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INDUSTRIAL HYGIENE FOUNDATION OF AMERICA, INC.

MELLON INSTITUTE, 4400 FIFTH AVENUE

PITTSBURGH 13, PA.

Report on Case of

FRANK CARLISLE

(IHF Lab No. A61-2)

for

JOHNS-MANVILLE CORPORATION

November 1961

by

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Dr. Paul Gross of the Industrial Hygiene Foundation, Report on the Case of Frank Carlisle for Johns-Manville (1961) Published by AsbestosClaims.Law
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Introduction

The data and material for study pertinent to the case of Frank Carlisle, received by the Industrial Hygiene Foundation laboratory, consists of the following:

1. A varifax copy of the complete medical file at Johns-Manville of Frank Carlisle.
2. A letter of transmittal dated August 4, 1961 and signed by C. U. Culmer, M.D.

Medical History

This 60-year-old negro male, had a number of physical examinations with negative chest findings in each instance. X-rays dating from 1948 to 1961 were reviewed as follows:

5/7/54 - The 1948 and 1951 films are considered to be within normal limits.

There are certain minute changes in the film of 1/18/54 which "are compatible with the very earliest changes which can be detected in early pneumoconiosis, non-specific in character".

9/14/54 - No change. No evidence of TBC.

2/1/55 - No change.

2/23/56 - No change. Classification of PM I seems correct.

3/4/57 - No change. PM I, stable.

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2/25/58 - No change.

1/17/61 - "A new 3.5 cm. fairly dense and somewhat mottled spherical zone in the left upper lobe center and far laterally is suspected of representing active TBC. Not present in previous films. Otherwise unchanged and otherwise essentially negative."

2/20/61 - Unchanged from previous. Active TBC until proved otherwise.

Medical Department Check List

2/1/55 - Possible pre-pneumoconiosis PM II

2/23/56 - No change (maybe should be PM I)
Corrected to PM I or PM II

3/19/56 - PM II

3/4/57 - No change.

Lost Time

Ten days, 2/14/58 - 2/24/58 due to influenza. Note dated October 23, 1954, "Possible early pneumoconiosis. Should be removed from exposure to noxious dust. (signed) C. U. Culmer, M.D."

Frank Carlisle was admitted to Lake County Tuberculosis Sanatorium 2/16/61 and discharged 6/7/61 improved with the recommendation to continue medications (streptomycin and INH).

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A lobectomy, left upper was performed 3/7/61. The tissue report was as follows:

3/7/61 Negro, male, 60 years.

Gross Description

Specimen consists of the upper lobe of the left lung that is a mass measuring 15 x 9 x 4 cm. The pleura is focally roughened and torn and wrinkled. The lung is atelectatic at two levels of the firm nodules that have been previously sectioned. The first is in the apex and measures 3.2 cm. in diameter. The cut surfaces reveal a gray tan material. The hilar structures are examined. They are firmly ligated. The other areas examined are firm and at one level there is a well-defined mass and numerous surrounding small nodules. Numerous representative sections are taken at all levels.

Microscopic Description

Multiple sections throughout the lesions described grossly reveal numerous small and large areas of caseous and fibrous necrosis surrounded by atelectasis of the lung together with fibrous tissue proliferation and lymphocyte infiltration. There are occasional Langhans type giant cells.

In the areas of caseous necrosis are classical asbestos bodies being irregular nodular rod shaped brown staining structures.

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Diagnosis

1. Chronic fibrocaseous tuberculosis of lung, left upper.

2. Asbestosis of lung, left upper.

The diagnosis of tuberculosis can be substantiated as the smears were acid fast positive. These smears were taken from the cut surfaces of the necrotic caseous material at the time of surgery.

A letter to Charles U. Culmer, M.D., dated 3/20/61, and signed by Charles K. Petter, M.D., stated in part as follows: "On March 7 we resected the left upper lobe of Mr. Frank Carlisle. This was done before waiting the outcome of sputum cultures because Oat cells were described in his bronchial secretions."

