PNEUMOCONIOSIS FROM EXPOSURE TO KAOLIN DUST: KAOLINOSIS *

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Although the term kaolinosis occurs in a widely circulated medical dictionary, the name is apparently not used in the few available published reports of pneumoconiosis associated with exposure to the inhalation of dust of clay. At the present time there are only two proved varieties of chronic pulmonary disease developing from the inhalation of particulate matter in the atmospheres of industrial plants or workings: silicosis and asbestosis. The present report sets up a definite disease that may be disabling and may reach an advanced or even fatal stage. While the condition to be described has certain features that are not clearly distinguishable from silicosis, it has other features which warrant the special designation, at least until some particular part of kaolin may be shown to be the disease-causing factor. Inasmuch as there are a few published records of pneumoconiosis occurring in "fuller's earth" workers, such comparisons as seem warranted also will be made with that condition.

In a report by Campbell and Gloyne[1] upon a necropsied case in 1942 in which exposure to the dust of fuller's earth in Surrey, England, was of 38 years' duration, the condition was described as scattered, irregular, black, fibrotic patches from ¼ to ¾ inch in diameter, not considered to be characteristic of silicotic nodules. Also described is thickening of the walls of the bronchioles, perivascular fibrosis of arterioles, and "reticular" fibrosis of alveoli surrounding dust particles. In the clay concerned in that report montmorillonite was the main constituent while sericite and kaolinite were not present. The soft patchy pneumoconiosis without massive fibrosis differed from silicosis.

McNally and Trostler[2] found in roentgenologic examinations in 1941 of 49 men working in fuller's earth in Illinois a few with densely mottled areas in the upper lobes of the lungs, but in most of these men there were merely increased bronchial markings.

The few other reports cited by these writers[2] and the paper by Tonning[3] indicate the condition observed as "reticular" fibrosis without silicotic nodulation or massive fibrosis.

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Kaolin

No attempt will be made here to give a detailed analysis of the composition of kaolin as it occurs in different deposits in various parts of the world, nor to determine whether any particular component of any deposit of kaolin, including free silica (SiO_2), may be the actual offending substance. Perhaps it will be acceptable for the present purpose to define kaolin simply as a kind of clay derived by disintegration of an aluminous mineral such as feldspar or mica. It is commonly called china clay, and in simple terms clay may perhaps be defined as a dispersed system of mineral fragments of hydrated aluminum silicate in which particles smaller than 2 μ predominate. The term clay refers to a physical condition and not a definite chemical composition. The elements which may be found in kaolin deposits are: silicon (SiO_2), aluminum (Al_2O_3), iron (Fe_2O_3), titanium (TiO_2), calcium (CaO), magnesium (MgO), potassium (K_2O), sodium (Na_2O), manganese (MnO), copper (CuO), and sulfur (SO_3), with water, carbon, and organic matter. The essential constituent of clay or kaolin is the mineral kaolinite, a hydrated aluminum silicate.

The material from which the dust concerned in this study came was apparently derived by open mining and was processed in an industrial plant for distribution and use in various industries. The exposure apparently occurred in such a processing plant. In previous times, before the recognition of such hazards or possible hazards, doubtless some phases of the processing created very dusty atmospheres. So far as our own observations go, those conditions seem to have been largely corrected, but this study shows that wherever workers are exposed sufficiently to inhalation of air heavily laden with particles of kaolin, at least some may develop chronic disease of the lungs as a consequence.

An additional factor that may need to be taken into account is from the addition of chemicals in the course of processing the clay. Those sometimes used are soda ash, trisodium phosphate, and sodium pyrophosphate.

Pathologic Studies

The material concerned in this study consists mainly of the lungs of two men who had worked for considerable periods in a kaolin processing plant. Upon the death of these two persons, necropsies were performed elsewhere and the lungs were sent to us for examination. Subsequently, we were furnished with the chest roentgenograms* of

* The descriptions and interpretations of the roentgenograms are given by courtesy of Dr. H. S. Pettit, Professor of Roentgenology.

each and with a note on the symptoms and duration of occupational exposure.

Case 1

The first case was that of a man, 36 years old, who had apparently worked in the plant for a period of some 17 years. Although it is probable that in the early part of this exposure the atmosphere about his work was heavily laden with dust, at least in the latter part conditions were much improved, and it is believed that his more recent exposure was not particularly hazardous. There was no other suspected dust exposure. No record of the clinical state in this case is available.

