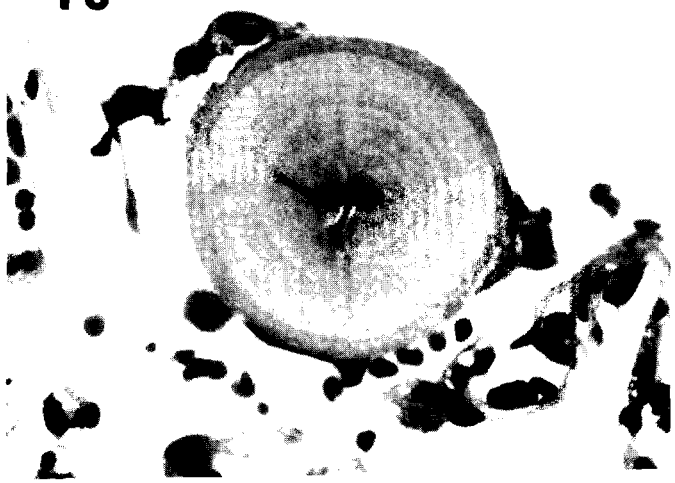
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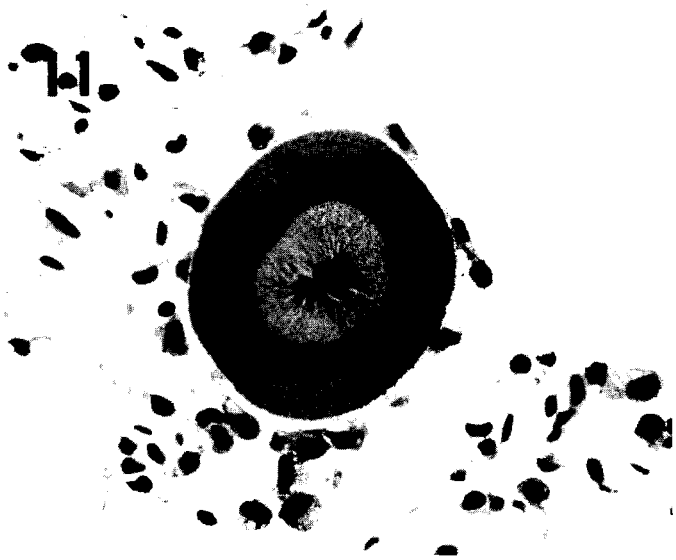
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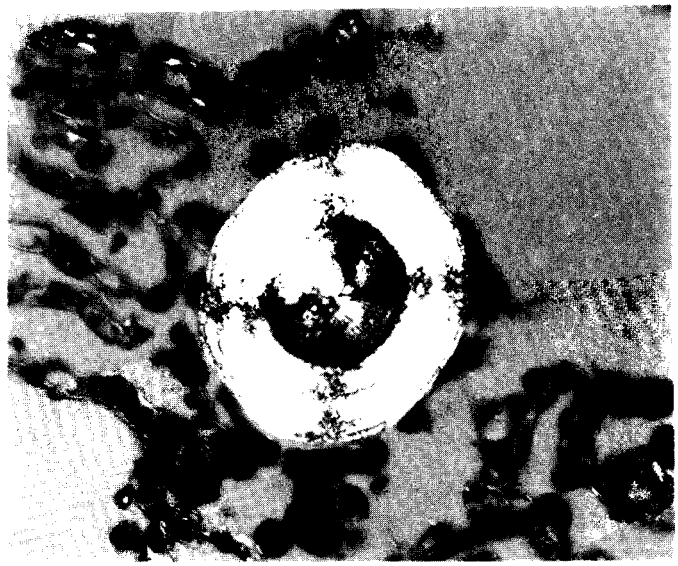

