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CASE REPORT

Pseudoasbestos Bodies and Asteroid Giant Cells in a Patient with Graphite Pneumoconiosis

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GRAPHITE, a general term, is used to describe a form of carbon which occurs naturally, and a synthetic product made by the carbonization of oil. Natural graphite has a crystalline structure and is mined from seams that occur in siliceous rock. Because complete separation of the carbon from rock dust is not attempted and because the raw material is usually ground between granite stones, the commercial product contains various amounts (up to 10% by weight) of silicon dioxide (silica). Therefore, it is not surprising that massive exposure to graphite dust produces a pneumoconiosis resembling somewhat the anthracosilicosis of coal miners.[1-11] The pulmonary lesions consist of a heavy overload of dust, bands of fibrosis, patchy emphysema, cavitations and a variable degree of vascular sclerosis.[1-4, 12-14] The cavities contain an oily suspension of graphite dust. Nodular silicotic lesions are not found unless there is an unusually large silica component.

The disease has been reproduced in rats.[12, 15] Some idea of the incidence of the condition in industry may be gained from the fact that the literature contains 512 published cases, 24 of them with necropsies. Bovet[12] began the series to which the others have been added (Table I). The crystals of graphite and the fibres found in coal dust[3, 4, 14, 16] both give rise to peculiar structures, which are induced by the deposition of an iron-rich protein around the carbon particles. These are similar to asbestos bodies and talc bodies,[17] but the central core differs in each case. The awareness of this difference is important now that knowledge of the relationship between asbestosis and pleural mesotheliomata[18] has stimulated a more frequent examination of sputum for asbestos bodies.

A special feature of the pneumoconiosis in a graphite miner described by Jaffe[1] was an abundance of multinucleate giant cells containing asteroid inclusions. This paper describes another worker from the same mine who died at the Kingston General Hospital and showed at autopsy all of the features already described, with the addition of caseous tubercles in the lungs. An atypical mycobacterium had been cultured from his sputum.

This 50-year-old white man had been employed grinding graphite from 1938 to 1958. He farmed both before and after working in the mines. His exposure to silica dust was minimal in the graphite mill. Radiographs made in 1954 showed fine granular lung markings with dense mottling in both upper lung fields, less pronounced on the left.

In 1960, he was admitted to a sanatorium, complaining of nocturnal chest pain lasting from 15 to 20 minutes, associated with a cough productive of one-half cup of greyish mucoid sputum per day. He had no history suggestive of previous pulmonary tuberculosis. He had never been immunized with BCG vaccine but previously had had a 10-mm. reaction to atypical purified-protein derivative (PPD). He showed no reaction to typical PPD. He did not smoke. On admission, he had some degree of dyspnea at rest. Chest expansion was poor. Bronchial breathing was noted at both apices; medium rales and prolonged expiration were detected bilaterally. Radiographs showed a mass in the left upper lobe and a cavitated mass in the right upper lobe (Fig. 1). Two sputum smears in October 1960 and one in December 1960 contained acid-fast bacilli. Subsequent smears were negative. Culture of the three positive smears revealed a slow-growing, slightly chromogenic, acid-fast bacillus which was classified in Runyon's Group III. This organism was resistant to streptomycin, para-aminosalicylic acid and isoniazid, and was non-pathogenic for guinea-pigs and chickens.

By June 18, 1962, the density of the upper lung fields had increased; cavitation was noted in the left upper lung mass, and the cavity in the right upper lobe contained a fluid level (Fig. 2). The patient's condition deteriorated gradually. His illness was complicated by an antibiotic-resistant *Pseudomonas aeruginosa* bacteremia and progressive right heart failure. He died on June 28, 1962.

