FILE NAME: Asbestos Corp Ltd (ACL)

DATE: 1952

DOC#: ACL009

DOCUMENT DESCRIPTION: Medical Report from Thetford Clinic File

ALBERT CHAMPAGNE
Albert Champagne
Thetford Clinic File No. 2335
(Saranac Laboratory No. P-577)

The data and material pertaining to the above named case were received from Paul Cartier, M.D., Director of the Thetford Industrial Clinic, Thetford Mines. The consist of the following:

1. Abstract of occupational history
2. Abstract of medical history
3. Chest roentgenograms
4. Gross tissue specimens of the lung, heart, liver, spleen, and kidneys

Age: 63

Died: November 22, 1952

Occupational History:

Asbestos Corporation Ltd.

Driller - open pit	3 years
Bagger	1 year
Teamster	30 years
Tinsmith Shop	2 years

"Apart from 1 year as a bagger, this employee could not be considered to have worked in dust occupations."

Medical History:

First serious illness began in 1946 at which time the employee took sick leave for six weeks because of a bad cold. In 1949 he noticed transitory pain in the left chest and in both arms specially after effort. This was associated with increasing tiredness. The employee died suddenly on November, 22, 1952 while attending a family party.

Chest Roentgenograms:

"Interpretation of stereographic chest roentgenograms purporting to be those of one ALBERT CHAMPAGNE, bearing identification #2335 and dated Dec. 16, 1946.

ASBESTOS CORPORATION LTD.

The diaphragms are normal in position and contour, but there is slight blunting of the left costo-phrenic angle, and a suggestion of blunting on the right. The cardiac silhouette and great vessels are within normal limits. Trachea is in the midline, and the upper mediastinum is not widened.

R. & L. The lung fields are clear, and there is no enlargement or distortion of the lung roots.

Film of 2/22/51, compared with the above, reveals:

R. & L. There is now definite blunting of the left costo-phrenic angle, which extends a short distance up the lateral aspect of the hemithorax. There is also noted a very fine curvilinear area of increased homogeneous density along the dome of the hemidiaphragm, having the appearance of calcification. There is now seen clouding of the extreme left apex and outer portion of the 1st anterior i.s.

Film of 3/19/53, compared with above, reveals no change.

OPINION: While the findings in this case are not typical of asbestosis, it is quite conceivable that the changes in both costo-phrenic sulci, with associated thickening of the pleura in these regions, especially on the left, could be a very early manifestation of such a condition. However, I believe that the density in the left apex, first noted in 1951, would, because of its location, suggest the presence of a tuberculous lesion.

LEONARD J. BRISTOL, M.D.,
Head, Dept. of Radiology."

Pathology:

Gross Description

Lungs: The left lung is of about normal size but is extensively lacerated by the embalmer's trocar. The pleura is torn but is generally smooth and thin. The secondary lobular septa are somewhat accentuated by the presence of black pigment. The tissue is generally homogeneous in consistency. On section the pulmonary tissue appears generally air-containing and the alveoli appear essentially uniform in size, though perhaps somewhat coarse. The secondary lobular septa are well seen and are somewhat accentuated by black pigmentation but are without evidence of excess fibrous tissue. At the extreme apex of the upper lobe there is a narrow zone of gray scarring immediately beneath the pleura and measuring 1 cm. in greatest depth. Within this area of scarring, there are noted several smoothly lined spaces varying up to 5 mms. in diameter and interpreted as distended emphysematous alveoli.

The right lung resembles the left.

Trachea and Bronchi: The trachea and bronchi are of normal sizes and their walls are of normal thickness. The mucosa is generally intact, smooth and pale.

Tracheobronchial Lymph Nodes: These are of about normal size and consistency. On section the surface is seen to be black in color and moist.

Heart: The heart is distinctly enlarged but the chambers of the two sides seem of normal relative sizes. The epicardium and endocardium are everywhere smooth and thin. The valve rings are of normal relative sizes and their cusps are delicate and freely moveable. The coronary arteries arise normally and follow normal courses, though they are somewhat tortuous. On numerous cross sections all vessels are noted to be partially calcified, especially in their proximal portions. The left anterior descending vessel shows several yellow sclerotic plaques, narrowing the lumen to 1/4 or 1/5 its normal size. In the region of one of these plaques there is grayish-red thrombus material occluding the small remaining lumen. The myocardium is for the most part homogeneous in color and consistency and measures 1.9 cms. in thickness at the base of the left ventricle; 0.8 cm. on the right. Examination of the myocardium in the interventricular septum near the apex reveals numerous fine gray streaks of scar tissue. The myocardium here, however, is of normal thickness.

Aorta: The aorta is somewhat larger in diameter than normal and its elasticity is distinctly diminished. On section there are noted numerous yellow subintimal sclerotic plaques.

Spleen: The spleen is small and its capsule is wrinkled and grey. On section the tissue is seen to be somewhat autolysed but not otherwise remarkable.

Liver: The portion of liver submitted is not remarkable in the gross except for autolysis.