The roentgenograms of his chest, taken toward the end of his exposure, were described and interpreted as follows:

Postero-anterior and lateral films of the chest showed a massive confluent consolidation of the upper half of the right lung field, an area of confluent consolidation measuring 8 cm. in diameter in the upper third of the left field, and extensive nodular infiltration of the lower two thirds of the left lung field. The trachea was displaced to the right. No definite area of cavitation was apparent. There was emphysema of the right base. *Summary:* Advanced pneumoconiosis with infection. Extensive confluent consolidation and nodular infiltration.

The description of the portions of the lungs examined by us was as follows:

Gross Observations. The material received consisted of four inadequately fixed portions of lung together weighing 1195 gm. and bearing two large branches of pulmonary vessels containing apparent antemortem thrombi. In one portion of lung there was a roughly wedge-shaped, firm, hemorrhagic area 4 cm. in diameter. The alveolar spaces appeared to be uniformly expanded, producing a vesicular emphysema. There were three areas of whorled fibrous tissue which were of the consistency of heavy tire-tread rubber and presented margins which in some regions were so sharp as to appear encapsulated. These measured up to 7 cm. in diameter and extended from hilum to periphery, where the overlying pleura measured up to 0.5 cm. in thickness. Scattered throughout the remaining pulmonary tissue were many other smaller, but otherwise similar, shot-like fibrous nodules. There were scattered areas with cystic spaces which appeared to be related to inadequate fixation. Hilar nodes were present, heavily pigmented, measuring up to 2 cm. and presenting much the same appearance on the cut surface as the fibrous nodules described, but they were softer.

Microscopic Findings. Nodular areas of whorled collagenous fibrosis contained deposits of brownish black particulate matter. Many alveolar spaces were dilated and their walls were variably attenuated or thickened by a fibrous reaction. In some areas there was fragmentation and clubbing of the alveolar septa and there were pigment-laden macrophages in alveolar spaces as well as within their walls. Some fibrous tissue increase was noted also about blood vessels. One large

vessel contained a recent thrombus and there were hemorrhage and necrosis of an adjacent portion of the lung. The pleura showed a marked fibrous thickening. Lymph nodes showed the same dense fibrous scarring described for the lungs, and heavy pigmentation. When examined under polarized light, innumerable small refractile bodies were seen, yet they were by no means so numerous as the particulate matter visible under ordinary illumination. Lymph nodes presented an entirely similar appearance under polarized light. An additional conspicuous feature was the profound distortion of erythrocytes, practically all of them being of spindle shape (incidental erythrocyte sickling).

The diagnosis recorded from these studies was: *pneumoconiosis (kaolinosis) with pulmonary thrombosis and infarct of the lung.*

Case 2

The second case was that of a man, 35 years old, who had worked in a kaolin processing plant for about 21 years. It is probable that he, too, was exposed to very dusty atmospheres, at least in the early period of his employment. It is reported that 5 years prior to his last illness he had been given a diagnosis of silicosis with suspected concomitant tuberculosis. He had symptoms of dyspnea during his last 3 years, more marked during the final year and extreme for a week or so prior to death. There was associated slight cough productive of small amounts of dark colored sputum, as well as ankle edema at least 3 years before his death. Acid-fast bacilli were reported from the washings of his stomach at that time, but sputum examined shortly before his death was negative for such organisms.

The description and interpretation of his roentgenograms were as follows:

Films of the chest showed a generalized pulmonary emphysema and large confluent areas of consolidation in the upper half of each lung field. The confluent mass in the left hemithorax measured 9 cm. in diameter and its edges were relatively smooth. The two masses in the right upper lung field had a combined diameter of 11 cm. Between them there was an area of cavitation measuring 2.5 cm. in diameter. Below the confluent areas of each lung field there was a fine granular and nodular infiltration. The heart was relatively small. *Summary:* Advanced pneumoconiosis with infection, confluent consolidation, nodular infiltration, cavitation, and emphysema.

The description of the lungs examined by us was as follows:

Gross Observations. A right and left lung, which had been sectioned previously, were received. Within the substance of each lung were hard, nodular, bluish gray areas which might be described as resembling blue marble, and which measured up to 11 cm. in greatest diameter. The right upper and middle lobes were practically replaced by these nodules and the upper half of the right lower lobe was similarly involved. The left lung showed extensive involvement of the upper lobe and of the upper third of the lower lobe. The pulmonary tissue not involved by the large firm nodules showed a pronounced dilatation of the alveolar spaces and a rather diffuse firmness. In the right upper

and middle lobes were cystic spaces with glistening linings, measuring up to 4 cm. in diameter. The pleural surfaces of both lungs were thickened and shaggy, and presented large bullous emphysematous blebs. A major branch of the pulmonary artery in the left lung showed organizing thrombus. Hilar lymph nodes measured up to 2.5 cm., were heavily pigmented, and in part replaced by the same firm, whorled nodularity described in the lungs.

