November 6, 1956

Charles F. Shook, M.D.
Medical Director
Owens-Illinois Glass Company
Toledo, Ohio

Dear Doctor Shook: Re: Lee Bryston

My apologies for the delayed transmission of my report on this most interesting case. I had hoped to receive some x-rays to review and also intended appending a few microphotographs. As our photographer left us earlier this year I have had to train another technician to undertake this task with consequent delays. When I have some good photographs I shall send them to you.

I hope the mystery of this man's exposure can be cleared up.

With kind regards,

Yours sincerely,

G. W. H. Schepers, M.D., D.Sc.
Director

GWHS:mem
Enclosures (6)

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OCD 68

MEDICO-LEGAL OPINION

in
respect
of

OCCUPATIONAL CHEST DISEASE

by

G. W. H. Schepers, M.D., D.Sc., F.C.C.P.
Director
Saranac Laboratory
7 Church Street
Saranac Lake, N.Y.

November 5, 1956

Subject: Lee Bryston - Aged 57
Deceased - July 8, 1956
Employee - Libbey Glass Division, Owens-Illinois Glass Co.
Saranac Laboratory No. P-56-682

Referred by: Charles F. Shook, M.D.
Medical Director
Owens-Illinois Glass Company
Toledo, Ohio

Tissues Furnished by: K.R. Howard, M.D., Plant Physician
Owens-Illinois Glass Co., Libbey Glass Division
Toledo, Ohio

I. GROSS PATHOLOGY

The specimen examined consists of the left lung and portions of the right lung, fixed in formalin and received wrapped in cloth and sealed in saran wrap. In the case of the left lung the pulmonary vessels and bronchi had been cut flush with the mediastinal surface.

The pleural surfaces are smooth with small scattered black subpleural isolated foci of pigmentation. In rare foci there is a central whitish portion suggestive of scarring. None of these pleural foci are palpable.

The lung hilus reveals the presence of a firm mass of enlarged glands interposed between secondary branches of the left pulmonary artery. On section this glandular mass is seen to be composed of a conglomeration of several confluent, component nodes which are densely adherent to one another and to surrounding blood vessels and bronchi. The texture of the mass is friable with several concretions of stony consistency embedded in it. Minute foci of apparent caseation are also present.

The main bronchi appear normal but the main branches of the pulmonary artery are somewhat distended and isolated atheromatous plaques are discernible on the exposed surfaces.

In the region of the posterior edge of the upper lobe there is a large, firm, inverted pyriform mass which had been cut into before the specimen was received at the Saranac Laboratory. This mass, which measures approximately 4 cm in its greatest or vertical diameter, and about 3 cm in width at its base, has a grey appearance, a friable center, no definite capsule, and has blood vessels and bronchi leading into its apex. There are light pleural adhesions over the free surface of this mass but no pleural thickening.

The cut surface of the lung reveals a universally distributed vesicular emphysema and sparse, small foci of pigmentation. Isolated such foci are palpable, especially where they lie close to the mass previously described. Pigmented foci in other areas are not palpable. The largest of the palpable foci measures about 3 mm in diameter.

On section the basal lobe also reveals diffuse vesicular emphysema with some bulla formation toward the apex. A few scattered semiconsolidated nodules are visible and palpable, some measuring up to 4 mm in diameter. At the base of the lobe there appears to be some degree of hypostatic congestion.

The general appearances of the right lobe are similar to those of the left side - emphysema, hypostatic congestion, foci of pigmentation, and isolated, sparse, palpable nodules being present. In addition, there is a mass measuring about 2 cm by 1 cm along the posterior edge of this lobe. It is greyish in appearance and has a friable center.

The bronchi in the lung substance do not appear to be diseased. The smaller blood vessels are relatively inconspicuous and several of the medium-sized vessels are occluded apparently on account of ante mortem thrombosis.

II. HISTOPATHOLOGY

On microscopical examination the large pyriform mass found in the left upper lobe proves to be an atelectatic condensation of a large part of this lobe with superimposed conglomerate silicosis. The mass contains the degenerated remnants of bronchi and large blood vessels and is practically airless, very few small islets of alveoli surviving among the silicotic masses. In this zone the silicotic components comprise three elements. The first or main feature consists of large concentrically lamellated; hyalinized nodules in which there is no evidence of necrosis of the kind associated with tuberculo-silicosis. The secondary element is composed of masses of koniophores, plasma cells and fibrocytes with a light supporting network of collagen bundles and reticulum. These cellular zones tend to be arranged around the periphery of the hyaline nodules and are the main factor in binding them together. Among these cells there are degenerated and semi-degenerated blood vessels and bronchi. These structures constitute the third component of the mass. The bronchi have retained a lumen with an epithelium in some areas. In other parts the respiratory passages can only be identified by the survival of degenerated, variably ossified and calcified cartilagenous masses arranged in roughly circular patterns. In those areas where these bronchi still are patent, the mucosa is degenerated with hyperplasia of the muscularis and marked hypertrophy of the bronchial arteries.

