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Pathology of Talc Pneumoconiosis with Report of an Autopsy

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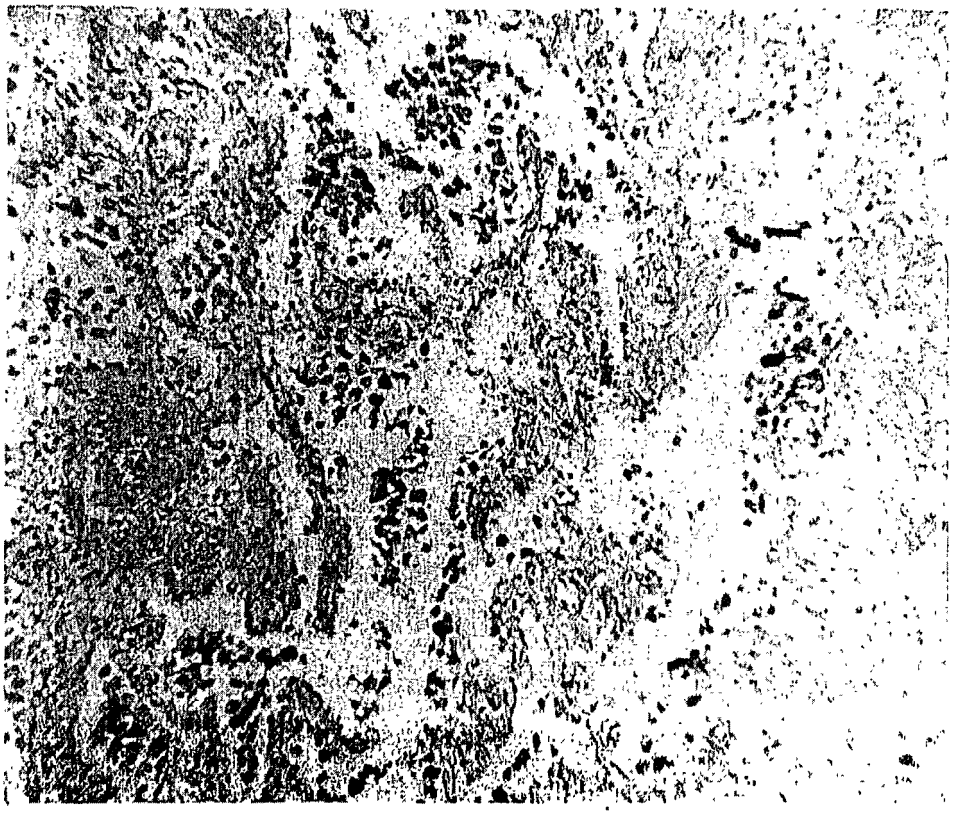
PNEUMOCONIOSIS is the general term applied to the effects of inhaled dust on the lungs. Only in recent years have most of the known important facts about this group of conditions been established by observations and experimental work. Some forms of pneumoconiosis such as anthracosis, due to the inhalation of coal dust, and siderosis, due to the inhalation of iron ore dust, are generally considered to be relatively harmless. Silicosis, from the inhalation of dust containing much uncombined silicon dioxide ("free silica"), and asbestosis, due to the inhalation of dust from the fibrous silicate, asbestos are generally recognized as serious forms of pneumoconiosis capable of causing pulmonary fibrosis and consequent disability. Silicosis was established as a disease entity about the beginning of this century and asbestosis rather more recently in 1924 by Cooke(1). The various aspects of silicosis and asbestosis have considered in detail by Lanza, et. al.(8). A brief consideration is given by Gardner(6).

Among other types of dust that have received recent attention in connection with the production of pneumoconiosis is talc, which is mined and milled in St. Lawrence County, N.Y., as well as several other parts of the United States. Reports of the working conditions and effects in this area have been made by Dreesen(2) and by Siegal, Smith and Greenburg(11). Porro, Patton, and Hobbs (10) have reported clinical and roentgenographic findings on fourteen cases of pneumoconiosis in talc workers including autopsy findings on five of the cases. In addition observations on the talc industry in Georgia have been reported by Dreesen and DallaValle (3). All of these observers have found cases with increased pulmonary densities and with disability in advanced cases.

The pathologic findings in the lungs of talc workers consist grossly of a general fibrous stiffening of the lungs and often old fibrous pleural adhesions. Microscopically there are found asbestosis bodies and pigment laden macrophages in the alveoli and respiratory broncioles. In addition there is thickening in many alveolar walls and septa with the reaction consisting of histiocytes, fibroblasts, loosely arranged collagen fibers, scar tissue and a few lymphocytes as well as some amorphous pigment. There is hyperplasia of the lymph nodules along the septa. In the alveoli poorly formed giant cells are sometimes present. There are also varying degrees of atalectasis, compensatory emphysema and sometimes metaplasic of alveolar epithelium into a layer of cuboidal cells in fibrotic areas. In addition there is sometimes complicating silicosis, tuberculosis, or other pulmonary disease. These findings are similar in nature to those described in asbestosis by Gloyne (7) and others.