Microscopic Findings. Massive areas of whorled collagenous fibrous tissue were heavily pigmented by deposits of brownish black amorphous material and particulate matter. In some locations alveolar spaces were dilated and some septa showed fibrous thickening while others were attenuated or disrupted. Many spaces were filled with pigment-bearing macrophages and the same sort of material which was widely deposited throughout the fibrous nodules. Cross sections of several branches of the pulmonary artery showed partial or complete occlusion. Pigmented macrophages were scattered through their walls and in the fibrous tissue which occupied the lumina of some. Several of these vessels had crinkled walls suggesting actual compression by the surrounding dense scar tissue. The pleura was markedly thickened and encompassed large bullous spaces. Lymph nodes showed heavy pigmentation and also contained large areas of whorled collagenous fibrous tissue. When examined under polarized light, both the pulmonary tissue and lymph nodes showed a great number of small refractile bodies.

The diagnosis recorded in this case was: *pneumoconiosis (kaolinosis)*.

Summary

While roentgenograms in these two cases apparently do not furnish features upon which definite differentiation between silicosis and kaolinosis may be made, there are two observations of possible significance that may be made from them. The first is that, while the same features may show in cases of silicosis, the massiveness of the involvement of the upper parts of the lungs in kaolinosis is remarkable. Kaolin dust is composed of very fine particles and is a comparatively light dust, possibly having a better opportunity to reach the upper lung areas than heavier dust composed of larger particles. Secondly, although much pulmonary air space is obliterated, emphysema is prominent in the open areas.

The bluish color created by the deposits in the lungs was a distinctive feature. While the reactions causing the development of blue color in clay materials are apparently not clearly understood, it is believed that, because of their adsorptive qualities, clay crystals when mixed

with organic amino compounds may develop a blue reaction under some conditions. Whether this reaction and the consequent development of blue color would occur under all circumstances of deposition of kaolin in the lungs can not be ascertained except from extensive observation, but it should constitute a distinctive feature when it is found, identifying the nature and source of the deposit even if it were not otherwise known.

From the microscopic descriptions it will be noted that while nodular fibrosis with massive whorled collagenous deposits, as in silicosis, composed the more bulky changes, fibrosis of the alveolar walls and thickening and emphysema were conspicuous. This explains the prominence of respiratory difficulties, at least in the one case in which information was available.

Also to be noted especially are the associated vascular lesions. These consisted of extensive obliterative arteritis in which visible particles of foreign material were scattered through the vessel walls, even in the fibrous tissue which occupied the lumina of some arterial branches; and, in the first case, thrombosis of the main branches of the pulmonary artery.

It is regretted that accurate description of the condition of the heart was not available in either case for evidence of the general circulatory effect created by the condition found within the lungs.

Conclusion

Sufficient exposure to inhalation of dust of at least some kaolin deposits will cause in some individuals a chronic fibrous disease of the lungs that may be disabling and even prove to be fatal. The term kaolinosis is therefore established as signifying a definite disease state, beyond merely the location of kaolin in the lungs.