The solidified areas found at other sites in the lung are comparable in a histological sense to the above described mass. They also contain conglomerate hyalinized silicotic nodules, areas in which dust-laden macrophages and fibrocytes predominate and calcified remnants of degenerated bronchi with partly occluded blood vessels.

The lesser nodules found at various sites in both lungs are essentially hyalinized multilamellated collagen bodies of the silicotic kind with surrounding masses of koniophores and fibrocytes. Several of these nodules lie adjacent to small bronchi and a few surround small blood vessels.

The alveolar walls in most parts of the lung show a moderate to marked degree of thickening. In some areas this is due primarily to cellular infiltration. In other regions there is dense collagen deposition. In relation to both these features there is marked reticulum formation. Elastic tissue is conspicuous by its absence. In some areas the thickening of the alveolar walls is partly due to hyperplasia of smooth muscle. Where cellular or fibrous infiltration has not occurred there is moderate distension of alveolar wall capillaries which feature emphasizes the loss of mural capillaries over extensive areas of the pulmonary parenchyma.

These alveolar walls are stretched thin in many areas and are broken down at numerous other sites. This is due to the presence throughout the lung parenchyma of marked hypertrophic emphysema. Some of the distended air spaces represent dilated alveolar ducts and terminal bronchioles.

The pulmonary arteries show hypertrophy of their muscular coats in most parts of the lung. In the hilar region there is additional focal intimal atheromatosis. In some regions the lesser arteries are markedly stenosed through fibrous hyperplasia around them.

II. HISTOPATHOLOGY (cont'd.)

In several of the pulmonary blood vessels thrombosis had occurred. As these clots are only partly organized it is evident that the thrombosis was part of the terminal process and not an essential feature of the main disease process. Small areas of infarction were also present.

The bronchial epithelium reveals predominance of goblet cells. The muscularis mucosae is markedly hypertrophied in most areas but there is no measure of cellular infiltration to the mucosa. The bronchial glands are moderately hypertrophied.

At the pulmonary hilus the bronchi and blood vessels are bonded together by fibrocellular tissue which surrounds conglomerate partly degenerated silicotic nodules. No evidence of tuberculosis could be found in these sections despite the suggestion of caseation found on macroscopic examination. The gritty elements in these lymph glands represent areas of calcification.

III. HISTOCHEMISTRY

(Thomas M. Durkan)

Portions of the left upper lobe, of the right lobe, and of a mass in the apex of the lung were submitted for analysis. The weight of the tissue samples was as follows:

	Left Upper Lobe	Right Lobe	Apical Mass
Weight of tissue (moist)	32.5 gm	89.3 gm	7.6 gm
" " " (dried at 100-105° C)	7.4	19.9	2.5

Each tissue sample after being dried was pulverized, mixed, and divided into two parts. One part was ashed in a muffle furnace and the total silica content of the ash determined by standard methods. The other part was digested with 30 per cent hydrogen peroxide to destroy organic matter and the insoluble material surviving that treatment was subjected to petrographic and x-ray diffraction examination.

Tabulated below are the findings of the analyses and, for comparison, the results of similar analyses for (1) a normal group of individuals not exposed in their occupation to dust, (2) a group exposed to siliceous dust but who did not develop silicosis, and (3) a group of silicotics. For the normal, non-silicotic, and silicotic groups the average value is given and, in parentheses, the range of values is shown.

