A Case of Pulmonary Silicosis Accompanied by Increase of Muscle Fibers in the Lung

MANABU TAKAHASHI and TŌICHI KIRIHARA

Department of Pathology (Prof. Kazuo Inouye)
Yamaguchi Medical School, Ube,
(Received July 20, 1960)

As aptly reviewed by Liebow and his colleagues[2], numerous reports have been published on hyperplasia and hypertrophy of pulmonary musculature in chronic pulmonary diseases. It seems worthwhile to make further observations of such materials for better understanding of the pathology. However, little attention has been paid to the muscle elements of the lung in the studies of pulmonary silicosis which is a notable chronic affection of the lung. The present report describes an autopsy case of pulmonary silicosis in which marked increase of muscle fibers in the lung was found.

REPORT OF A CASE

Clinical history: The subject was a man of 50 years of age who had worked as a coal miner for about 25 years. In October 1949, at his age of 40, he was suspected to have silico-tuberculosis by the x-ray examination. The x-ray finding of the chest was not improved by the administration of streptomycin and isonicotinic acid hydrazide. In the following few years, he occasionally noticed edema in the legs. He had malaise, slight fever, and productive cough. He felt palpitation and dyspnea after a slightest exertion. In March 1955, he was admitted to the Yamaguchi Medical School Hospital. The chest x-ray taken on admission exhibited confluent nodular lesions in the upper two thirds of both lungs, a marked thickening of the pleura, and numerous subpleural blebs. He was diagnosed as having pulmonary silicosis without complicating tuberculosis, and was treated with cortisone and ACTH. Grafting of cold-stored skin was also attempted for treatment. All these measures showed little therapeutic effect and his vital capacity of 1800ml on admission was reduced to 1010 ml by the end of the year. Circulatory disturbance became gradually aggravated, and he died on January 6, 1960.

GROSS EXAMINATION

The body was that of a male person of rather large stature. There were no gross abnormalities except a circular cicatrization on the left arm at the site of skin graft

and numerous small areas of black pigmentation on the hands. The fingers and toes showed no clubbing.

Thorax: Because of heavy pleural adhesions, removal of the lungs was extremely difficult, necessitating excisions on the medial and posterior sides of the lungs. The bases of the lungs were free with no accumulation of fluid.

Left lung: The pulmonary tissue was generally dark and felt hard. A large mass of about 5 cm in diameter was palpable in the posterior side of the upper lobe. This lesion was firm and gritty, and was cut with distinct resistance, exposing the surface of dark mottled color a part of which resembled gray marble in appearance. The surrrounding pulmonary tissue was geneally emphysematous. Large emphysematous blebs were observed under the pleura of the upper lobe and occasionally of the lower lobe. There was no cavity or no area of caseation.

Right lung: The right lung was very much like the left, except that the solidified masses in the pulmonary tissue were smaller and more numerous than in the left.

Peribronchial lymph nodes: They were large and dense. The cut surface showed dark gray mottled color.

Heart: The heart was enlarged twice a normal, mainly due to dilatation of the right ventricle. The right ventricular wall was hypertrophied to some extent. All the valves were competent.

Spleen: The spleen was atrophic, weighing only about 50 gm. The capsule was thickened and ragged. On cut surface, the trabeculae were prominent and the follicles indistinct.

Liver: The liver was flat and had several deep grooves running antero-posteriorly on the upper surface, but otherwise appeared normal.

Other organs were grossly unremarkable.

MICROSCOPIC EXAMINATION

Lung: Corresponding to the location of the solidified masses, there were large areas of well vascularized connective tissue which was infiltrated with plasma cells and dust-laden macrophages. Within these granulomatous areas, there were observed solitary or confluent nodules which were composed of dense collagen fibers arrannged in a laminated fashion. A black pigment was deposited between these fibers. The outline of the nodules was clearly defined but the central parts sometimes appeared hyaline or finely granular (Fig. 1). Calcification of the nodules was rarely encountered. Multinuclear giant cells and epitheloid cells could not be found.