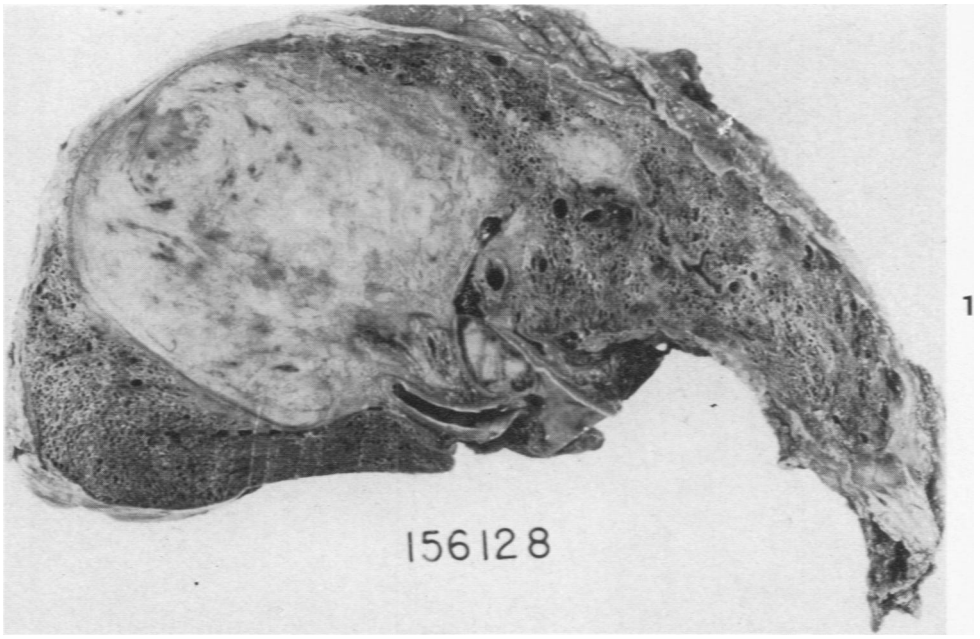
REFERENCES

1. Campbell, A. H., and Gloyne, S. R. A case of pneumokoniosis due to the inhalation of fuller's earth. *J. Path. & Bact.*, 1942, 54, 75–80.
2. McNally, W. D., and Trostler, I. S. Severe pneumoconiosis caused by inhalation of fuller's earth. *J. Indust. Hyg. & Toxicol.*, 1941, 23, 118–126.
3. Tonning, H. D. Pneumoconiosis from fuller's earth; report of a case with autopsy findings. *J. Indust. Hyg.*, 1949, 31, 41–45.

LEGENDS FOR FIGURES

Fig. 1. Sectioned lung, case 1, 156128. Massive nodular pneumoconiosis of upper portion of lung, with diffuse emphysema and pleural thickening.

Fig. 2. Sectioned lung, case 2, 207039. Massive nodular pneumoconiosis of upper portion of lung, large subpleural cavity, diffuse emphysema, and pleural thickening.



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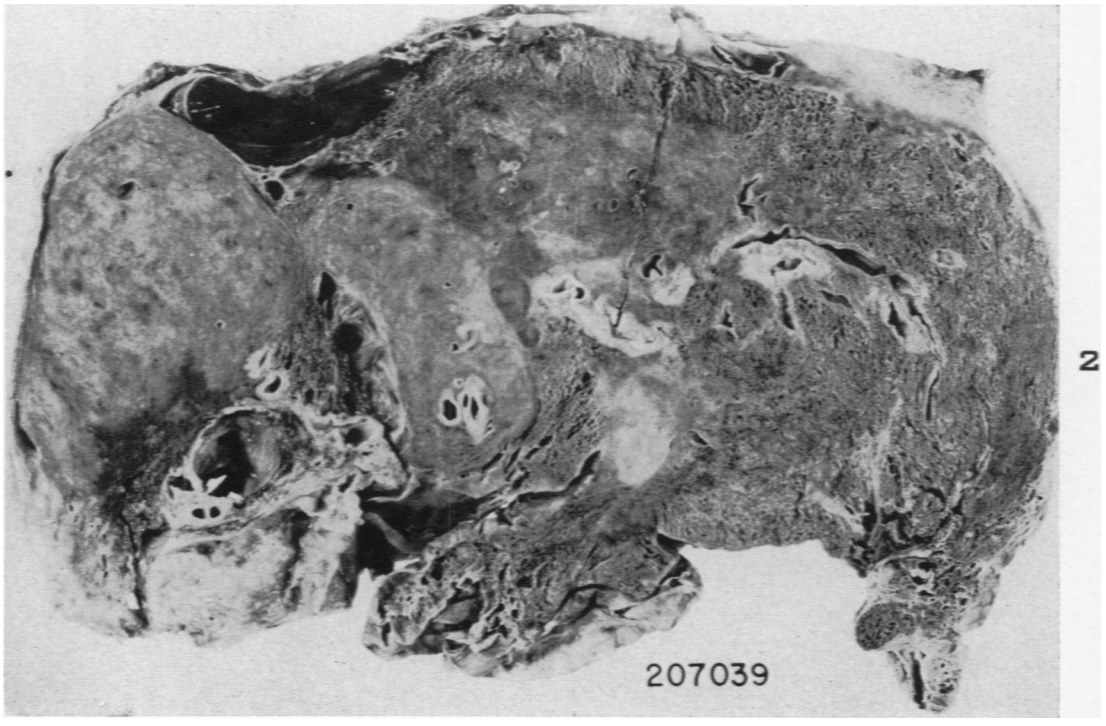

