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# TALC PNEUMOCONIOSIS



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There is increasing evidence that occupational inhalation of pulmonary irritants is one of the major causes of pulmonary fibrosis. Free silica is a common etiologic agent. Talc, a hydrous magnesium silicate, is an unusual cause for such pneumoconiosis. The following case demonstrates the roentgen and clinical characteristics of talc pneumoconiosis.

## REPORT OF A CASE

The patient, a 65-year-old white man, was referred for roentgen study of the chest on June 19, 1950, because of a chronic cough which was intensified during the preceding week. He had a cough of many years' duration accompanied by production of frothy sputum. On June 9, 1950, sneezing, coryza, intensified cough, and purulent postnasal discharge and expectoration developed. There was accompanying onset of exertional dyspnea. Walking three blocks caused dyspnea.

For a period of 24 years since 1926, he had been employed in the handling and packaging of elastic cord (rubber thread) coated with talc powder. In the handling of this rubber, he removed rubber ribbons, heavily coated with talc, from their containing box. He fed one end of the rubber into a reed that separated the strands; the rubber strands then passed onto a spindle which made skeins or cones of rubber thread. From the moment the containing box was opened, the atmosphere of the working

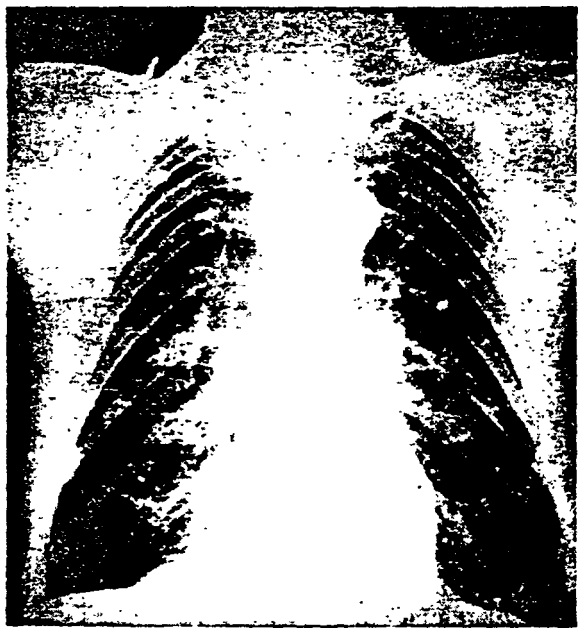

Fig. 1.—Chest, posteroanterior projection. This demonstrates diffuse pulmonary fibrosis, pulmonary emphysema, pleural thickening, and talc plaque in right upper hemithorax.

area was cloudy with talc powder. No exhaust or ventilating fans were present. In 1914 a right thoracotomy for empyema was performed.

Physical examination on June 9 revealed a thin, white man with a temperature of 99.4 F, a pulse rate of 112, and blood pressure of 110/74. There was mucopurulent nasal secretion. There was a thoracotomy scar over the right lower posterior hemithorax. Chest expansion was restricted. Coarse moist rales as well as areas of bronchial breathing were present through both lungs. No clubbing of the fingers was present. The pati was treated with bed rest. He received penicillin orally, in muscularly, and by nose drops.

On June 13, he was clinically improved and returned to wc Temperature and pulse rate were normal. Cough was less pro nent, but dyspnea was still marked on walking four blocks uphill. There was minimal night cough when he was recumbe

Fig. 2.—Chest, lateral projection. Note increased anteroposterior ameter accompanying the pulmonary emphysema, foci of pulmon calcification.

There were momentary episodes of respiratory obstructi Coarse moist rales were still heard over the lower lobe of right lung. Fluoroscopic and radiographic study of the chest June 19 revealed marked reduction in thoracic respiratory n bility. There was reduced diaphragmatic excursion dur respiration.

*Lungs.*—There was evidence of diffuse pulmonary empi sema, characterized by low position of the diaphragm and creased anteroposterior thoracic diameter. There was diff linear intensification of the pulmonic markings owing to pulm nary fibrosis. This was most prominent in the right lung. Sta foci of calcification due to previous tuberculous infection w visualized in the upper lobe and hilar region of the left lung ( 1 and 2).

*Pleura.*—There was thickened pleura present, most promin on the right, where it produced stragihtening of the right he diaphragm. A linear homogeneous opacity, 9 cm. in length a 1 cm. in width, was demonstrated in the upper right hemithor. Its axis was obliquely vertical, and it apparently represente talc pleural plaque.

*Heart and Mediastinal Structures.*—There was no abnorn mobility during respiration. The heart was not measura enlarged. There was increased convexity of the left cardiac bor owing to prominence of the right ventricle. Elongation of aorta due to arteriosclerosis was present. The sputum was ne tive for tubercle bacilli.

A mixed catarrhal vaccine series was administered in an tempt to improve immunity to respiratory pathogens. Penici has been used periodically for acute episodes of tracheobr chitis with purulent secretions. The patient has been relativ comfortable though not entirely free of cough.