Small blood vessels were frequently grouped around the nodules. The arteriolar wall, epsecially the media, was greatly thickened and the lumen was either narrowed or obliterated. It was noted that most of the muscle fibers in the hypertrophic media was longitudinally arranged. The muscle fibers of the media were replaced to some extent by collagen fibers. The internal and external elastic membranes of the

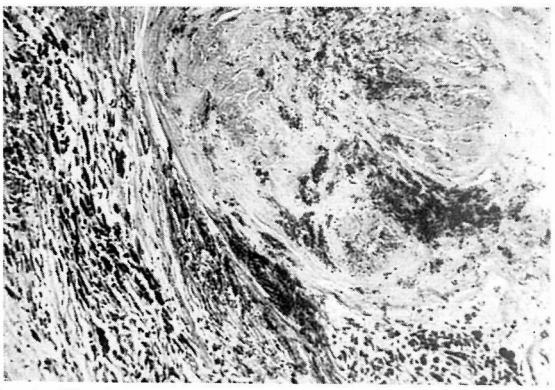

Fig. 1 A silicotic nodule in the lung (Hematoxylin and eosin, × 100)

arterioles were well preserved and showed no disruption. Even in the obliterated arterioles, they could be seen on both sides of the muscle fibers, suggesting localization of the latter in the vascular media. Perivascular connective tissue showed marked proliferation, while the intima was little affected. Thrombosis was not found.

Independent of the muscle fibers described above which were associated with arterioles, there were observed free muscular bundles running parallel in the granulation tissue with no accompanying vascular elements such as intima and adventitia (Fig. 2). In general, muscle tissue could be identified beyond doubt in the hematoxylin and eosin preparations; the muscle cells being more plump and more intensely acidophilic than the fibroblasts, the nuclei larger and ovoid or rod-like with rounded ends rather than spindle shaped, in contrast with the nuclei of fibroblasts. Muscular element around the bronchioles showed no proliferation.

The pulmonary tissue at the periphery of the granulomatous areas showed variable degrees of desquamative pneumonia with the presence of dust-laden macrophages and occasional accumulation of edema fluid in the alveolar spaces. The other part

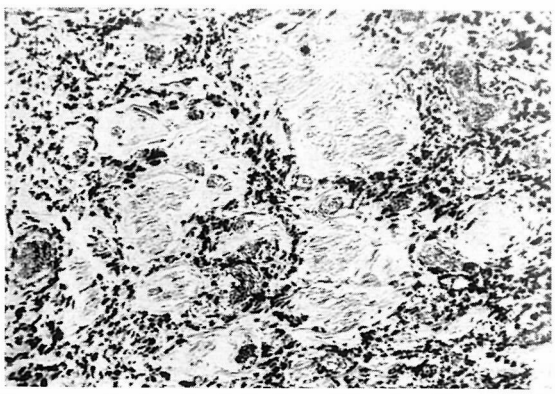

Fig. 2 Free muscular bundles in the lung (Hematoxylin and eosin, × 100)

of the lungs showed either emphysema or congestion, but their topographic relation with the granulomatous areas was not constant.

The visceral pleura was greatly thickened with proliferation of connective tissue which was generally well vascularized although partly hyalinized. The arterioles in the pleura showed remarkable medial thickening with muscular hyperplasia and hypertrophy which were more marked than those observed in the pulmonary tissue (Fig. 3). Most of the arterioles were obliterated although their intima and adventitia were little affected.

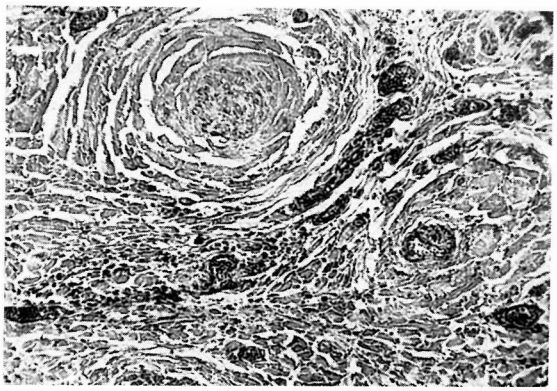

Fig. 3 Proliferated muscle fibers in the pleura (Hemtoxylin and eosin, × 100)

Peribronchial lymph nodes: Collagenous nodules of various sizes were observed in the central part of the lymph nodes, mostly in the lymph sinuses, although the larger one bulged into the adjacent follicle. These nodules were composed of laminated collagen fibers, completely devoid of cellular element and sharply demarcated from the surrounding lymphoid tissue. The peripheral zones of most follicles were marked by heavy infiltration of plasma cells and macrophages containing deposits of fine black particles. The intercellular deposit of amyloid substance was occasionally encountered in the central part of the follicles. Blood vessels in the lymph nodes were normal.

Spleen: The capsule was thickened and ragged, the trabeculae prominent. Typical nodule of collagen fibers could not be found. Dust-like particle could not be found in spite of careful examination with high magnification.

