PNEUMOCONIOSIS DUE TO GRAPHITE DUST

BY

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Lassar Dunner (1945a) recorded the clinical and radiological findings in five men who had been exposed to "pure graphite" dust. They had been exposed for 17, 18, 20, 20, and 34 years respectively, and no other dusts were known to have been implicated. Three of the men had cough, sputum, and dyspnoea; the other two had no complaints. Radiographs showed reticulation with nodulation, and the question was raised in correspondence (Dunner, 1945b) whether there was a tissue reaction consistent with pneumoconiosis. Dunner believed that there was, and in later publications (Dunner and Bagnall, 1946; Dunner, 1948) described a form with a "snowstorm" appearance which became nodulated and in one case formed a cavity. It is doubtful, however, if such a thing as "pure graphite" exists commercially (except for manufactured varieties like "kish") because, as explained by Dassanayake (1948), graphite is mined from vertical lodes in granite rock, and the miners develop similar lung changes to those found in metalliferous miners.

The two fatalities here recorded followed long exposure to a mixed dust of graphite, free silica, various silicates, and possibly other substances used in the manufacture of crucibles. The hazard was not strictly comparable with Dunner's cases, therefore, but was more like that of coal-miners, judging from King and Nagelschmidt's (1945) analyses of dusts extracted from the dried lungs of South Wales coal-miners. There they found both free silica (quartz) and silicates (mica and kaolin) mixed with the coal. In our cases coal was replaced by graphite.

Case Reports

Case 1.—J. F., a man aged 59 years, had been employed by the same firm for 37 years. He was leader of a gang unloading raw material for making crucibles from barges at the company's wharf. This work was done partly out of doors and partly in the factory, where dust exhaustion plant was used. The material unloaded consisted chiefly of graphite, but sand and probably other substances were handled from time to time, as the formula of the crucible "mix" was complicated and included various silicates in addition to graphite and sand. The graphite was received from Ceylon and Madagascar. Latterly the sand had been delivered in bags.

This man first attended the Brompton Hospital in 1939, when silicosis was diagnosed. He complained of shortness of breath for one year, tightness in the chest, a productive cough with much thick sputum, lassitude on slight exertion, and some lower abdominal pain. Apart from influenza in 1937 he had had no previous illnesses. He weighed 140 lb. (64 kg.), afebrile, not wasted, and apart from short distant breath sounds he had no abnormal physical signs. The pulse and respiration rates were normal. The blood pressure was 150/90 mm. Hg. The blood sedimentation rate was 25 mm. (Westergren) in an hour, the vital capacity 2,400 ml., and sputum repeatedly failed to disclose any tubercle bacilli. A radiograph (Plate 1a) showed emphysema at both bases and nodulation in the upper and middle zones of both lungs with some massive fibrosis in the upper zone of the right lung. In the next year he gained half a stone in weight but became increasingly short of breath.

For the last eighteen months of his life he was unfit for regular employment, and in December, 1942, his health rapidly deteriorated. He was admitted to St. James's L.C.C. Hospital, and died the same day.

Autopsy.—The body was thinly covered, and there was no clubbing of the extremities. The upper respiratory and digestive tracts were healthy. Soft, recently formed adhesions obliterated the pleural sacs posteriorly and there was old-standing thickening of the visceral pleura over the right middle lobe of the lung. The inter-lobar fissures were closed by adhesions on this side. Both lungs were markedly pigmented and emphysematous bullae were present at the apices and bases. On section copious glistening black mucous secretion exuded from the cut ends of the bronchi. The bases of the lungs were congested and there were some small white patches of bronchopneumonia in these areas. Small black spherical fibrotic nodules, varying in size up to a third of an inch (0.8 cm.) in diameter, were distributed irregularly throughout the lungs, and were especially prominent immediately beneath the pleura. In the upper and lower lobes on both sides these nodules had become confluent to form rounded fibrotic masses 1 to 3 in. in diameter. One of these masses in the
upper part of the right lower lobe contained a ragged cavity filled with lustrous black mucoid secretion. The mediastinal glands were enlarged, pigmented, and fibrotic. The heart was enlarged and the chambers of the right side dilated, but no valvular lesions were present and the coronary arteries were patent and healthy. The thoracic aorta showed extensive atheroma. The liver exhibited signs of congestive failure and the spleen contained several small infarcts. No significant lesions were noted in other abdominal organs. The brain and meninges were healthy.

