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INDUSTRIAL HYGIENE FOUNDATION OF AMERICA, INC.

4400 FIFTH AVENUE PITTSBURGH, PA. 15213

Report on

EDGAR A. GERVAIS
A67-1

by

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for

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Report on

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This report is based on sections prepared from eight blocks of lung tissue received in formalin from Dr. Benjamin Castleman, Massachusetts General Hospital, and on five sections of lung tissue from the same source.

Sections cut at the Industrial Hygiene Foundation Laboratory

Two of the eight lung sections appeared to be solid tissue. The other sections were spongy and were characterized by the presence of excessively large spaces 3 to 5 mm in diameter.

The solid tissue owed its appearance to a combination of atelectasis and fibrosis. Air spaces were represented by irregular, slit-like spaces some of which were partially lined by cuboidal epithelium, whereas others were lined by stratified squamous epithelium. In places, the latter was atypical in that the polarity of some nuclei was irregular and some nuclei were excessive by large and hyperchromatic. Many of these slit-like spaces also contained macrophages.

The stroma was moderately dense and collagenous and was infiltrated in an irregular manner by monocytes, plasma cells, and lymphocytes. This cellular infiltration was most profuse in the collagenized, thickened subpleural region which was also characterized by the presence of large, congested,

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thin-walled vessels that had the appearance of enlarged venules. In this region, scattered deposits of anthracotic pigment were present as well as of golden-brown pigment resembling hemosiderin. Much of the latter lay within macrophages.

Similar, though less profuse cellular infiltration was found in some of the thickened atelectatic septa. The hemosiderin-filled(?) macrophages were found in larger numbers here than in the subpleural regions.

In one of the six sections, some of the enlarged, cystic-appearing air spaces were nonetheless adequately partitioned by alveoli. The alveoli contained numerous alveolar macrophages. In a few air spaces, a small amount of fibrin and an occasional sprinkling of leucocytes were seen. The septal walls in this section were generally thin although a few were found thickened by fine connective tissue fibrils. In many air spaces the alveolar membrane was lifted, as a sheet, off the septal wall—a picture similar to that seen in cadmium fume pneumonitis. Elsewhere, the alveolar surface had a beaded appearance, caused by an excessively large number of prominent alveolar cells on the surface. An interlobular septum was greatly widened by severe edema that separated its stroma into discrete, very fine fibrils. This edema commonly extended along the adventia of vessels. The subpleural stroma was similarly involved.

The other sections of lung tissue showed somewhat more septal(alveolar)fibrosis but this was not uniform and many septa were normal. The fibrous thickening differed materially from that seen in the subpleural atelectatic regions in

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that the stroma here was composed of widely separated, delicate fibrils and was often associated with clustered, bloated macrophages filled with hemosiderin-like pigment. In some of the larger septa, infiltration by monocytes, plasma cells and lymphocytes was often found. Small clumps of black pigment were also scattered in this tissue. Some of the edematous stroma contained dilated and congested, thin-walled vessels. Many of the elastic fibers were unduly prominent, apparently having imbibed either hematoxylin or a brown color.

Prolonged search of hematoxylin-eosin stained sections revealed only one ferruginous body. This one was shaped like a dagger without a hilt, having a pointed end. The other end was fractured. There was no suggestion of a central core, of segmentation, or of clubbing.

The air spaces contained many macrophages. Most of them were of moderate size and well preserved. Some, however, were bloated and filled with hemosiderin-like pigment. A number of multi-nucleated giant cells were also present. In one region, the air spaces were filled with an acidophilic, mucin-like material.

One of the most striking features of most of the sections was the presence of large cysts devoid of partitions. Some of these cysts were partially lined by cuboidal epithelium; some, by stratified squamous epithelium; but most of them were not lined by cells. Many were obviously greatly dilated bronchioles with identifiable muscle fibers in the wall. The walls of these structures showed focal infiltrations by monocytes, plasma cells, and lymphocytes to varying degrees. Congestion of vessels and edema of the wall were also present.

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Another significant aspect of these structures was that between some of these cysts alveolated tissue was absent and edematous fibrillar stroma with the cellular infiltration seen elsewhere was substituted.

Some, but not all, of the larger bronchi also showed similar cellular infiltration and edema of the wall with vascular congestion. Focal squamous metaplasia was also frequently present.

Many of the veins showed considerable edema of the adventitia but no significant vascular sclerosis and no occlusions were observed.