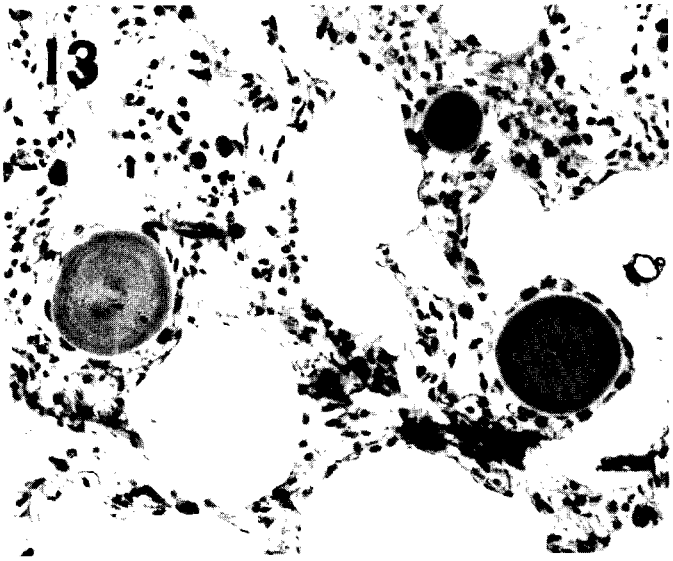
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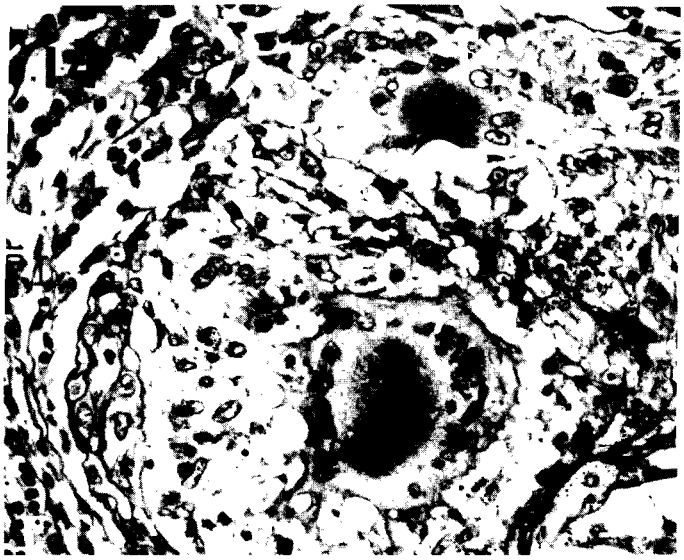