**Histology.**—The lung was extensively infiltrated with polygonal scale-like black particles with straight edges and sharp angles, ranging in size from 20 to 30 μ in their longest diameter to small fragments of the dimensions of pathogenic bacteria. Many of the particles were collected together in clumps, and the smaller ones were frequently phagocytized. A few were surrounded by golden-yellow crested envelopes ("graphite bodies") like the "anthracosis bodies" of coal-miner's lungs. On dark-ground illumination the particles retained their black appearance and could only be detected by their bright luminous edges, which had a wavy outline not shown up by transmitted light. These were not refractive with crossed Nicol prisms, but a few small refractive particles of quartz (2–3 μ) were seen.

The lesions in the lung tissue were patchy. In some parts there was relatively little dust, and the picture was that of a terminal bronchopneumonia with polymorphonuclear cell reaction. In others there were varying degrees of graphite infiltration with a collagenous response. In the least affected of these areas the lesion was confined to the respiratory bronchioles, which contained free particles of graphite, dust-laden macrophages, and cellular debris, and which were generally surrounded by peribronchial sheaths of reticulin fibres and imprisoned graphite particles. Similar sheaths were seen around the vessels. As the lesion extended the neighbouring alveoli became involved. They were filled with dust-laden macrophages and a few free graphite particles. The capillaries in the walls were obscured by cellular reaction around them, and finally they became compressed and unrecognizable. In peripheral portions of the lung the "pleural drift" of dust particles characteristic of pneumoconiosis was well marked, this accumulation of pigment resulting in collagenous thickening of the subpleural layer. These perivascular and peribronchial areas of reticulin-staining fibres became confluent in many places to form patches of fibrosis with irregular outlines in which all that remained to identify lung structure was an occasional short strip of low cuboidal epithelium from a small bronchus or a ring of wavy elastic tissue from an obliterated vessel. Side by side with these patches were whorled nodules of silicosis, the fibres of which had in many cases become swollen and hyalinized; a few had the irregular margins with tendrils trailing into lung tissue as described by Belt and Ferris (1942) in coal-miners' lungs ("Medusa head" or "mixed" nodules), but this was not a conspicuous feature. No evidence of tuberculosis was found.

**Case 2.—**J. M. C., a man aged 55 years, had been employed in the same factory as the previous patient. Before going to this factory he had been a stoker in the Royal Navy between the ages of 18 and 23 years; and between the ages of 23 and 27 years had worked as a p'asterer's labourer. He had then worked for 27 years as a mill hand grinding various raw materials used in the manufacture of crucibles. Two years before he came under observation he had been granted a certificate of total disability by the Silicosis Board.

When he first attended the Brompton Hospital, in March, 1945, his symptoms had already extended over the last eight years. He complained of increasing shortness of breath on exertion and cough with a little viscid sputum. The cough had been much worse during the previous five months. Four years previously he had had dry p'eurisy on the left side.

He was an elderly man with considerable dyspnoea on exertion. There was no clubbing of either fingers or toes. The chest expansion was poor, but otherwise there were no significant signs of disease in the lungs. His blood pressure was 110/65 mm. Hg. His weight was 123 lb. (56 kg.). Five months previously he had been 130 lb. (59 kg.), and ten years previously 151 lb. (69 kg.). His blood sedimentation rate was 19 mm. in one hour (Westergren), haemoglobin 100 per cent, leucocytes 12,000 per c.mm. of blood, with poly:orphs 80 per cent, lymphocytes 12.8 per cent, and eosinophils 3.6 per cent. Sputum was 1 to 2 oz. (30 to 60 ml.) daily, mucoid in character, and repeatedly failed to show tubercle bacilli.

A radiograph (Plate 1b) of the chest showed diffuse nodulation with many massive shadows in both lungs. There was evident gross emphysema.