An electrocardiogram on Feb. 13, 1951, revealed low amplitude of the QRS component in the limb leads. It was otherwise natural.

COMMENT

Talc pneumoconiosis has been described in talc miners and millers. Since talc is used industrially as a filler, as a dusting powder, and as an absorbent, the possibility of excessive exposure is not a limited one. Bethene, in 1935, experimentally demonstrated its irritant characteristics when pleural adhesions were caused in cats and dogs by the intrapleural insufflation of talc powder. Since the free silica content of talc is 0.5% or less, its irritant characteristics are apparently not a result of its free silica content.

Clinically, talc pneumoconiosis usually caused dyspnea, chronic cough, chest pain, and fatigue. The dyspnea is usually more intense than the roentgen manifestations of fibrosis would suggest. Limited chest expansion is invariably present. The patients are usually thin, minimally cachectic in appearance, and in 70% of cases exhibit curving nails or clubbed fingers. The patient described herein exhibited all of these changes except clubbing of the fingers.

The roentgen changes resulting from talc pneumoconiosis are characterized by fibrosis, emphysema, pleural thickening, pleural and subpleural plaques ("talc plaques"), and a minimally increased incidence of tuberculous infection. The fibrosis is of a fine linear and granular character, similar in type to that visualized in asbestosis. It tends to be most intense in the midportions of the lungs, extending into the basilar segments of the lower lobes. A moderate degree of diffuse pulmonary emphysema is present. Partially owing to the emphysema and partially owing to the pulmonary fibrosis, there is a prominence of hilar and intrapulmonic segments of the pulmonary arteries.

The pleura exhibits diffuse thickening and fibrosis. In addition, linear areas of pleural density, so-called talc plaques, occur. These were seen in 6.3% of all talc miners.[1] They occur in the peripheral and subpleural aspects of the visceral pleura. These may vary from single, linear, homogeneous, sharply marginated opacities, several centimeters in length, to irregularly shaped, extensive opacities of varying density. It is felt these occur secondary to accumulation of talc in peripheral lymphatics with lymphatic dilatation and stasis, associated with calcium localization.

There has been suggestive statistical evidence of increased susceptibility to significant tuberculous infection.

SUMMARY

A case of talc pneumoconiosis is reported with typical clinical and roentgen characteristics. This report seemed indicated in order to direct attention to talc as an occupational hazard and to the probable significance of other inhaled pulmonary irritants as causative agents in unexplained pulmonary fibrosis.

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## BRONCHOGRAPHY IN IODINE SENSITIVITY

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The instillation of iodized oil into the bronchial tree is widely used in the diagnosis of bronchiectasis, the delineation of distorted or obstructed bronchi, and the determination of communications between bronchi and the pleura, skin, or air cysts. All the radiopaque preparations commonly used in bronchography today contain iodine. Those most frequently used are lipiodol,* which contains 40% iodine in poppyseed oil, and iodochloral,* which contains 27% iodine and 7.5% chlorine in organic combination with highly refined peanut oil. Bronchography with these agents is usually innocuous, but untoward reactions, such as iododerma and asthma-like reactions with fatal outcome in two cases, have been reported.[1] It is imperative, therefore, that proper inquiry be made as to sensitivity to iodine prior to the use of any iodine-containing contrast medium.

A patient with suspected bronchiectasis was recently admitted to the hospital for study. The presence of pronounced iodine sensitivity made the use of iodized oil as a contrast medium for bronchography inadvisable and even hazardous. However, since the use of corticotropin (ACTH) had been reported in the treatment of sensitivity to iodine,[2] it was decided to carry out the iodized oil study after preliminary desensitization with corticotropin. A satisfactory bronchogram with minimal reactions was obtained. Because of this favorable outcome, the following report is considered of interest:

REPORT OF CASE

I. O., a 46-year-old white woman, was first seen in October, 1950, because of severe paroxysmal cough, copious foul expectoration, and pain in the left lower part of the chest posteriorly. A diagnosis of suspected bronchiectasis had previously been made, in 1944, after an iodized oil study. At that time severe swelling of the submaxillary glands developed, along with "blotches all over the body resembling strawberry hives," which lasted 10 days after the bronchogram. Subsequent bronchograms, in 1945 and 1947, again produced reactions manifested by swelling of the submaxillary glands, fever, and hives. Although these studies revealed equivocal evidence of bronchiectasis in the lower lobe of the left lung, a segmented resection was done in 1947. Symptoms were then few, until November, 1948, when cough recurred. This had become progressively severer and was productive of several cups of foul sputum daily at the time of the patient's admission to the hospital.

Physical examination disclosed nothing essentially abnormal except in the lungs, where there was impairment of percussion and distant breath sounds over the lower lobe of the left lung. No rales were heard.

Fluoroscopic and roentgenographic examinations revealed portions of the posterior segments of the 9th and 10th ribs to be missing. The left side of the diaphragm was slightly elevated

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