Pulmonary findings at necropsy.—On gross examination, there was thickening of the pleura at the apices and generalized pleural adhesions were ap-

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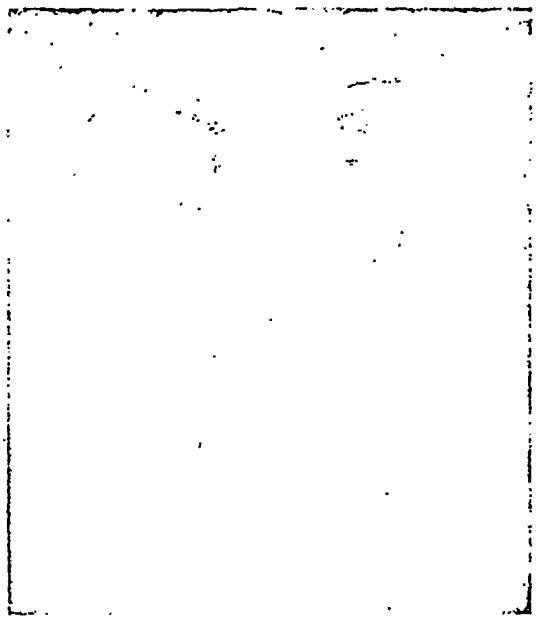

Fig. 1.—Radiograph taken on admission to hospital in 1960, showing fine to medium reticulation and masses in both apices, the right one cavitated.

parent. The lungs were black, nodular to the touch and heavy, and weighed approximately 900 g. each. In both apices, there were irregular, ragged cavities that had tough, indurated walls and were nearly filled with a grey-black, sticky, mucoid material. Fibrous cords bridged portions of the cavities (Fig. 3).

On microscopic examination, two main lesions were found. The first, foci of caseous necrosis mostly

Fig. 2.—Radiograph taken June 1962, showing medium to coarse reticulation with fluid level in right cavity, and cavitation of mass in left upper lobe.

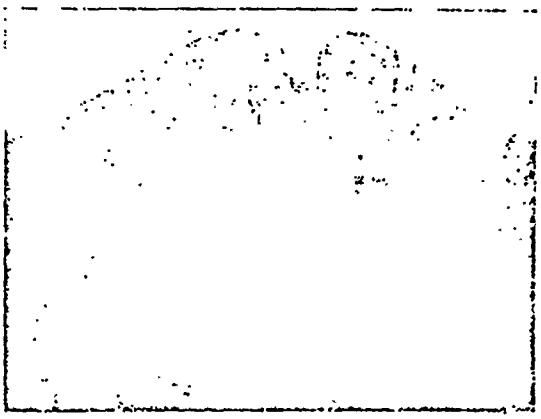

Fig. 3.—Cut surface of right upper lobe, showing large ragged cavity.

involving units of acinar size, were scattered in the indurated tissue of the upper lobe. They were poorly encapsulated and involved areas of lung that had been heavily pigmented. Most of the caseous cells

Fig. 4.—Tuberculo-graphite necrosis showing greater fibrotic reaction surrounding the graphite necrosis (L) than the tubercular (?) necrosis (R). (× 140.)

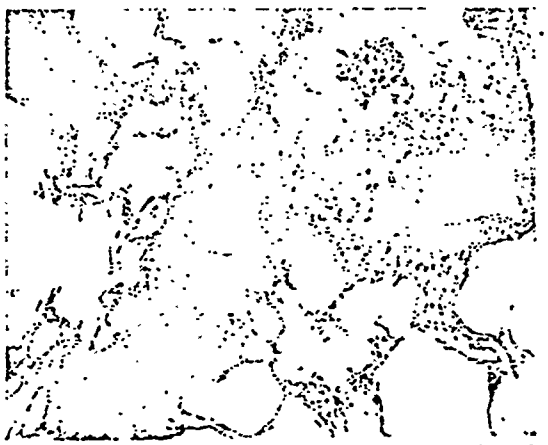

Fig. 5.—Emphysema and coarse peribronchial and perivascular fibrosis with massive carbon pigmentation. (× 100.)