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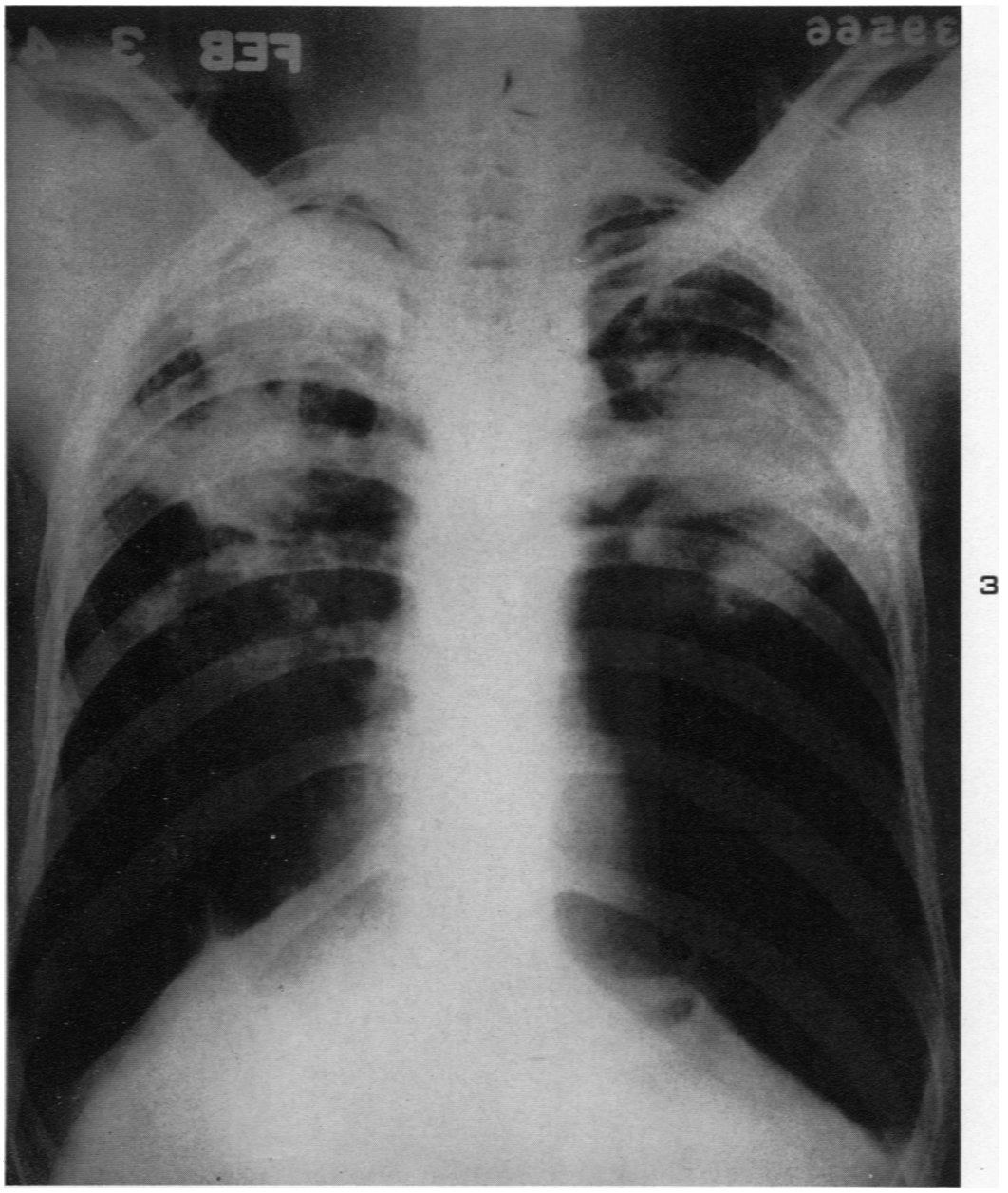
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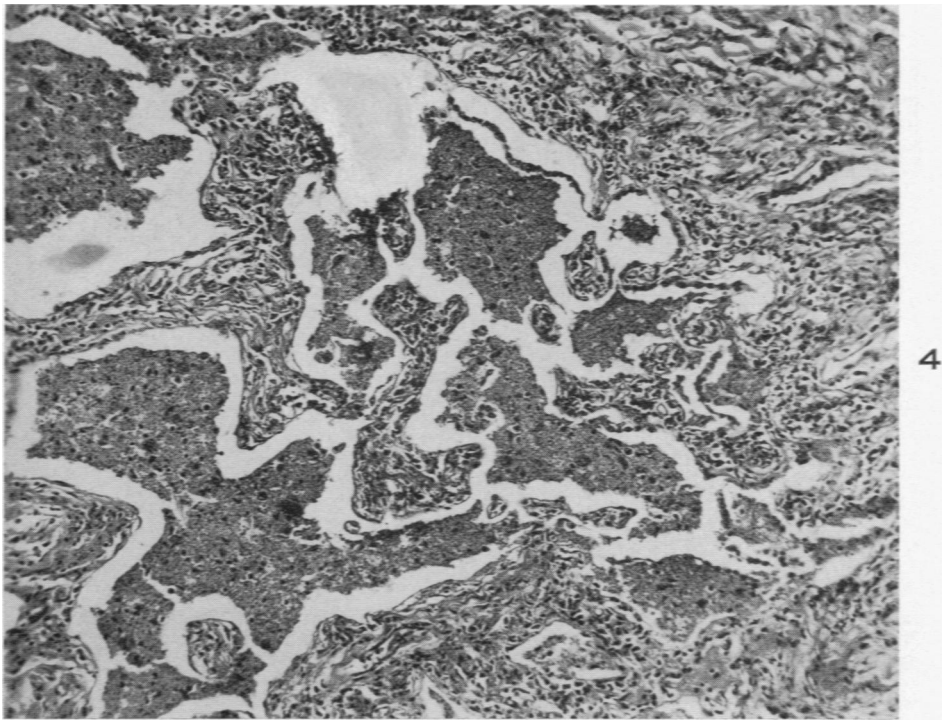