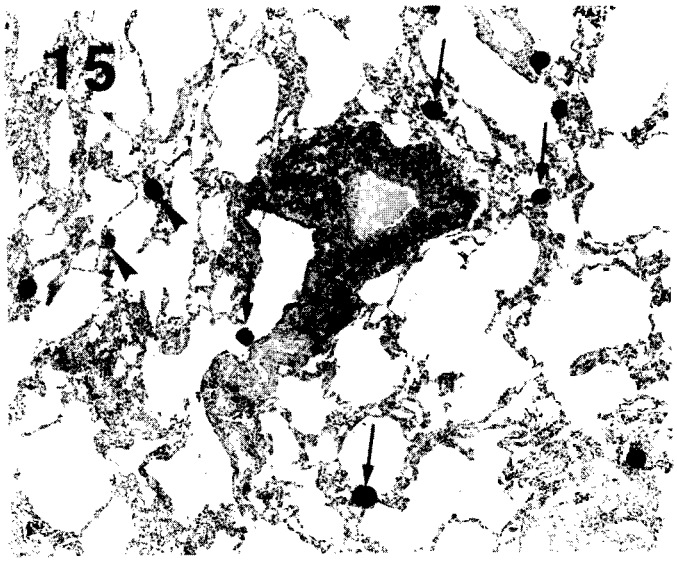
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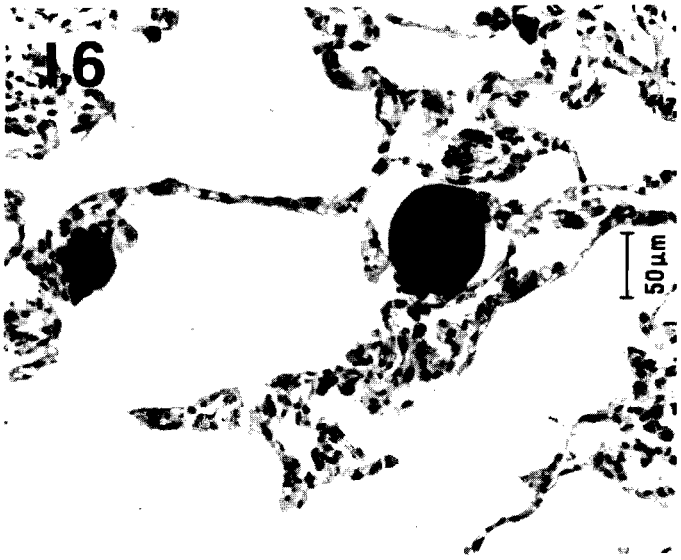
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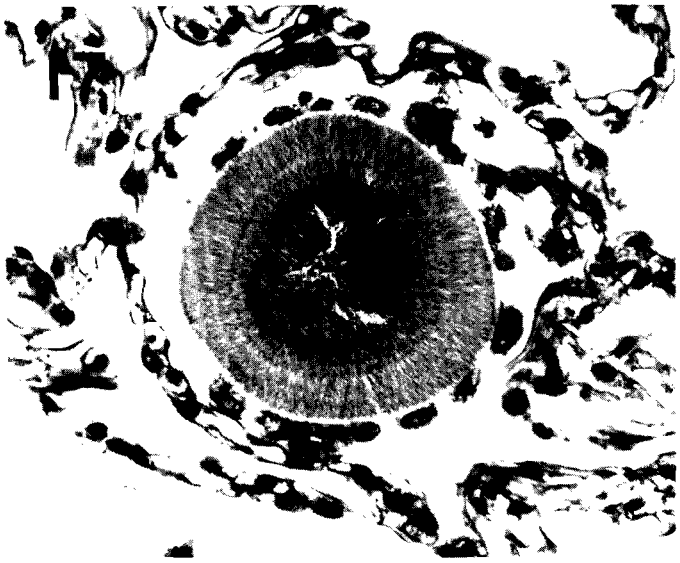