**Autopsy.**—A post-mortem examination was made on Aug. 21, 1947. The body was much wasted. No clubbing of the extremities was noted. The upper digestive and respiratory tracts were healthy. Recent adhesions were present at the bases of both pleural sacs, and there were patches of thickened visceral pleura over both lungs with some scarring. Numerous small grey subpleural nodules of fibrosis were also noted. Both lungs were markedly pigmented and emphysematous. On section lustrous black mucinous secretion oozed from the cut ends of the bronchi. Distributed throughout both lungs were numerous small, rough, pigmented fibrotic nodules with focal emphysema around them. In both upper lobes and in the right middle lobe some of these nodules had become confluent to form large masses of fibrosis, and the mass in the left upper lobe contained two irregularly shaped cavities ½ to ½ in. (1.2–1.8 cm.) in diameter with rough ragged walls and filled with black lustrous mucinous secretion. The mediastinal glands were moderately enlarged, pigmented, and fibrotic. The heart was dilated and the muscle pale and flabby, but there was no valvular disease. The other organs were normal.
PNEUMOCONIOSIS DUE TO GRAPHITE DUST

Histology.—As in the previous case the lung was extensively infiltrated with graphite particles. In unstained sections they showed as thin plates with greenish edges, most of them being three- or four-sided figures varying from 10 to 20 μ in size. They were not refractile with crossed Nicol prisms, but a few small refractile particles of quartz were noted.

The histological pattern in the lung resembled that of the previous case but indicated a more advanced stage of fibrosis. The infiltration with graphite particles was much more marked, and the confluent areas of perivascular and peribronchial fibrosis were extensive. Conglomerate nodules of silicosis and mixed nodules were present. No active tuberculosis could be found, but there were minute areas of calcification near the cavity in the lung and in one hilar gland.

DISCUSSION

Graphite is widely distributed in nature. It is generally found with quartz (Milner, 1940) and usually as rounded plates or irregular dull black grains recognized chiefly by their appearance in reflected light. According to Strong (1945), it contains from 50 to 97 per cent of carbon in its natural form. Dunner and Bagnell (1946) gave figures for powered graphite purchased in the open market and for a fraction thereof prepared by blowing a current of air through it. The latter yielded the following: loss on ignition (mainly carbon) 52.7, total silica 25.1, and soluble silica 0.26 per cent.

Graphite has long been used industrially for the manufacture of pencils. It is also used extensively for the production of plumbago crucibles, when it is mixed with fireclay; for the manufacture of crucible steel and alloys and for arc-light carbons; for polishing and coating iron articles to prevent rusting; in an admixture with rubber as an acid-proof coating, and as a pigment and lubricant (Strong, 1945). It is therefore an important article in commerce. A crystalline form of graphite, "kish," is deposited in iron furnaces from molten iron on cooling. In all these various industrial processes there is a risk of exposure to the inhalation of the particles.

The two men whose fatal illnesses are described in this paper had both been exposed to dust in which there was a high proportion of graphite mixed with small amounts of free silica and various silicates. The resulting effects resemble those found in the coal-miner’s lung with graphite substituting for coal.

SUMMARY

1. Two fatal cases are recorded of pneumoconiosis due to a mixed dust containing graphite, free silica, and certain silicates. They both had massive progressive fibrosis superimposed upon dust reticulation.

2. At autopsy the lungs were blackened from the presence of graphite dust, which had also mixed with bronchial mucus to form a characteristic slimy glistening black secretion oozing from the cut bronchi. Massive nodules of silicosis were present with ragged cavities in them containing similar black secretion.

3. The histological pattern of the pulmonary lesion was that of a dust reticulation with superimposed conglomerate nodules of silicosis and mixed nodules resembling those of the coal-miner’s lung, as described by Cummins and Sladden (1930), Belt and Ferris (1942), Heppleston (1947), Gough (1947), and others.

4. "Graphite bodies" similar to the "anthracosisis bodies" found in the lungs of coal-miners were present in considerable numbers.

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REFERENCES


Plate II—(a) Case 2. Posterior aspect (cut surface) of right lung, showing nodular and massive silicosis.
(b) Case 2. Posterior aspect (cut surface) of left lung, with multilocular cavity.
PLATE III.—(a) Case 2. Glistening black mucinous secretion from cavity. (b) Case 1. Section of lung (× 50) showing perivascular and peribronchial reticulin fibrosis. Foot stain.
PNEUMOCONIOSIS DUE TO GRAPHITE DUST

PLATE IV. (a) Case 1. Reticulin fibres with 'graphite particles (x 250). (b) Reticulin fibres with bronchioles and alveoli filled with graphite dust. Haematoxylin.

(b)
PLATE V.—(g) Case 2. Frozen section of lung with silicotic nodules and focal emphysema. Haematoxylin, Eosin, Masson, section of lung (x 250) showing graphic body.
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