Work History

Date	Duration	Job	Dust Exposure
6/13/46	1 mo. 2 days	Unclassified	Asbestos & calcite
7/15/46	1 yr. 2 mo 28d.	Unloader	Fiber & silica
10/13/47	5 yr. 9 mo. 7d.	Mixer operator	Asbestos & silica
4/20/53	3 yr. 4 mo. 13d.	Grinder operator	Asbestos & silica
9/3/56	4 yr. 3 mo. 2d.	Car unloader	Fiber & silica
2/11/61	----	Medical leave	

Age at time of first employment at Johns-Manville, 46 years.

Period of potential exposure, 14 years, 10 months, 28 days.

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Report of Findings

Gross Description

Specimen consists of a lobe of lung fixed in formalin 4 x 9 x 12 cm. as well as numerous cut fragments of lung tissue. The specimen shows numerous parallel cuts. The pleura is mostly smooth but shows focal opaque areas. It has a slate blue color mottled with black. The consistency is rubbery but an occasional hard foci are palpable. These are irregular in shape, up to 1 cm. in diameter. The full extent of the densely hard regions cannot be judged because it is precisely in these regions that sections have been cut and removed. Most of the smaller fragments of tissue consist of dense, hard, partially anthracotic tissue containing irregular gray areas of necrosis. In several of them, ragged cavities 3 x 7 mm. in diameter are noted.

Elsewhere, the lung tissue is dark gray, mottled with black showing only occasional patches where air spaces are recognizable. Mostly, the lung tissue appears atelectatic.

Bronchi and vessels are not unduly prominent and show no gross abnormality. No lymph nodes are found.

Microscopic Description

The lung tissue is partially solidified and partially atelectatic. The solidified portions of the lungs consist of nodules, some of them confluent, which measure about 2 to 3 mm. in diameter. Many show caseous

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necrosis. On the periphery of many of these necrotic nodules, young tubercles with Langhans giant cells are present. Most of the nodules are well delimited by circumferentially arranged collagen fibers. Beyond these a more reticular, looser tissue infiltrated by lymphocytes is noted.

In the caseous regions, there is destruction of reticulin so that coarse fragments and finer remains of networks may be found. Occasional asbestos bodies are also found in some of the necrotic areas. In some of the caseous centers only the intraalveolar reticulin fibers have been destroyed so that the outlines of some of the original air spaces stand out in bold relief. Reticulin is also resorbed within newly formed tubercles. Here, fragments of reticulin fibers are found within a number of the giant cells.

There are also non-necrotic, collagenous nodules, similar in size and structure to the caseous ones. These contain silica. Very little or no silica is found within the necrotic nodules or in the surrounding tissues.

There is relatively little air-containing tissue. Here, the alveolar walls are slightly to moderately thickened by an accretive interstitial pneumonitis.

Much of the atelectatic tissue consists of thickened and, frequently, collagenized alveolar walls. Within the collapsed air spaces,

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scattered and clustered asbestos bodies are found. Very little mineral dust is found in this tissue.

The vessels are largely uninvolved but an occasional one is noted which shows moderate to severe mural thickening. The pleura shows focal fibrous thickening. No evidence of neoplasm is found.

Diagnosis

1. Tuberculosilicosis, active.
2. Asbestosis.
3. No evidence of neoplasia.

Comment

The most prominent feature of this diseased lung tissue is the caseous necrosis within nodules and the presence of miliary tubercles on the periphery. These nodules contain little or no silica.

There are, in addition, non-caseous nodules with hyaline centers, which contain a moderate amount of silica.

The question which arises is how many, if any, of the caseous nodules were once silicotic nodules? The presence of hyaline collagenous material near the periphery of some caseous centers suggests a silicotic origin of at least some of the nodules. The absence of significant amounts of silica does not exclude a silicotic origin since the silica may have been mobilized and removed during the inflammation.

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Another unanswered question is how much of the diffuse alveolar wall thickening is caused by asbestosis and how much by tuberculosis? There are, at present, no criteria for differentiating the two inflammatory thickenings. The presence of asbestos bodies does not establish that nearby thickened alveolar walls are asbestotic inasmuch as the tuberculous inflammation is also sufficiently close to have produced the same thickening.