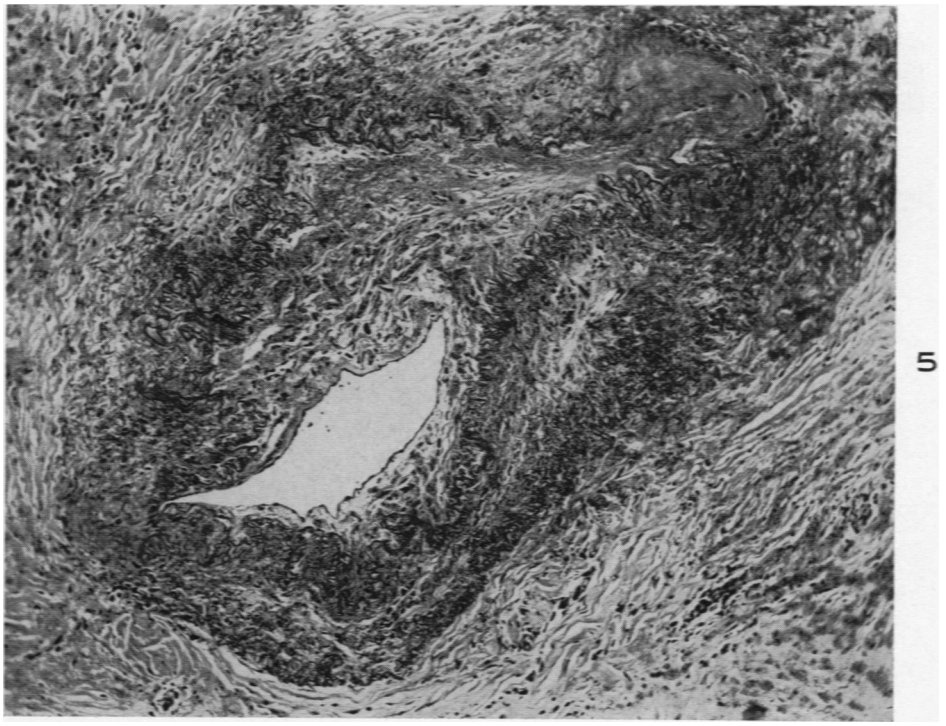
FIG. 3. Chest roentgenogram, case 2. Massive upper confluent pneumoconiosis.

FIG. 4. Case 1. Massive accumulation of dust-laden macrophages and débris in alveoli, with surrounding fibrosis. × 125.

FIG. 5. Severe vascular lesion, with perivascular and intimal fibrosis. × 100.

FIG. 6. Perivascular and obliterating intravascular fibrosis. × 125.

FIG. 7. Dense hyalinizing fibrosis with alveolar obliteration and dust deposit. × 125.



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