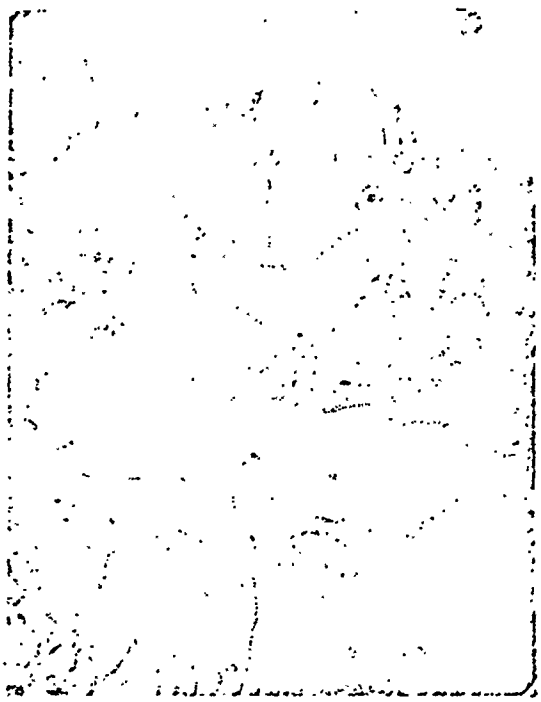

Fig. 6.—Asteroid giant cell in thickened adventitia of pulmonary vein. (× 450.)

Fig. 7.—Giant cells containing graphite crystals and carbon fragments coated with protein pseudoasbestos bodies). (× 1000.)

could still be recognized as leukocytes and were identifiable, therefore, as small areas of tuberculous pneumonia (Fig. 4). Acid-fast bacilli were found in these areas.

The second type of lesion was the graphite pneumoconiosis. No silicotic fibrous nodules were found, but there was variable fibrosis, ranging from fine reticulation to dense bands of collagen that had

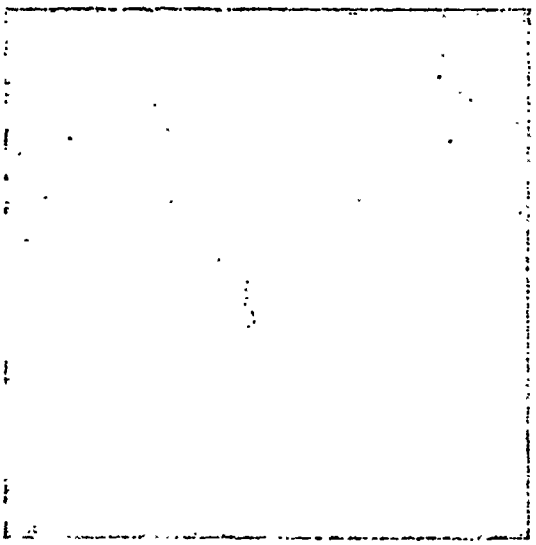

Fig. 8.—Pseudoasbestos body in lung after flaming. (Turnbull's blue, × 1000.)

a predominate perivascular and peribronchial distribution (Fig. 5). Similar changes were described by Bovet[12] following pure graphite inhalation in rats A few of the medium-sized arteries were thrombosed. The remaining lung tissue showed severe emphysema. In the areas of massive pigmentation, there were particles of carbon, free in air spaces, in foreign body giant cells, and also forming the core of pseudoasbestos bodies. Giant cells containing asteroid bodies were numerous (Fig. 6). Some of the particles were coated with small globules of protein and lay either free or in giant cells (pseudoasbestos bodies) (Figs. 7 and 8). All of these changes were least in the basal segments where emphysema predominated.

With the Prussian-blue method, the pseudoasbestos bodies had a deeply stained mantle, indicating an iron-rich complex. The other graphite crystals did not show this mantle (Fig. 6).