50 µm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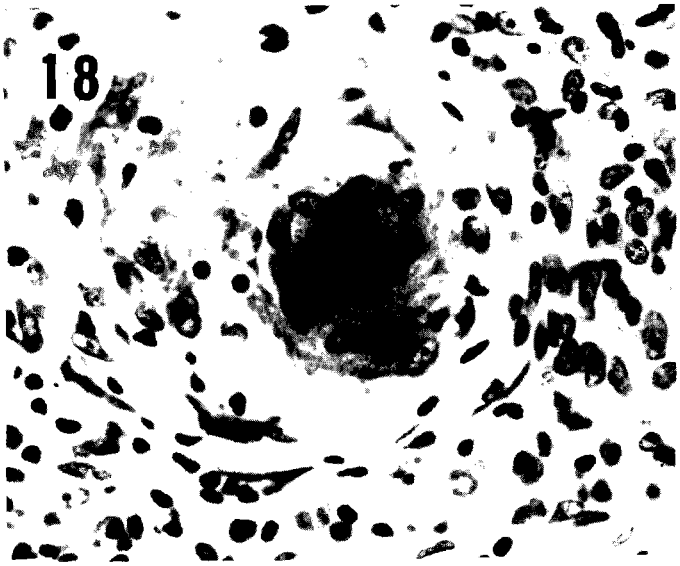
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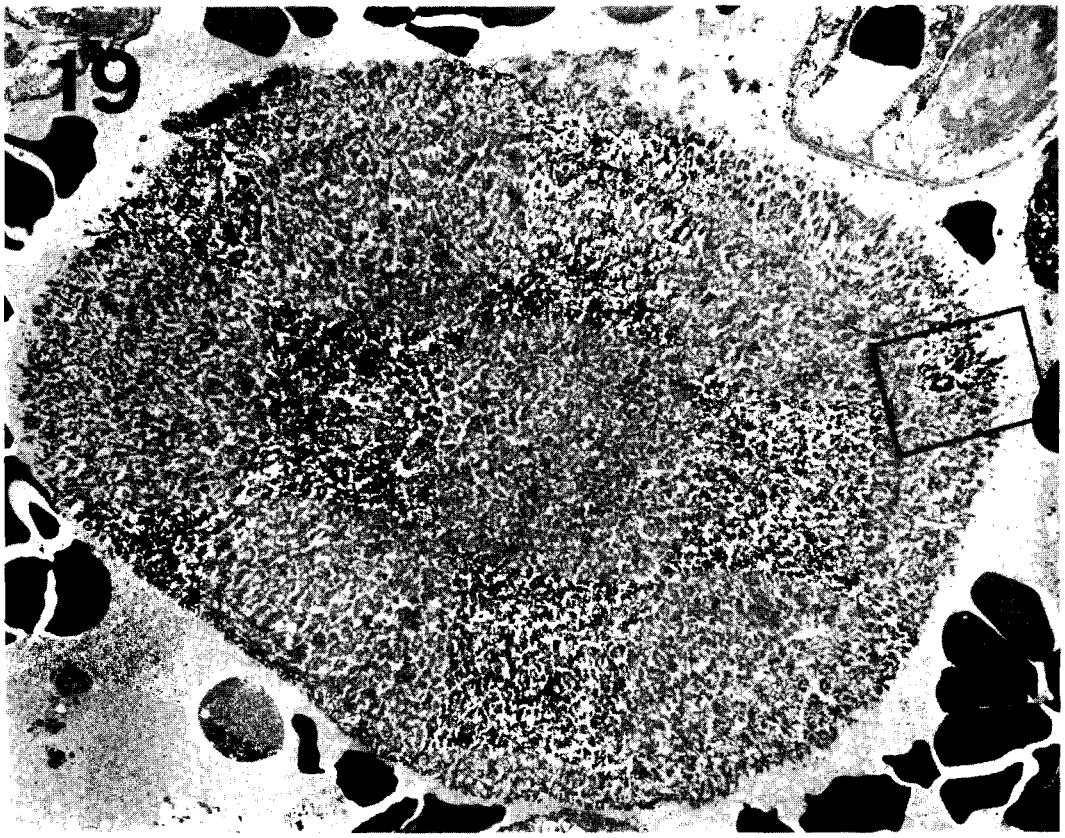
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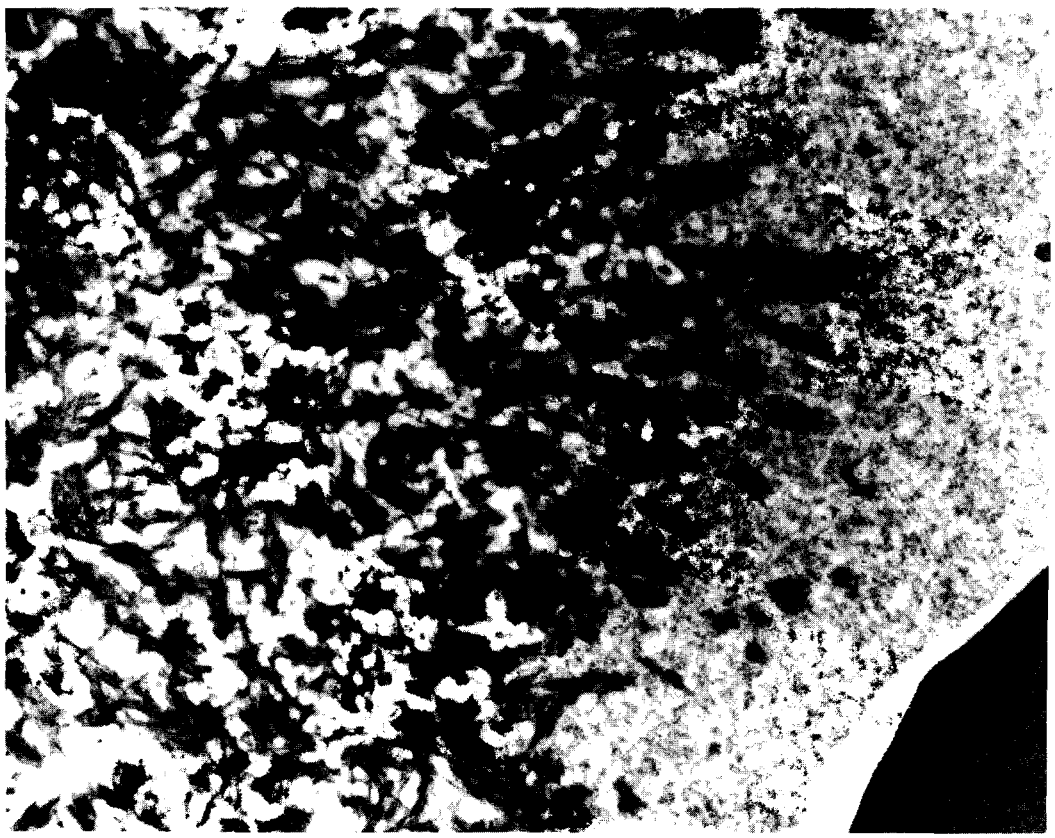

