A Current Hypothesis of the Lymphatic Transport of Inspired Dust to the Parietal Pleura*

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In the light of a case report, we will describe a hypothesis of the lymphatic transport mechanism of inhaled dust particles to the parietal pleura. The autopsy of a molder who had been employed for 41 years in iron foundries showed heavy mixed-dust pneumoconiosis. In the parietal pleura there were black linear streaks following the course of intercostal arteries. These formations were microscopically interpreted as periarterial lymphangitis and fibrosis caused by the dust. According to our hypothesis, the dust particles have been transported from the lungs via the lymphatic vessels partly in retrogression. The mechanism is supposed to be the same in the transport of small asbestos particles causing pleural plaques.

I
n cases of pneumoconiosis, especially those caused by fibrous dust, parietal pleural thickening and plaques are often encountered, in addition to ordinary pulmonary changes. The pathogenesis of pleural plaques is still obscure. Several theories have been advanced for similar lesions in asbestosis, but none of them has been generally accepted. Siegal,1 Kiviluoto2 and Thompson3 presented the so-called "mechanical theory," according to which the asbestos fibers penetrate the visceral pleura and, because of the respiratory movement, rub the parietal pleura, causing ulcerations. Later, the lesions become calcified. According to the chemical theory of Lawson,4 the alkaline phosphatase in the parietal pleura is activated by the magnesium and talc of asbestos dust, leading to calcification. Meurman5 suggested that the plaques originated from the combined effect of both physical and chemical factors.

A case of heavy grade mixed dust pneumoconiosis (anthracosilicosis) is presented. A linear distribution of dust particles in the region of the parietal pleura was found at autopsy. The localization of coal and silica particles corresponded to the distribution of the intercostal lymphatics. It is suggested that the dust particles might have been transported there by a retrograde lymph flow. Similarly, small asbestos fibers might also be transported to the parietal pleura.

Case Report

Clinical Findings

A 77-year-old man had been employed for 41 years in iron foundries as a molder. The working conditions had always been those of an old iron foundry, dusty and sooty. After exposure for 34 years, the patient began to feel dyspneic, and two years later a diagnosis of stage I pneumoconiosis was made. Calcification was detected roentgenologically in the hilar regions on both sides. There was slight nodulation in the middle part of the lungs, with emphysema in the upper part. The left ventricle was rounded. In spite of these findings, the patient continued his work for another five years, after which time the pneumoconiosis and emphysema had progressed nearly to stage II. The patient felt continually dyspneic, the severity of which varied from time to time as did his cough and mucous production. In 1970, his condition deteriorated and the patient died of a pulmonary embolism.

Autopsy Findings

The right lung weighed 1,150 gm and the left 900 gm. There was extensive anthracosis and moderate emphysema of both lungs. The cut surface presented black nodules, and reticular formations were seen on the visceral pleura. In the peripheral branches of the pulmonary arteries emboli were found. There was anthracosis of the mediastinal and retroperitoneal lymph nodes. In the parietal pleura on both sides linear anthracotic streaks were found distributed intercostally (Fig 1). The heart weighed 480 gm and, in addition to coronary sclerosis, showed hypertrophy and myofibrosis. The other main findings were: small thrombi in pelvic veins; small infarcts in both kidneys; small softened areas in the occipital regions of the brain; and heavily pigmented skin in the scrotum.

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Figure 1. Macroscopic view of right parietal pleura at autopsy. Dark lines indicate collection of dust corresponding to localization of perivascular intercostal lymphatics.

Microscopic Alterations

The pulmonary changes resembled mixed-dust pneumoconiosis and, more accurately, the compound silica-coal dust nodule type of coal workers' pneumoconiosis (Fig 2). In the lung parenchyma, as well as in the perivascular and peribronchial regions, fibrotic nodules rich in both coal and silica particles were seen. Mucoid degeneration was encountered in the walls of large elastic arteries and medial sclerosis and intimal fibrosis in the smaller arteries and arterioles. In the parietal pleura there were small fibrotic nodules under the mesothelium on both sides (Fig 3). They consisted of inflamed and fibrotic perivascular lymphatic vessels (Fig 4)

Figure 3. Fibrotic nodules lying intercostally in parietal pleura.

containing silica and coal particles (Fig 5). Deposition and some kind of fibrosis were also encountered in cervical, mediastinal and retroperitoneal lymph nodes. The mixed-dust pneumoconiosis (anthracosilicosis) was regarded as the basic cause of death, with the pulmonary embolism as the immediate cause.