Liver: The capsule was thickened and subcapsular connective tissue proliferation was marked. Hepatic cells were generally atrophic. There were congestion and occasional hemorrhages in the central zone of the hepatic lobules. Periportal connective tissue was slightly increased but showed no tendency towards cirrhosis. Collagenous nodules could not be found.

Kidney: Cloudy swelling of the proximal convoluted tubules and enlargement of the glomeruli.

The other organs appeared normal.

Discussion

As Gardner[1] has pointed out, silicotic lesions may closely resemble tuberculosis. In the present case, however, the histologic pictures of the lung and of the lymph node were thought to support the diagnosis of pulmonary silicosis without complicating tuberculosis. The reasons are as follows: 1) The essential alteration was represented by the nodules composed of concentrically laminated collagen fibers. 2) Epitheloid cells, Langhans giant cells or any other cells that suggest a tuberculous nature of the lesion were practically absent. However, the presence of these cells by no means excludes the possibility of silicosis. 3) There were no areas of caseation or coagulation necrosis.

The interesting feature of this case was the existence of a hyperplastic and hypertrophic muscle element in the lung and in the pleura. Most of the muscle fibers were derived from hypertrophic media of arterioles which existed around the silicotic nodules. This explanation was supported by the demonstration of elastic membranes which encircled the muscle fibers. The change of these arterioles corresponded to the so-called "*mésovascularite hypertrophique musculaire*" that Mosinger[3] described as one of the vascular lesions in pulmonary silicosis. It is noteworthy that the affected arterioles in this material had a prominent longitudinal muscle element that is typical of bronchial arterial systems. More interesting was the occurrence in this material of free muscle fibers which existed as isolated bundles and were apparently unrelated to vessels or bronchioles.

The existence of muscular hyperplasia and hypertrophy in the lung was recognized by Rindfleisch[4], as early as 1872, in a study of "brown induration" of the lung. In 1953 Liebow and his colleagues[2] briefly reviewed the previous literature and added their own observation of 40 lungs of various chronic pulmonary diseases including pulmonary emphysema, bronchiectasis, bronchitis, bronchiolitis, pulmonary abscess and others. They stated that the hyperplastic or hypertrophic muscles that occur are derived from 1) bronchi and more distal air spaces, 2) blood vessels, especially the longitudinal layer of the bronchial arteries, 3) lymphatics, and 4) interstitial tissue not clearly associated with other structures.

The free muscle fibers observed in the present case may have originated from the interstitial tissue but a derivation from lymphatics can not be ruled out. Although the major part of the proliferated muscle fibers in the lung and in the pleura was apparently derived from hypertrophic media of arterioles, nothing certain can be said about the mehanism by which such a change of arteriolar media was brought about. Since hypoxemia increases tonus of the pulmonary arterioles[5], it seems reasonable to assume that hypoventilation consequent to pulmonary silicosis might have caused "work hypertrophy" of pulmonary arterioles. Further investigation is necessary in

order to substantiate the assumption.

SUMMARY

An autopsy case of pulmonary silicosis was studied histologically. Besides the ordinary histologic picture of pulmonary silicosis, the following findings were of interest: 1) Hyperplasia and hypertrophy of muscle fibers were frequently encountered in the thickened pleura as well as in the pulmonary parenchyma. 2) The proliferated muscle element could be classified into two types; one that was derived from the arteriolar media and one that was apparently unrelated to the blood vessels and existed as isolated bundle. As to the pathogenesis of the former, "work hypertrophy" of the arteriolar media was suspected, while the derivation of the latter was not determined.

REFERENCES

1) GARDNER, L. U.: The Similarity of the Lesions Produced by Silica and by the Tubercle Bacillus, *Am. J. Path.* **13**: 13-23, 1937.
2) LIEBOW, A. A., LORING, W. E., and FELTON, W. L. II,: The Musculature of the Lungs in Chronic Pulmonary Disease, *Am. J. Path.* **29**: 885-911, 1953.
3) MOSINGER, M.: Sur les altérations vasculaires dans la silicose pulmonaire, Nodules silicotiques perivasculaires, *Arch. mal. profes.* **18**: 776-781, 1957.
4) RINDFLEISCH, E.: Die Musculatur der kleinen Bronchien und des Lungenparenchyms, *Centralbl. med. Wissensch.* **10**: 65-66, 1872.
5) RÜTTNER, J. R. und GASSMANN, R.: Lungengefässveränderungen bei Silikose, *Schweiz. Zschr. allg. Path.* **20**: 737-744, 1957.