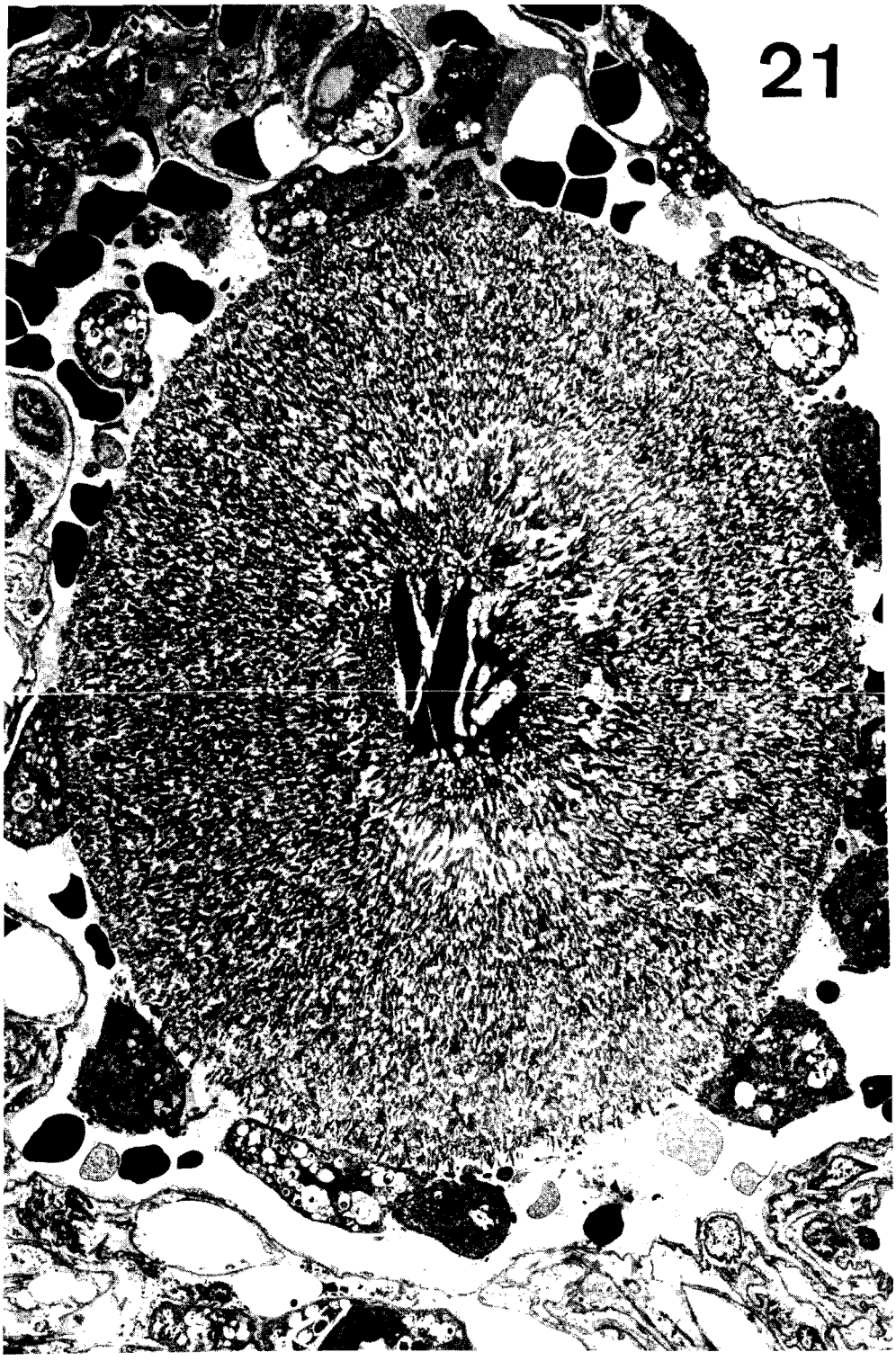
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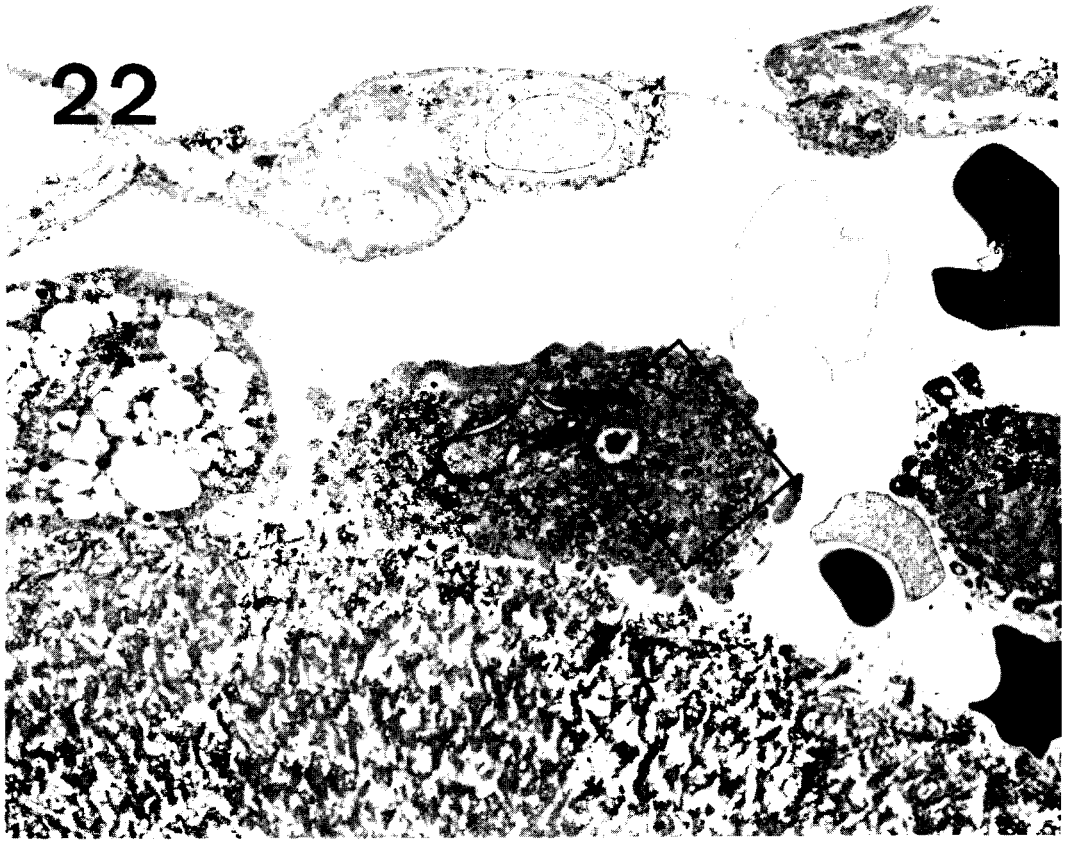
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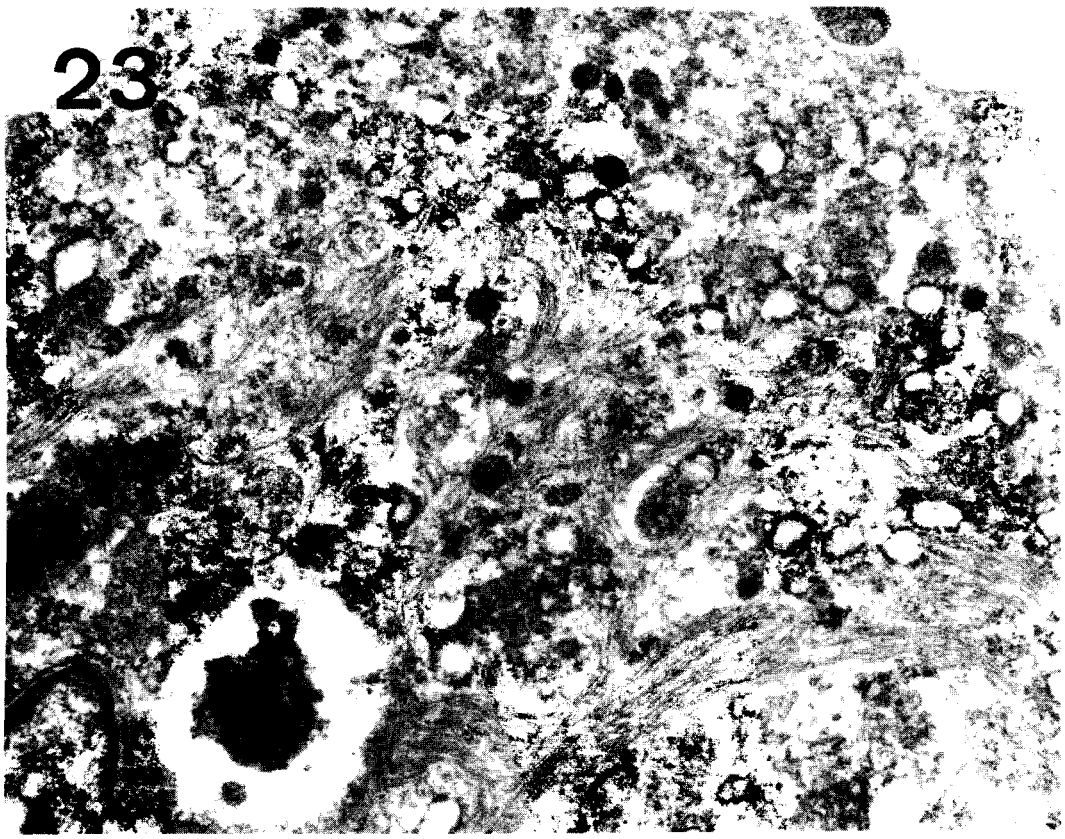
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Fig. 18. A giant cell with lysozyme activity. The reaction products are localized at the center of the cytoplasm of the cell and are nodular in appearance. × 400