DISCUSSION

The pulmonary roentgenologic findings of this type of dust pneumoconiosis correspond to the changes of silicosis. Pleural plaques are not often seen on x-ray films in connection with silicosis, and were not seen in this case. In the present case, attention was paid to the extensive extrapulmonary distribution of coal and silica particles in the region of the retroperitoneum and, especially, the thorax.

The affection of the cervical and mediastinal lymph nodes seems to be the natural result of particle transport via the lymphatic vessels. Which mechanism could have caused the intercostal distribution of coal and silica particles under the parietal pleura? Three possibilities in our opinion should be taken into consideration: (a) penetration from the lungs through the visceral pleura and the pleural cavity and resorption into the parietal pleura; (b) propagation through blood vessels; and (c) transport via lymphatic vessels.

Of the above, the transpleural route seems to be the least probable for the following reason: If coal
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Figure 5. Detail from Figure 4. In polarized light silica particles in addition to black coal deposits seen as bright flecks.

and silica particles had reached the pleural cavity, one could have expected a more uneven and more reticular distribution of particles.

If the transport had been along the blood vessels, changes caused by the dust probably would have been present in more well vascularized organs than just the pleura. Degenerative changes in intercostal arteries would also have been anticipated.

A retrograde transport of the particles via thoracic and intercostal lymphatics seems to offer the best explanation of the findings (Fig 6). Particles may have been carried, either free or phagocytosed by macrophages, through the normal lymphatic vessels from the alveoli to hilar and mediastinal lymph nodes. When the phagocytosing capacity of the reticuloendothelial cells in these nodes has been exceeded, the dust particles have been conveyed centrally to the anterior and posterior bronchomedial lymphatic trunci on both sides, according to von Hayek7 (Fig 6). The lymphatic vessels from the parietal pleura also empty into these trunci. As a consequence of dust load, particles may be carried by retrograde flow from bronchomedial trunci to intercostal vessels leading to lymphangitis and reactive fibrosis. The alterations in the retropitoneal lymph nodes could also be explained by the retrograde transport of particles. Such a retrograde flow has been mentioned earlier in connection with silicosis and other pneumoconioses.8

In spite of many doubtful, and even negative, opinions,3,9 we consider it possible that the same mechanism could also be the basis for the development of the pleural plaques in asbestosis. Some investigators10,11 stress the role of small particle size (< μm) in the pathogenesis of asbestosis. The size of silica particles inhaled into the lungs is less than 5 μm; asbestos particles of these sizes also occur and might be carried to the parietal pleura in the same way. The lymphatic route is also mentioned by Meurman.12 In the vicinity of the anthophyllite mine in eastern Finland nonoccupational pleural plaques are encountered at roentgenologic mass surveys in 6 to 9 percent of the population.13 This may be due to the fact that especially the smallest asbestos fibers move easily in the air. Because of their small size, they might be conveyed to the parietal pleura by the same mechanism. Hourihane14 mentions that asbestos fibers, but not asbestos bodies, have been found in parietal pleural plaques. In the experimental material of Kanazawa,15 asbestos particles were detected in pleural milky spots in one case. Nagelschmidt16 reports that he has seen material resembling asbestos in the parietal pleura, but he has no explanation for this finding. Selikoff17 also reports on pleural calcifications in the same paper: "The localization of the calcification in the anterior mediastinal pleura corresponds to the course of lymphatic vessels." Selikoff17 continues that in tomography investigations small linear calcified areas have been detected in the middle of thick pleural fibrosis. These findings support our hypothesis.

Figure 6. Schematic drawing of thoracic lymphatics. Arrows and dotted vessels show hypothetic transport route of dust particles on left side.

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