Carbon pneumoconiosis in a synthetic graphite worker

W. B. LISTER and D. WIMBORNE
Pneumoconiosis Medical Panel, Manchester, and Pathology Department, Salford Royal Hospital


A case of carbon pneumoconiosis in a synthetic graphite worker was described by one of us (Lister, 1961). This man has now died and it seems appropriate to report on his clinical progress and the pathological findings as no further cases of pure synthetic graphite pneumoconiosis have appeared in the literature although animal experiments have been described (Nau, Neal, Stembridge, and Cooley, 1962).

Our patient was aged 60 when first seen and had been turning and grinding synthetic graphite bars for 17 years. Details of the dust composition were given in the earlier article. Previously he had been a hod carrier in the building trade for 25 years. He complained of cough, sputum, midsternal pain, exertional dyspnoea, tiredness, and giddiness.

There were no abnormal physical signs in the chest but there was a marked thoracic kyphoscoliosis. X-ray examination showed pneumoconiosis category M3. Pulmonary function tests showed moderate impairment of maximum breathing capacity and flow rates, reduced vital capacity, and a transfer factor at the lower limit of the normal range.

He left his occupation two months after pneumoconiosis had been diagnosed but worked a few months as a sweeper and errand man at his old firm, with only occasional exposure to graphite dust. He then worked part-time, at a laundry, stacking parcels, and finally retired at the age of 68 years.

There was little change in his general condition up to the time of his last examination in September 1969. His weight fluctuated between 10 st 3 lb (64.9 kg) and 8 st 10 lb (54.9 kg). He smoked 10 cigarettes a day. Symptoms varied little throughout the period of 10 years but from 1967 he complained of swelling of the ankles and had a phlebothrombosis of the left leg in 1969. Clinical examination revealed poor chest expansion but no abnormal physical signs, except in September 1969 when there was prolonged expiration and a few scattered expiratory rhonchi. His blood pressure remained stable, around 170/90 mmHg. Lung function tests showed a gradual deterioration:

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<th>FEV₁,₀</th>
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<tr>
<td></td>
<td>Observed</td>
<td>Predicted</td>
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<tr>
<td>1959</td>
<td>2.0</td>
<td>3.0</td>
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<td>1969</td>
<td>1.2</td>
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<td>66</td>
<td>56</td>
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Radiologically there was no significant change during the entire period of observation—category M3. He was said to have been 'reasonably well' on the day before death. On 21 November 1969 he was found collapsed on the floor at his home where he
lived alone. He was taken to the Casualty Department of Salford Royal Hospital where he was found to be in hypothermia (83°F), comatose, pulseless, and with no recordable blood pressure. He died soon afterwards.

Post-mortem findings (D.W.)

Death was due to thrombotic occlusion of a branch of the left coronary artery, the coronary vessels being markedly atherosclerotic. There was systemic and cerebral atherosclerosis. Both ventricles of the heart were hypertrophied. The lungs were emphysematous and, generally, finely nodular. There were a number of minor lesions of various organs that do not appear relevant to the problem of pneumoconiosis.

Histological findings

Lungs  There was a general distribution of roughly spherical, fibrous nodules varying in size from microscopic to 5 mm diameter, about one-third being larger than 2 mm. Some consisted of whorls of fibrous tissue, others of strands radiating from a centre (see Figure). The fibres were coated with dense collections of opaque, black granules (H & E stain). Prussian blue stain revealed iron in very small amount in some of the nodules. In addition, scattered throughout were small collections of black granules in the walls of alveoli. These had not excited fibrous tissue formation. Many other alveoli had no granules in the walls yet the lumina were filled with fibrous tissue containing dense, black agglomerations of granules. There were also macrophages loaded with black granules lying free in some acini, often associated with macrophages laden with haemosiderin (Prussian blue reaction positive).

Hilar lymph node  This showed gross sinus hyperplasia with marked deposition of black pigment in the pulp outside the sinuses but never in the lymph follicles. There was no fibrosis. The sinuses contained haemosiderin granules (Prussian blue positive).

FIGURE  The edge of a fibrotic nodule containing carbon pigment (H & E ×48). P indicates desquamation of pigment-loaded alveolar cell.
Incineration (R. D. Hunt)
The ashed material from the lung bases and hilar lymphatics showed little or no birefringent particles, indicating the absence of silicious material.

Analysis of the lung material dried to a constant weight at 60°C gave the following result:
- Carbon 88.8% -9.5%
- Fe 0.36% -0.37%
- Mg 0.027% -0.028%

Discussion of the pathological process
The lungs show the end-result of a long-standing process together with incidental changes due to chronic heart disease, chronic bronchitis, and senile emphysema. Yet cells loaded with pigment are seen in the many septa which are still of normal thickness and free of fibrosis, and similar cells lie free in the alveoli. In the present case many of these cells are certainly carrying recently inhaled carbon particles and iron particles derived from the red cells, but others are carrying dense pigment like that in the fibrotic nodules, and the latter phenomenon is reported as a typical process in silicosis. As in most cases of pneumoconiosis, the lungs were examined years after initial exposure to the offending dust, and years after exposure had ceased, and the active carriage of dense pigment is therefore surprising. This case shows that where the pigment granules are in fibrous tissue they are statically bound. There are active macrophages and sepal cells carrying pigment in the walls and lumina of the many alveoli which are not thickened or fibrosed, whereas the fibrotic nodules show no pigment-carrying, free cells. Many of the nodules consist of almost acellular collagen. Many sepal cells containing pigment have lost their nuclei and are in the process of desquamation into the alveoli.

Since the now aged, emphysematous lung is still capable of delivering pigment to the sputum from the alveolar walls, it must be assumed that it was even more capable of doing so when initial exposure to the dust hazard occurred many years ago. Hence it was the fibrous reaction which prevented clearance of the dust, and such a reaction could be due to the dust itself or to coincidental infection or immune response. To put it another way, a subject not suffering from bronchitis or other lung infection nor having a tendency to fibrous reaction in the lung might be able to inhale injurious
dusts for long periods and neither develop fibrotic nodules nor accumulate the dust in the lung tissues. In this context it is of interest that a significant relationship has been noted between pneumoconiosis and rheumatoid arthritis (Caplan, 1953). In rheumatoid arthritis a prolonged antigen-antibody reactive state is associated with fibrous 'healing', and it is the latter which produces the major defects of the disease.

With regard to the causative dust in this particular case, the post-mortem findings prove that the diagnosis in life was correct and that nodular fibrosis was caused by prolonged inhalation of 'nearly pure' carbon in the form of synthetic graphite and in the absence of any significant silica content.

Since the presence of silica or silicates is found to be unnecessary for the development of nodular fibrosis, the question arises what factor (? antigen) is responsible and whether this factor is perhaps responsible for the development of other or all types of pneumoconiosis including silicosis, the admixture of silica producing only a more serious type of disease. In view of the rarity of pneumoconiosis cases with no exposure to silica, any investigation of this problem will require great patience and perseverance.

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References

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