INDUSTRIAL HYGIENE FOUNDATION OF AMERICA, INC.

MELLON INSTITUTE 4400 FIFTH AVENUE

PITTSBURGH 13, PA.



Memorandum to: Dr. Kenneth W. Smith, Medical Director
Johns-Manville Corporation
22 East 40th Street
New York 16, New York

Re: Case of Walter Burek
IHF Lab. No. A58-8

From: Paul Gross, M.D., Research Pathologist

Date: January 7, 1959

INTRODUCTION

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

1. A letter dated October 31, 1958, which gave the employment and medical history of Mr. Burek. This letter was signed by D. T. LuBow, M.D.
2. Portions of both lungs fixed in formalin.

SUMMARY OF OCCUPATIONAL HISTORY

Mr. Burek was 68 years old at the time of death and had been retired from Johns-Manville for 3 years. He was first employed by Johns-Manville on November 5, 1946. Prior to that time his employment had been the following:

farming - 12 years

hard coal mines (inside) - 6 years

woolen mills - 6 years

dye works - 6 years

At Johns-Manville, Mr. Burek worked almost 7 years as miscellaneous handler in the Magnesia Department. During this time, he was assigned to cleaning dust bins where he was exposed to Diatomite and Asbestos dust. On October 3, 1949, he was transferred to another department where he worked as sweeper and janitor. This job lasted about 5 years and during this time he was exposed to Asbestos dust. The total period of potential exposure to Diatomite dust was approximately 7 years and to Asbestos dust, approximately 12 years.

SUMMARY OF MEDICAL HISTORY

In June, 1958, Mr. Burek was hospitalized because of pain in the lateral portion of the right chest and the upper right quadrant of the abdomen. Following his discharge, a thoracentesis in early July resulted in clear, yellow fluid. This was negative for acid-fast organisms and tumor cells. Because his condition became progressively worse, Mr. Burek was again hospitalized in October, 1958. Another tap of his right chest again yielded yellow fluid. He was bronchoscoped with negative results and died 4 days later on October 31, 1958. The x-ray diagnosis was pulmonary emphysema with changes consistent with fibrosis and chronic bronchiectasis.

"At autopsy, performed by Dr. Gewanter, there was obvious extensive diffuse pulmonary emphysema with fibrosis predominantly in the lower lobes, markedly thickened and adherent pleura with extensive effusion at the right base. In addition, there was definite thickening of the right ventricle and widening of the pulmonary artery."

REPORT OF FINDINGS

Gross Description:

Right lung weighs 500 gms. and measures 4 x 14.5 x 17 cm. The specimen appears to represent somewhat less than one-half of the lung as judged by its thickness. The pleural surface is greatly thickened and leathery. Its thickness varies from 1 to 3 mm. The interlobar fissures

are obliterated. The upper lobe is composed of crepitant, spongy gray tissue. Beneath the thickened pleura, there is marked enlargement of the air spaces, up to diameters of 5-6 mm. Similarly, enlarged air spaces are found in isolated small foci scattered irregularly over the upper lobe. The rest of the right lung is largely solidified, firm to hard in consistency, and composed of a dense, compact gray tissue, mottled with small black foci. Gray fibrous streaks and thick vessels as well as bronchi are visible upon the cut surface.

Left lung weighs 381 gms. and measures 4 x 9.5 x 16 cm. This specimen also appears to represent less than one-half of the lung. The pleura is irregularly thickened to a maximum of 2 mm. The interlobar fissure is obliterated. Only the upper one-half of the upper lobe is composed of crepitant, spongy tissue. The remainder of the lung is composed of solidified, firm to hard, gray-lightly mottled with black tissue in which gray strands, vessels, and bronchi are prominent. In addition, at the lateral portion of the lower lobe, there is a triangular area, the apex of which is directed upward and begins immediately beneath the pleura. This area extends 9 cm. down to the base to a maximum width of 2.5 cm. and then extends across the base to form a subpleural zone 3 cm. thick. These zones are honey combed with greatly dilated and tortuous bronchial passages which are filled with thick, gelatinous pus. Between the pus-filled cavities, dense connective tissue is present.

No lymph nodes are represented in either specimen. No major vessels or bronchi are present.

Microscopic Description:

Right Lung. Tumor in the form of adenocarcinoma is found in 8 of 10 sections. The tumor is composed of deeply staining columnar epithelium which lines existing air spaces and bronchioles and thereby forms glandular structures. These tumor-lined air spaces form ill-defined masses, some of which occupy about one-fourth of the section in several instances. Small aggregations of such tumor-lined air spaces are also found irregularly scattered throughout the right lung inclusive of the greatly thickened pleura. Tumor tissue is found within the lumen of a number of pulmonary veins and in the lumen of lymphatic vessels.

There is generalized atelectasis and the effectiveness of many of the partially collapsed air spaces is further reduced by masses of asbestos bodies and debris or frank pus filling the lumen.

The alveolar walls are irregularly thickened by relatively acellular collagen. More nodular or fusiform septal fibrous thickening is also present. Such foci tend to be more cellular and contain also fine dust granules. Many of the granules appear to be of translucent, crystalline character and appear greenish.

In some sections, very few alveoli are found. There is much dense fibrous tissue, poor in cells. Among this connective tissue are found

numerous irregular, very long channels of varying caliber. These contain pus and are lined by bronchial-type epithelium. Bronchi are greatly enlarged and filled with pus. In some sections, these pus-filled bronchi and bronchioles alternate with bronchioles and air spaces that are filled with foam cells.