One conclusion is, however, quite definite—there is no suggestion of neoplastic change in the lung tissue submitted.

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Fig. 1 - Area of caseous necrosis with adjacent inflammatory tissue. Several nodular structures containing Langhans-type giant cells are present near the necrotic tissue. The inflammatory tissue consists of a loose, cellular fibrous tissue which is infiltrated by lymphocytes.

Fig. 2 - The reticulin pattern of the same field as in Fig. 1 shows persistence of the alveolar structure only in the region of caseous necrosis. Elsewhere, air spaces have been obliterated by proliferation of fibers which form a fairly dense network. Dissolution of reticulin fibers is noted in the region of the nodular structures (young tubercles). Several giant cells contain fragments of reticulin fibers.

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Fig. 3 - A large area of necrosis bounded by a somewhat dense, partially collagenized fibrous tissue infiltrated by lymphocytes. Several asbestos bodies are present in the necrotic area.

Fig. 4 - The reticulin pattern of the same field as in Fig. 3 shows extensive destruction of reticulin with only delicate remnants of fibrils remaining in the center of the necrosis. The network of fibers is dense near the margin of the necrosis.

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Fig. 5 - An area of necrosis with an outer zone of hyaline collagen still retaining some fibrocytic nuclei. This is delimited by fibrous connective tissue which is moderately cellular. In the connective tissue, several newly formed tubercles are present.

Fig. 6 - The reticulin pattern of the same field as in Fig. 5 shows the necrotic region is largely devoid of reticulin fibers. Only a few fragments remain near the edge. The network of fibers is fairly dense, particularly in the region adjacent to the necrosis. There is resorption of reticulin fibers in the region of the newly formed tubercles.

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Fig. 7 - A dense, irregular mass of hyaline collagenous tissue adjacent to which several Langhans giant cells are present. There is moderate lymphocytic infiltration.

Fig. 8 - Reticulin pattern of the same field as in Fig. 7. There are fairly dense, thick, parallel fibers in the region of the collagen. Elsewhere, the fibers are thinner and form a network. In the region of the giant cells there is resorption of the fibers.

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Fig. 9 - A region of atelectasis where alveolar walls are irregularly thickened, some by collagen. Asbestos bodies are present in the air spaces.

Fig. 10 - Reticulin pattern of the same field as in Fig. 9 shows the predominantly accretive type of alveolar wall thickening. Asbestos bodies are sharply outlined by fine, delicate lines.

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Fig. 11 - A greatly thickened vessel lying in a fairly dense collagenous tissue containing some black pigment, presumably carbon.

Fig. 12 - Reticulin pattern of the same field as in Fig. 11.

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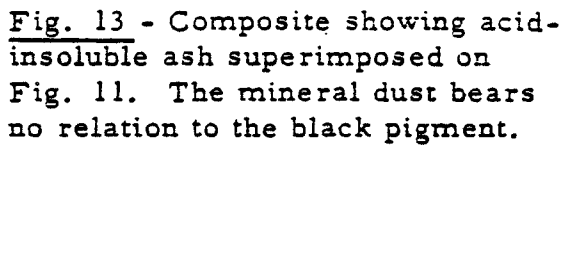


Fig. 13 - Composite showing acid-insoluble ash superimposed on Fig. 11. The mineral dust bears no relation to the black pigment.

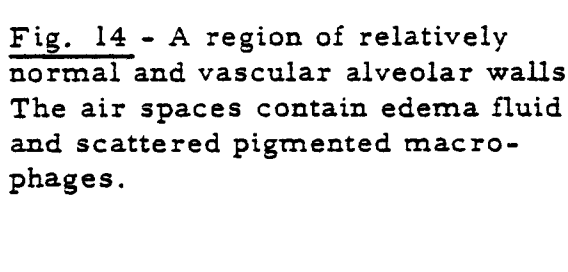


Fig. 14 - A region of relatively normal and vascular alveolar walls. The air spaces contain edema fluid and scattered pigmented macrophages.

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Fig. 15 - The reticulin pattern of the same field as in Fig. 14 shows slight to moderate accretive thickening of alveolar walls.

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