The asbestosis bodies are the most specific finding. These are brown, beaded and dumbell shaped rods, in length, usually several times the diameter of a leukocyte. They have been shown to be composed of a thin silicate fiber which is covered by brown pigment which contains iron, probably hemosiderin. They were originally noted in the lungs of asbestos workers and were first observed in the lungs of a talc worker by Gardner(5) from a case observed clinically by Patton(9) and autopsied by Free(4). Talc is made from several silicates of similar chemical composition to asbestos and some of which are fibrous although not so much so as the minerals from which asbestos is made. These bodies have been found in no other conditions.

The following case with autopsy is the first available in a talc worker since those previously reported(10) and is of interest due to the similar pulmonary findings:—
History: J. R., age 46, male, white, a talc miner for 14 years. He entered the A. Barton Hepburn Hospital, Ogdensburg,



General View of Lung X300 showing rod shaped asbestosis bodies and macrophages in the air spaces and a fibrocellular reaction in the interstitial tissue.

N.Y., Sept. 7, 1945 under the care of Dr. J. R. Patton. He had been unable to work for three years because of difficulty in breathing. Two weeks before, he started to expectorate bloody sputum and complain of pain in the left side of the chest. X-ray findings revealed the left lung field below the level of the first interspace to be completely obscured and in the right lung field, a dense reticulation. The dyspnea continued to be severe and the patient expired on Oct. 30, 1945.

Gross Autopsy Findings: The body is that of a white, fairly well nourished, well developed male, 46 years of age. There is cyanosis of the finger tips and slight hypostatic lividity and fully developed rigor mortis. The legs are edematous up to the mid-portion.

The abdominal panniculus adiposus is yellow and moderate in amount. The peritoneal cavity contains a considerable amount of serous fluid. The liver is three fingers below the costal margin and on section shows a pronounced nutmeg appearance. The spleen is large and soft with prominent Malphigian corpuscles. The pancreas and gastrointestinal tract contain no gross lesions. The kidneys are congested and the medulla of the adrenals is soft from autolysis. The bladder and prostate are normal.

Both pleural cavities are partially obliterated by fibrous and fibrinous adhesions. A small amount of cloudy fluid is present on the left side. The pericardial sac contains a small amount of serous fluid.

The lungs are firmer than normal, yellowish as in chronic passive congestion, and have areas of firm triangular bluish discoloration. On section these are hemorrhagic and the lung cuts more firmly than

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The left auricle is dilated and hypertrophied and there is a similar hypertrophy of the left ventricle. Section of the heart shows some sclerosis of the vessels. There is marked stenosis of the mitral valve which permits a small finger to enter. There is slight arteriosclerosis about the aortic valve.

Microscopic Findings: There is scarring of the myocardium and some Aschoff bodies are present.

In the liver there is marked central lobular congestion and necrosis.

One section of lung consists largely of hemorrhagic infarct. Other sections reveal many alveoli containing fluid, erythrocytes, and macrophages containing hemosiderin. Some of the alveoli contain asbestos bodies. Localized emphysema and atalectasis is present. Many of the interlobular septa and some alveolar walls are thickened by a fibrocelluar reaction consisting of histiocytes, scattered lymphocytes, fibroblasts, collagen fibers and amorhous brown pigment. No tubercles or silicotic nodules are found.

Pathologic Diagnosis: Rheumatic myocarditis and endocarditis with mitral stenosis; pneumoconiosis (talc) with fibrosis; chronic passive congestion of the lungs; hemorrhagic infarcts of lungs; chronic passive congestion of lungs, liver, viscera and lower extremities; ascites.

DISCUSSION

The lung findings in this case are those of a moderately advanced talc pneumoconiosis similar to the findings in all previous such cases coming to autopsy. In addition there are many heart failure cells. These are the same as the dust laden macrophages in pneumoconiosis but are in greater numbers here in proportion to the amount of pneumoconiosis.

A photograph showing the fibrocellular reaction and asbestosis bodies in the air spaces is shown.

The fibrosis present in this case is considered to be due to pneumoconiosis. Infection, especially tuberculosis, may cause greatly increased fibrosis in pneumoconiosis but there is no evidence of this or other pulmonary infection in this case.

The heart disease is the principal disease. However in pneumoconiosis with fibrosis disability may occur from the fibrous alone with the principal symptom being dyspnea which is caused by the destruction of lung tissue and perivascular fibrosis.

SUMMARY

1. The general aspects of pneumoconiosis and the pathology of talc pneumoconiosis are considered.

2. A case report including autopsy findings in talc pneumoconiosis is reported.

3. The findings in this case are similar in general nature to those previously reported and are similar to those reported in asbestosis.

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