Emphysematous changes are present but are overshadowed by the fibrosis and atelectasis.

In addition to asbestos bodies, which generally occur in sizable clusters, and the greenish crystalline particles, more massive deposits of dense black pigment are present. These are generally perivascular in position. Much of this black pigment is extracellular and there is associated perivascular fibrosis. The blood vessels show considerable sclerosis. The pleura is enormously thickened by a hyaline, acellular connective tissue, which contains compressed tumor glands in some regions. No hyperplasia of alveolar lining cells is noted. There is no gradual transition from normal to tumor lining cells.

Left Lung. Many of the sections show the presence of tumor. This tumor infiltration is more insidious than is the case in the right lung. The tumor masses are much smaller and more widely dispersed.

The fibrosis is extensive and is similar to that on the other side.

There are relatively few air spaces capable of function. Most air spaces are rendered useless by tumor investment, or by an inflammatory

exudates vary. In some places, the air spaces contain pus only. In other places, the exudate is largely fibrinous or fibrino-purulent.

There is extensive bronchiectasis. The lumen of these enlarged bronchi is filled either with frank pus or muco-purulent material.

A number of emphysematous foci are found. These are surprisingly few and small. They are surrounded by thick fibrous tissue.

There is a large nodule with a scalloped periphery which is apparently composed of smaller confluent nodules. It contains irregularly distributed gray dust granules and appears typically silicotic. This nodule is adjacent to a large pulmonary artery and seems to have been originally a lymph node.

One section contains a sharply circumscribed mass of cartilage with some fibrillary stroma. This fibrillary stroma is infiltrated on the periphery by small or narrow tumor glands which are present in moderate numbers elsewhere about the periphery of the cartilaginous mass. This mass of cartilage shows no feature of malignancy and is considered to be haemartoma.

DIAGNOSIS

1. Asbestosis, severe.

    a) Diffuse interstitial pulmonary fibrosis.
    b) Pleural fibrosis, severe.
    c) Emphysema, focal.

2. Adenocarcinoma of

3. Purulent bronchiectasis.

4. Cartilaginous haemartoma of left apex.

5. Mixed pneumoconiosis, slight.

   a) Silicosis, focal.
   b) Anthracosis, slight.

6. Focal bronchopneumonia.

COMMENT

The most important and significant feature in these lungs is the severe reduction in air spaces capable of function. Only in the apical portions of both lungs is spongy lung tissue to be found. The main involvement is due to severe diffuse interstitial fibrosis associated with the presence of many asbestos bodies and asbestos fibers. Contributing also to this impairment is the replacement of normal alveolar lining by carcinomatous cells.

The question arises whether this tumor is primary or secondary. Against the view that this is a primary tumor are the following observations. The tumor occurs diffusely scattered as innumerable foci of microscopic size. No grossly visible tumor nodules are recognizable as primary foci. Microscopically, the foci show no transitions between non-tumor and tumor cells. The tumor cells are smaller and darker staining than the usual alveolar cell carcinoma. It must be admitted, however, that this distinction between primary and secondary tumor is not without an element of error.

LEGENDS OF ILLUSTRATIONS

Fig. 1.- Gross appearance of lungs. The great thickening of the pleura covering the right lung and the obliteration of the interlobar fissures are easily recognized.

Fig. 2.(5600) - Severe emphysema associated with irregular fibrous thickening of septal walls. Some pigment is present. There are many asbestos bodies to be found among the pigment.

Fig. 3.(5609) - Dilated, irregular bronchioles increased in number and filled with pus. Very great fibrous thickening of septal walls with greatly reduced alveolar lumens. Many of these are plugged by brown pigment and masses of asbestos bodies. Some black pigment, much of which is perivascular. Vessels are greatly thickened.

Fig. 4.(5604) - Diffuse alveolar fibrosis with air spaces reduced to mere slits. Many of these slit-like air spaces plugged by debris, pigment, and asbestos bodies. Vessels are greatly thickened.

Fig. 5.(5605) - A more extensive region of acellular, hyaline fibrosis in upper right. In lower left, irregular slit-like spaces are lined by tumor cells. Emphysema in the center. Some anthracotic pigment and many asbestos bodies are present.

Fig. 6. (5601) - Many tortuous bronchioles filled with pus or a mixture of pus and mucus. A small bronchus (below center) shows focal squamous metaplasia in interior portion. There is severe fibrosis throughout and severe vascular sclerosis.

Fig. 7(5602) - There is severe diffuse fibrosis with obliteration of air spaces. Some air spaces contain fibrin and polys. Bronchi contain pus. A bronchiole in center near top shows squamous metaplasia. Vessels are markedly thickene

Fig. 8(5611) - A lymph node near a large branch of the pulmonary artery shows replacement of much of the lymphoid tissue by silicotic hyaline tissue. Some anthracotic pigment is also present.

Fig. 9(5603) - Air spaces are lined by carcinomatous epithelium forming many papillary projections. A large space above center contains many long asbestos bodies in cellular debris.

Fig. 10 (5510) - An isolated cluster of tumor filled air spaces near a large vessel.

Fig. 11 (5605) - Thickened pleura infiltrated by glandular tumor structures. Tumor replacement of lung tissue in lower right corner.

Fig. 12 (5594) - A pulmonary artery containing a tumor thrombus.