The graphite particles varied in size from 2 to 20 μ with a mean of 11 μ for single crystals; those in groups measured around 50 μ. In order to facilitate these measurements, the tissues were cut in sections 10 μ thick, and flamed until the paraffin had melted. The pseudoasbestosis bodies were a darker brown after this process. When examined under polarized light, a few doubly refractile particles were seen scattered sparsely through the sections; there were more of these in the middle zones. Fat staining did not demonstrate fat in the asteroid bodies, but accumulations of fat, the same size as the asteroid bodies, were found.

Samples taken from the middle lobes and basal segments of the upper lobes were sent to Dr. J. E. Cowle of the Industrial Hygiene Division, Ontario Department of Health. On analysis the dried lung yielded 5.39% by weight of ash, 0.32% of free and combined silica, and 0.12% of free silica. The ash yielded 5.90% by weight of free and combined silica, and 2.20% of free silica (Table II).

TABLE II.—RESULTS OF ASH ANALYSIS OF LUNG TISSUE IN GRAPHITE PNEUMONOCONIOSIS (DRY WEIGHT)

Author	Ash (%)	Total SiO_2	Free SiO_2 (%)	Ash Total SiO_2	Ash Free SiO_2 (%)
Harding and Oliver[4].... (Case 2)	5.65	1.14	0.50	—	—
Harding and Oliver[4].... (Case 3)	7.56	1.77	0.47	—	—
Jaffe[1].....	7.33	0.23	—	3.18	—
Miller and Ramsden[3]...	1.50	0.41	—	27.0	—
Present case.....	5.39	0.32	0.12	5.90	2.20

DISCUSSION

The patient described in this paper worked in the same mine as Jaffe's patient, and the ash and total silica content of dried lung from these two men were in the same range (Jaffe's case: total ash, 7.33%; total silica, 0.23%; total silica content of ash, 3.18%—Table II). Correlation between the silica content of lungs and disease is notoriously difficult. On this point it is interesting that Vorwald, Macewen and Smith[7] (Table III) found levels of silica approximating

TABLE III.—COMPARISON OF SILICA CONTENT OF LUNG TISSUE IN VARIOUS PULMONARY DUST DISEASES

Disease entity	No. of cases	Ash (%)	Silica (%) Total	Silica (%) Free
Silicosis.....	54	9.22	2.18	0.68
Siderosilicosis.....	32	11.86	1.83	1.06
Dust exposure but no disease....	24	6.26	0.29	0.06
Present case.....	1	5.39	0.32	0.12

those in the present patient in persons who, although exposed to dust, showed no visible pulmonary disease. It is my opinion that the major disease in this patient was due to the graphite dust. Some degree of fibrosis has been found in pure carbon pneumoconiosis,[1, 2, 4-7, 10, 14, 19-24] such as that produced by carbon black,[3] and in other dusts completely free from silica such as nepheline.[25] In such fibrosis it is not easy to assess accurately the contribution of such factors as physical damage to cells, lymphatic block, recurrent unresolved inflammation and vascular obliteration.

The asteroid inclusions found in the present case and in Jaffe's[1] may eventually be recognized as a feature of graphite pneumoconiosis. Asteroid inclusions are often found at sites where red cells have broken down, but the physical basis underlying their formation has not been elucidated. In Jaffe's case, the fluid which escaped from the lungs contained abundant cholesterol crystals, suggesting that there had been old bleeding into the cavities.

Though necrosis is common in the massive lesions of anthracosilicosis,[26] cavitation is probably commoner in graphite pneumoconiosis and may contribute to hemorrhage and a consequent accumulation of lipoid material.

Figs. 7 and 8 show the close similarity between the "graphite bodies" and asbestos bodies, a similarity which should be kept in mind when examining sputum.

The precise role of the atypical mycobacterium isolated in this case is hard to evaluate. This bacillus may have contributed to the cavitation, but cavitation can occur without tuberculous infection in graphite pneumoconiosis. It was perhaps only an interesting complication which made the clinical diagnosis in 1960 a little more difficult.

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