III. HISTOCHEMISTRY (cont'd.)

Ash Content and Silica Content of Lung Tissue

Lee Bryston (P-56-682)		Normal Group	Non-silicotic Group	Silicotic Group
Ash content, per cent of dried tissue				
3.27%	Left lobe	3.0% (1.8-6.5)	-	7.4% (3.1-34.8)
3.28	Right lobe			
16.65	Apical mass			
Total silica content, per cent of ash				
5.11%	Left lobe	3.4% (0.4-8.6)	12.7% (1.6-40.0)	19.9% (0.3-62.8)
3.16	Right lobe			
4.91	Apical mass			
Free silica content, per cent of ash				
2.9 %	Left lobe	1.2% (0.1-2.6)	3.3% (0.1-16.6)	9.3% (0.2-33.2)
1.3	Right lobe			
2.9	Apical mass			
Free silica content, per cent of dried tissue				
0.09%	Left lobe	0.03% (0.00-0.16)	0.29% (0.00-1.68)	1.11% (0.02-5.78)
0.04	Right lobe			
0.48	Apical mass			

IV. DISCUSSION

The histological lesions found in this case can be identified as silicotic in type because of the presence of the characteristic whorled nodules and the histochemical demonstration of the presence of excessive quantities of free silica. It is, however, an unusual variety of silicosis insofar as diffuse alveolar mural sclerosis is combined with massive conglomerate lesions, discrete nodulation being sparse only and irregularly distributed. The morphological resemblances are with the type of silicosis encountered in the flux-calcined diatomaceous earth industry and with Shaver's disease.

The mysterious element in this case is the lack of an adequate history of exposure to free crystalline silica. From the analytical findings it appears,

IV. DISCUSSION (cont'd.)

however, that this individual did at some time have a slight to moderate exposure to siliceous dusts. Since the greater part of the silica recovered from the tissue was present as cristobalite, tridymite, or mullite, all of which are high-temperature products, it is probable that his exposure to silica occurred in a plant making ceramic products or during the course of certain operations, such as re-lining kilns, which liberate dust containing the high-temperature forms of free silica. The presence in the lung tissue of a substantial amount of tridymite, a form of free silica which is relatively uncommon but which is present in old bricks from kilns, is worthy of note. Most of the total silica content of the tissue specimens was accounted for by the cristobalite, tridymite and mullite components. It is unlikely, therefore, that this individual had any significant exposure to other siliceous dusts.

These facts narrow the etiology of this disease down to a specific variety of exposure which it should be possible to trace. It is stated that the duties of the deceased included one year as a laborer or cleanup man, during which time he spent a few hours off and on in the batch department. His main employment during his final 20 years of service was, however, as a guard and watchman. It is generally assumed that such guard duties are relatively free of occupational risks. To reconcile the presence of such an advanced degree of silicosis in this man with such a job status one must postulate the possibility that there may be risks attendant on the function of nocturnal guard or watchman which have not previously been suspected. This matter deserves some enquiry. It is conceivable, for instance, that when all the doors or windows of a ceramic plant are shut overnight, nocuous atmospheric substances may be concentrated in the building. Some night watchmen are also assigned duties such as dusting offices or work benches, sweeping floors or tending furnaces or other apparatus that cannot be turned off overnight. It should be possible to ascertain whether such factors were present in this case.

There remains a possibility that the deceased may have carried on a hobby or private backyard or basement industry in which he became exposed to siliceous dusts.

Some emotionally or mentally disturbed individuals have also been known to deliberately expose themselves to toxic substances.

There remains the possibility of an undue degree of susceptibility in this individual if it can be established that disease of the present type has not been previously noted in other employees. On first inspection of the apical mass the presence of bone, cartilage, muscle and bronchi and blood vessels suggested that the lesion was essentially a hamartoma. However, the widespread pulmonary fibrosis and the occurrence of similar lesions at other sites are rather against this possibility, while the demonstration of the combination of four varieties of free silica (tridymite, cristobalite, quartz, and mullite)

IV. DISCUSSION (cont'd.)

confirms the fact that the disease is dominantly of industrial origin. This does not yet dispose of the possibility that the deceased may have been one of those unusual individuals who retain inhaled foreign matter in unusual quantities and react excessively to them. The marked involvement of hilar root glands further suggests the possibility that early exaggerated reaction in these structures may have obstructed the egress of trapped siliceous dust both through blockage of lymphatics and stenosis of bronchi. Such an explanation accounts for the development of massive lesions in atelectatic lung lobes in rare cases of silicosis. There may also have been an infective factor which involved these hilar lymph glands at an early stage. No trace of such infection persisted.

V. DIAGNOSIS

The disease process found in the lungs of the deceased is an aberrant form of silicosis characterized by diffuse mural sclerosis with associated hypertrophic emphysema and focal conglomerate nodular fibrosis with associated regional pulmonary atelectasis, the etiological agents being tridymite, cristobalite, quartz, and mullite.

GWHS:mem

Nov 5, 1956

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