Fig. 19. A low-power electron micrograph of a medium-sized corpora amylacea in the alveolar space. × 1,500

Fig. 20. A high-power view of the peripheral radial stripes encircled in Fig. 19. The stripes are composed of the accumulation of short fibril bundles. × 12,000

Fig. 21. A typical large corpora amylacea free in the alveolar space. The fundamental components of the body consist of radiated bundles of fibrils. In the center of the body, crystalline inclusions can be seen. × 1,200

Fig. 22. The exudate alveolar macrophages adhering to the outer surface of the corpora amylacea. × 3,000

Fig. 23. The appearance of fibrillar components in the cytoplasm of the macrophage encircled in Fig. 22. × 12,000

not in the interior of the bodies. The surface positivity correlated with the radial fine stripes. Occasionally, some macrophages contained reaction products against various classes of immunoglobulins in their cytoplasm, but their positivity was assumed to be based on active phagocytosis or pinocytosis of reactive substances.

Electron microscopical findings

Electron microscopically, the fundamental framework of the corpora amylacea was composed of fibrillar elements, 10-20 nm in thickness. The framework of small bodies was entirely covered with fibrils with a homogeneous structure and arrangement. Neither concentric rings nor radial stripes were recognized. In medium-sized bodies, radial stripes of the fibrils appeared at the periphery of the bodies. Compared with the central fibrils, the peripheral fibrils showed some definite construction and arrangement. The peripheral fibrils were closely gathered and formed compact bundles of fibrils (Figs. 19, 20). All of the large bodies were composed of radiating bundles of fibrils. At the outer surface, the fibrils formed compact bundles in parallel arrangement. In addition to the fibrils, two other types of components frequently appeared: carbon-like particles, which were often scattered in concentrical deposits, and crystalline inclusions in the center of bodies (Fig. 21).

Macrophages surrounding the outer surface of the corpora amylacea frequently stuck to the bodies, in which case the demarcation line between the macrophages and corpus was not well-defined. Corpus-adherent macrophages were usually in various stages of degeneration. Their cell membranes were often missing in many places, especially at the demarcation point where the corpora and macrophages came in close contact with each other. In the cytoplasm of degenerating macrophages fibrillar components were often seen (Figs. 22, 23). These fibrils were observed not only in the cytoplasm of macrophages but also in the whole nucleus showing it to be in a state of degeneration.

Discussion

Since Friedreich (1856) first described the round laminated acellular bodies in the alveoli of the lungs, which were known as corpora amylacea pulmonum, several studies on their morphological features or their origin have been published. The electron microscopical appearance of these bodies, together with a description of histological and histochemical findings were presented by Michaels and Levine (1957). More recently, Spencer (1977) described these entities as degenerative and metabolic disorders.

In the present case, one of the interesting findings in the resected lung was the formation of a granulomatous focus which involved the appearance of corpora amylacea. The cause of the granuloma remains obscure in spite of a thorough examination. Corpora amylacea were usually found free in the alveolar space and were approximately 40-80 microns in diameter. Typical bodies were eosinophilic in hematoxylin and eosin staining. The darker center could be distinguished from the paler periphery with radial stripes. Frequently the exudate macrophages adhered to the outer surface of the corpora amylacea and sometimes a monolayer of macrophages completely encircled the circumference of the corpora amylacea. These findings of ours closely resemble those of previous reports in spite of the solitary appearance of the focus with an abnormally high density mass.

Congo red staining combined with polarizing microscopy is considered to be one of the most effective techniques for the detection of amyloid substances and is in general use for the diagnosis of amyloidosis. In the present study, concentrically laminated bodies with radial stripes showed an affinity to Congo red and always showed vivid birefringence. However, the small homogeneous bodies showed no affinity to Congo red. Our ultrastructural findings showed the presence of amyloid-like fibrils in the corpora amylacea. In the large corpora, some fibrils frequently formed bundles at the periphery of the bodies. Presumably the birefringence of large corpora is caused by these bundles of fibrils. Fibrillar structures were also found in the cytoplasm of alveolar macrophages but never showed any affinity to Congo red. This suggests that the corpora amylacea transform in the sequence of components or in the amylacea trnsform in the sequence of components or in the tridimensional structures of the components. It is well-known that amyloid A protein (AA) is a major component of the fibril deposits in secondary amyloidosis. Chronic inflammatory disease has been cited as one of the causes of secondary amyloidosis. In our case, the corpora amylacea were found in the pulmonary focus with a granulomatous appearance. However, the corpora showed no reactivity against anti AA serum.

With regard to PAS reaction, Steele et al. (1952) reported that corpora amylacea of the prostata, the lung, and the brain contained a polysaccharide. Michaels and Levine (1957) also made histochemical studies and concluded that the corpora amylacea consisted of protein together with a polysaccharide component. In the

present study the corpora amylacea, exudate macrophages and multinuclear giant cells showed positivity for the PAS. The small corpora and macrophages displayed granular staining pattern to the PAS. On the other hand, the large corpora demonstrated a rather homogeneous staining pattern. This reactivity against PAS provided a valuable piece of information about the developmental processes of corpora amylacea.