The pleural surface over most of the sections was smooth and intact. There was, in most regions, a considerable amount of subpleural fibrosis. In a few sections, the surface was disrupted. The character of the subjacent tissue suggested that adhesions, if any, were of a filmy, fibrous type.

Sections stained for iron and with a weak fuchsin counterstain revealed a remarkable absence of the tell-tale blue color characteristic of iron. Any asbestos body would have been easily visible as an elongated dark blue structure against a light pink background, even if "degenerated." The hemosiderin-like material found in macrophages gave a negative test for iron and therefore was not hemosiderin. A control section of lung from a guinea pig that was stained for iron with the other sections was positive.

Extremely little ash was seen in the sections that had been incinerated and treated with concentrated hydrochloric acid. No fibers were found. The ash that was found could be related to the anthracotic material which was present.

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Sections from Massachusetts General Hospital

One of these sections showed the presence of an undifferentiated carcinoma in relation to a large bronchus.

The other sections were essentially similar to those described above.

No ferruginous bodies were found.

Diagnosis (based on the sections examined)

1. Undifferentiated carcinoma of bronchus
2. Honey combing of lungs (bronchiolectasis)
3. Chronic pneumonitis, non-specific, of unknown etiology
4. Chronic bronchitis and bronchiolitis
5. Subpleural fibrosis

Comments

The most important question in this case is whether or not the lung cancer is an asbestotic one. If asbestosis is present, then it would be difficult to exclude the asbestos mineral dust as a causative factor even though the man may have been a heavy cigarette smoker and even though heavy consumption of cigarettes is recognized to be a much more common cause of lung cancer than is exposure to asbestos dust.

The question to be resolved, therefore, is whether or not the chronic pneumonitis (pulmonary fibrosis) present in this case has features characteristic of asbestosis.

But first it should be stated that there is nothing about asbestotic fibrosis per se of the lung parenchyma that is specific for this disease (except for the presence of asbestos fibers in the inflammatory tissue and these may be very difficult to see). So far as is known at present, there is only the association of the fibrosis with ferruginous bodies—asbestos bodies—that allows a pulmonary fibrosis to be designated as asbestotic with reasonable certainty.

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Despite a diligent search, even with the aid of specially stained slides only one ferruginous body was found. It, however, was so atypical that it could not be classified as an asbestos body. Thus, there is no basis for labelling this fibrosis as asbestotic other than that the history indicates that this man was employed at a plant that fabricated asbestos.

There are a number of reasons why the pulmonary fibrosis of this case should not be classified as an asbestotic inflammation:

1. The chronic inflammation of alveolar walls seen in this case is intimately associated with infiltration by inflammatory cells (monocytes, plasma cells, and lymphocytes) that either are not seen or are very sparse in asbestosis except when associated with secondary infection.

2. There seems to be some relationship of the edematous fibrous septal thickening with the presence of clustered macrophages bloated with hemosiderin-like pigment—a feature not seen in asbestosis.

3. It is conceivable that in "burned-out" (healed) asbestosis, asbestos bodies may be scanty and difficult to find. However, because of the fibrillar and often cellular character of the septal inflammation, the latter can by no means be considered healed or "burned-out." In lungs with an active asbestotic inflammation asbestos bodies are commonly plentiful. The failure to find such bodies in these lungs with an active, productive inflammation speaks against an asbestotic origin.

But even if one ferruginous body suggestive of an asbestos body had been found by chance, the question of its significance could have been raised

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inasmuch as in a recent investigation of 100 randomly selected lungs in Pittsburgh, one or more ferruginous bodies indistinguishable from asbestos bodies were found in 97% of the cases.

4. The edematous, fibrillary character of the inflammatory connective tissue in the present case is most unusual, to say the least, for an asbestotic inflammation.

5. On the surface, the patient, an employee of an asbestos fabricating plant, was exposed to asbestos dust. Actually, he was an Industrial Relations Manager; so that his exposure to asbestos dust was casual and minimal.

6. Workers in an asbestos fabricating plant are exposed not only to asbestos but also to cement, silicates, and silica. It is quite common to find in the lung sections of these workers a fair amount of dust with a preponderance of silicates (other than asbestos) and silica. The amount of mineral dust in the sections could serve as a rough measure of dust exposure. In view of the exceedingly scanty amount of siliceous dust in the lung sections of Mr. Gervais, it is concluded with reasonable certainty that his exposure to dust had been very light indeed.

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