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INDUSTRIAL HYGIENE FOUNDATION OF AMERICA, INC.

MELLON INSTITUTE, 4400 FIFTH AVENUE

PITTSBURGH 13, PA.

Report on

LUNG SECTIONS OF JOSEPH KINCEL

for

JOHNS-MANVILLE CORPORATION

February 1963

by

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INTRODUCTION

Received five slides labeled 43102, Middlesex General Hospital, from Dr. K. W. Smith. This report is based on a study of these five slides.

The sections contain an adenocarcinoma of the lung, apparently a primary tumor. This tumor is associated with a large amount of dense, acellular collagenous tissue containing much extracellular, finely divided black pigment but no asbestos bodies. At the periphery of the tumor, the tissue is heavily infiltrated by plasma cells and lymphocytes. Portions of the tumor show similar infiltration and also leukocytic accumulations.

The lung parenchyma at some distance removed from the tumor tissue also shows severe thickening of septa by edema and heavy infiltration by plasma cells and lymphocytes. Elsewhere, the lung tissue is characterized by profuse multiplication of alveolar cells, many of which have desquamated. The abundant desquamation gives the appearance of the so-called desquamative pneumonia, inclusive of occasional multinucleated giant cells. The alveolar septa here show various degrees of thickening, but most of the thickening is slight. Focally, there is considerable atelectasis in which non-thickened alveolar walls are tightly apposed to one another. Foci of atelectasis alternate with focal complementary hyperdistention. However, the diagnosis of emphysema cannot be made.

In a few areas of atelectasis, where many desquamated cells are found in the air spaces, several collapsed air spaces contain a number of

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asbestos bodies. There are, however, large stretches of tissues; and, indeed, entire sections where no asbestos bodies can be found even though extensive and pronounced alveolar cell proliferation or desquamation, or both are present.

Sections of the main bronchus show focal hyperplasia of the mucosal epithelium but no metaplasia or neoplasia. The mucous glands of the bronchial submucosa are hypertrophied and the stroma is infiltrated by lymphocytes. The peribronchial tissues are extensively fibrotic and heavily infiltrated by plasma cells and lymphocytes. No asbestos bodies are found in the sclerotic inflammatory tissue or beyond. There is focal anthracotic pigmentation of the peribronchial tissues.

Medium-sized arteries exhibit a remarkably severe degree of sclerosis with narrowing of the lumen, even in regions where there is no septal thickening. Foci of smooth muscle hyperplasia are also noted.

The lymph nodes included in the sections show no fibrosis except for one small nodule, the size of a small lymph follicle, composed of acellular collagen which is irregularly arranged. Relatively slight anthracosis is present in the medulla. No asbestos bodies are found.

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ACL Doc 24465 · Page 3 of 5 asbestosclaims.law/library/24465/3

DIAGNOSIS

1. Adeno carcinoma of lung.
2. Chronic pneumonitis, proliferative and exudative.
3. Pulmonary arteriosclerosis, severe.
4. Chronic bronchitis and peribronchitis, severe.
5. Anthracosis, moderate.
6. Past exposure to asbestos dust (asbestos bodies present).

COMMENT

If the chronic pneumonitis demonstrable in these sections is labeled as being asbestotic in etiology, one is confronted with the necessity for explaining why the inflammation is florid even though the last asbestos dust exposure occurred 17 years ago. An asbestotic inflammation of the lungs, removed from further contact with asbestos fibers for 17 years, would certainly be healed and in a "burned-out" stage within this time. This is not the case here.

Of equal importance for the diagnosis of asbestosis is the occupational history which, in this case, indicates a total exposure period of only three months and 16 days—none previous and none since. This exposure period is too short for the production of asbestosis.

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Furthermore, the finding of only a few closely grouped foci of asbestos bodies in the five sections examined, and the existence of large stretches of active chronic inflammation without any asbestos bodies is a feature which is certainly not characteristic of asbestosis. The presence of lymphocytes and plasma cells in the thickened alveolar tissue (not in proximity to the cancer) is another feature which demands an explanation other than one based on irritation from asbestos fibers.

The severe chronic bronchitis present in this case may, perhaps, be etiologically related to the chronic pneumonitis present.

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