Motoi et al. (1984) showed that most alveolar macrophages appearing in the lungs as the result of various inflammatory processes or circulatory disturbances were weakly positive for lysozymes. Miyauchi et al. (1985) investigated the ultrastructural localization of lysozyme in human mononuclear phagocytes and demonstrated that lysozyme was present in the granules and released into phagosomes. In our immunohistochemical studies, lysozyme was the most useful for the detection of the corpora amylacea because of their definite positivity. Alveolar exudate macrophages also demonstrated varying staining patterns for intracytoplasmic lysozyme. Moreover, epithelioid histiocytes and multinuclear giant cells contained easily detectable intracellular lysozyme. Usually the reaction pattern of lysozyme in the macrophages was granular, but in the corpora amylacea it was rather homogeneous. Among these different reaction patterns, transitional forms were often observed.

With regard to the source and cause of the formation of corpora amylacea, we considered that the corpora amylacea had originated from degenerating macrophages. The presence of numerous amyloid-like fibrils in the cytoplasm of exudate macrophages surrounding the corpora amylacea showed that the fibril components of the bodies may have their origin in the alveolar macrophages. The concretions in large bodies suggested that the bodies might have grown through sequential adhesion and degeneration of macrophages. The initial stage of their formation involved the aggregation, fusion, coalescence and compaction of degenerated macrophages or multinuclear giant cells. It is assumed that the concentric laminated layer of large corpora amylacea was formed by the sequential accumulation and adhesion of degenerated macrophages surrounding the corpora amylacea. These processes are very similar to those thought to be responsible for the origin of the udder of the cow. In 1901 Ottolenghi reported on corpora amylacea in the mammary gland of the cow. They were observed as round or oval inclusion bodies, 30-250 microns in diameter with distinct circular stratification and radial stripes. They were strongly refractive and were found free in the cavity, within the epithelium cells or even in the interstitial connective tissue and were considered by Ottolinghi to be amyloid in nature. In nearly all cases, multi-nuclear giant cells and other large uninuclear cells surrounded the bodies. McFadyean (1930) expressed the opinion that amyloid bodies in the mammary gland were formed from degenerated epithelial cells or leukocytes. It appeared that the nuclei of the epithelial cells underwent amyloid degeneration and that these nuclei united with the degenerated protoplasm and the albumin of the surrounding menstruum and thus gradually built up the bodies.

The occurrence of intracellular amyloid fibrils has been suggested by numerous authors. Dobashi et al. (1986) reported a case of systemic amyloidosis (AL) in which nodular amyloid deposits were frequently enclosed by the longitudinally stretched cytoplasmic processes of abutting reticulum cells and/or macrophages, and amyloid fibrils were formed in the cytoplasm of such cells. Furthermore, the same authors (Dobashi et al.) observed spontaneously occurring amyloidosis in white Pekin ducks and found intracytoplasmic formation of amyloid fibrils in mononuclear phagocytes, especially histiocytes. These processes suggested some similarity in the mechanisms of fibril formation in true amyloidosis and in corpora amylacea, but definite proof of the occurrence of intercellular fibrils must await more detailed studies.

In 1933 Puhr reported a case of remarkable pulmonary calcification in which whole alveoli were covered by spherical, concentrically laminated, calcified bodies. The presence of numerous exudate macrophages, the occasional appearance of multinuclear giant cells and a small number of non-calcified laminated bodies were also recognized. The appearance of these bodies was similar to that of corpora amylacea. However, Puhr distinguished this condition from corpora amylacea and named it microlithiasis alveolaris pulmonum. On the other hand, Michaels and Levine (1957) concluded that microlithiasis bodies and corpora amylacea were fundamentally similar in nature, except for the heavy calcification in microlithiasis bodies, and that they probably arose in the same way. In 1963 Baar showed an intermediate stage between the corpora amylacea and microlithiasis pulmonum. The laminated concretions in both cases, corpora amylacea and microlithiasis alveolaris pulmonum, were diffusely and evenly distributed throughout both lungs. However our present case of corpora amylacea showed a solitary mass together with the appearance of granulation tissues.

Recently Robitaille (1980) showed a close resemblance between corpora amylacea, Lafora bodies, Bielschowsky bodies, deposits in type IV glycogenosis, and certain intra-neuronal and intra-axonal bodies. Electron microscopically it has been demonstrated that all these structures are formed from branching filaments about 8 nm in diameter. Histochemically they stain strongly with PAS, silver proteinate and iodine. Biochemical studies have shown that corpora amylacea and Lafora bodies are composed principally of glucose polymers (polyglucosans), with a small variable component of phosphate and sulphate groups, and not more than 5% of associated protein. Therefore, Robitaille proposed a general term - polyglucosan bodies- which seems to be a justified and useful common genetic name. In 1987 Yokota et al. showed that corpora amylacea, the basophilic degeneration of myocardium and deposits of type IV glycogenosis contained materials which were antigenically common to Lafora bodies. These results have further aroused our interest in the research of amyloidosis.

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