Kidneys: The left kidney is of about normal size. Its capsule strips with ease to reveal a generally smooth surface although there are scattered depressed foci varying up to 5 mms. in diameter. On section the cortex and medulla are present in normal relative amounts, except in the sites of the depressed scars noted externally where the cortex is somewhat thinned. The cortical and medullary markings are normally prominent.

The right kidney resembles the left.

Microscopic Description

Lungs:

The sections exhibit an occasional irregular patch of reticular fibrosis. They occur most prominently as small focal lesions involving widely separated segments of the pleura and contiguous pulmonary parenchyma. At such points, the fibrotic process frequently extends with a short finger-like projections, often along the course of the interlobular septa. Elsewhere, in the deeper portions of the lobs an occasional microscopic patch is found along the course of some of the respiratory bronchioles and vascular trunks. It occurs also as small focal patches which involve the atria. As mentioned, the patches are irregular in that the fibrosis frequently radiates along the alveolar walls. In such instances those walls are thickened and distorted and the corresponding space is also distorted and frequently slightly dilated. Thus, there is often an associated mild focal emphysema. Elsewhere there is an occasional dilated space which apparently bears no relationship to the fibrotic process or to the pigmentation. The presence of such emphysema cannot be accounted for unless it be of the so-called idiopathic type. With respect to the fibrosis, there is no evidence of reaction to crystalline free silica.

The fibrotic patches contain scattered black pigment. In addition, the pigment occurs within macrophages which lie free in many of the alveolar spaces or which are embedded in the alveolar walls. Similar pigment is found also as thin collars in the loose connective tissue about some of the vascular channels. Not all the pigment in those locations is accompanied by an inflammatory reaction. Close scrutiny discloses an occasional spicule of dust. There are also a few golden-brown bodies which are usually globular. Some such bodies are elongated; but, few have matured to produce the segmented, dumb-bell forms.

Elsewhere in the pulmonary parenchyma there are scattered areas in which the alveoli are filled with blood. Usually those areas are poorly sectioned since they exhibit large gaps in the parenchyma. This defect is probably the result of poor fixation and technical difficulty in cutting the preparation for microscopic study. In any event, the blood appears fresh, suggesting that the extravasation was a terminal event. There is no evident infarction and intrapulmonary vascular thrombi are not discovered.

Some of the small bronchi and bronchioles are unusually dilated, suggestive of a mild ectasia. They are generally free of exudate or other material. The epithelial lining is normally thin and smooth. There is no evidence of an appreciable proliferation.

Tracheobronchial Lymph Nodes:

The nodes exhibit scattered black pigment, most concentrated in the medullary portions of the nodes. The pigmented areas show a slight increase in reticular fibers. In general, however, there is only an extremely mild tissue response to the pigment. Asbestos bodies are not discovered in the nodes.

Heart:

The myocardium particularly in sections of the left ventricle and of the septum, reveals focal and linear patches of fibrosis which obliterate and replace appreciable areas of muscle. Elsewhere the nuclei are often enlarged in relation to the respective muscle bundle. This feature suggests the presence of myocardial hypertrophy.

Liver:

There is no remarkable abnormality.

Spleen:

There is nothing significant. Post mortem change is pronounced.

Kidneys:

The cortex shows widely separated linear foci of scarring which are often rich in small round cells. The glomeruli within such foci are hyalinized and the tubules are obliterated. The arterioles reveal an appreciable intimal thickening often with almost complete occlusion of the respective lumen. These changes are interpreted as evidence of a moderate arteriolo-nephrosclerosis. In addition to the above, all sections exhibit post mortem change.

Comment:

The pulmonary changes pertaining to the extent of fibrosis and the degree of emphysema are indeed minimal. That fibrosis occurs in association with deposits of black particulate pigment and an occasional ill formed asbestosis body. However, not all deposits of black pigment are accompanied by fibrosis. This feature suggests that at least some of the dust had a low biological activity. The other possibility is that that dust was deposited at a relatively recent date and therefore did not have time to exert its adverse influence before death of the patient.

The focal pulmonary hemorrhage is interpreted as an antimortem event. It probably occurred terminally as it is related to his cardio-vascular disease. That disease was apparently a systemic one. Although it provoked a cardiac

failure resulting from involvement of the coronary vessels, nevertheless it appears that the disease was basically a hypertension which was reflected also in a moderately advanced arteriolo-nephrosclerosis. There is no evidence that hypertension and its consequent cardio-vascular-renal disease have a relationship to the inhalation of asbestos dust, or to its pulmonary deposition and resultant asbestotic fibrosis such as present in the lung of the case under review.

Opinion:

It is my opinion that Albert Champagne died of primary cardio-vascular renal disease with consequent sclerosis of the coronary vessels and fibrosis of the myocardium and with moderately advanced nephrosclerosis, which have no specific relationship to his occupation. His lungs exhibit only a very mild asbestotic fibrosis which is discovered pathologically but which was not sufficiently defined for detection roentgenographically during life.

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