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PULMONARY ASBESTOSIS IN SOUTH AFRICA.

(With Special Plate.)

BY

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It has been known for some time that workers exposed to the dusty atmosphere arising from some processes involved in the preparation of asbestos materials suffer from pulmonary disability. The mining of the mineral itself is probably not a source of danger, as asbestos is mined mostly in open quarries. After sorting, there remains waste rock which still contains fibre in payable quantity. Crushing of this rock causes a considerable degree of dust exposure, and exposure to the dusty atmosphere is still more exaggerated in the carding and spinning of asbestos in the mills.

Very little concerning the pathological changes in the lungs of persons working with asbestos has been found in the literature, and it was thought that a record of the following cases might be of interest.

On September 2nd, 1925, the medical officer of an asbestos mine in Southern Rhodesia forwarded to the South African Institute for Medical Research three small specimens of lung from a *post-mortem* case for histological examination.

CASE I (No. 9026).

The subject was a male adult native, who had worked for twelve months in the asbestos mill, and, except for a short period before his death, had had a good record of health. For nine weeks before death he suffered from acute tuberculosis, and the *post-mortem* findings showed miliary tuberculosis involving the lungs, liver, spleen, and pericardium. The object of the histological examination was to ascertain if there was any evidence of fibrosis in the lungs which might be directly ascribed to the nature of his employment, since workers in the mill are exposed to a very dusty atmosphere.

Apart from the tuberculosis and associated fibrosis, sections of the lungs showed a certain amount of connective tissue change which had no obvious connexion with the tuberculous process. It was also found that curious golden yellow segmented structures, with rounded or club-shaped ends (Fig. 1), were embedded in this fibrous tissue, together with very minute, doubly refractile particles, the latter presumably silica. Although the curious segmented bodies were remarked upon, no further investigation was carried out at the time, and the new-formed fibrous tissue was attributed to the presence of silica.

CASE II (No. 11016).

It was not until September 29th, 1927, when a portion of lung from a second case was sent by the medical officer from the same mill, that the foreign bodies and fibrosis were more fully investigated. This piece of lung was from a male adult native who had been employed in the mill for a period of two years. About September, 1926, he was admitted to hospital suffering from pneumonia, and had a long illness. He was discharged, and later (September 19th, 1927) readmitted in a dying condition. He was very emaciated, and had the physical signs of pulmonary tuberculosis with fibrosis. At autopsy the lungs were firmly bound down by adhesions, the root glands enlarged and hard, and the lungs on section hard and fibrous with an almost leather-like consistence. The mesenteric glands were also considerably enlarged.

Histological sections showed a generalized but moderate degree of fibrous thickening of the pleura, trabeculae, and alveolar walls. In addition, there was a much more marked fibrosis arranged in irregular-shaped nodules, chiefly related to the blood vascular system and bronchi. This fibrous tissue was cellular, the elements arranged in an irregular manner, and included some small lymphocytic accumulations (Fig. 2). There was no resemblance to the orderly, whorled arrangement and sharp definition of the silicotic nodule (Fig. 3). The bronchi were the seat of slight catarrhal changes, and many of the alveoli contained "dust cells" filled with phagocyted particles. There was no evidence of tuberculosis or acute pneumonic consolidation. Embedded in the fibrous tissues, lying free in alveoli (Fig. 4), and contained in phagocytic cells, there were numerous golden yellow segmented bodies (Figs. 5 and 6), a few large angular black bodies, and very minute doubly refracting particles. Phagocytosis was a very striking feature. Large numbers of the bodies were contained within ordinary mononuclear cells and irregular-shaped multinucleated giant cells (Fig. 7). Phagocytosis was not confined to the smaller particles, but even large rods were seen completely enclosed and often bent in order to allow them to occupy the space within the cells (Fig. 7). It was thought that these foreign bodies, together with the crystalline matter, may have been the cause of the fibrosis, as the connective tissue, both in distribution and formation, was very suggestive of changes resulting from a dust occupation of the lungs. At the same time the history of pneumonia and protracted recovery must be taken into consideration, since an unresolved pneumonia with subsequent fibrosis is not an uncommon occurrence amongst natives working on the mines in South Africa.

CASES III AND IV (Nos. 6419 and 6731).

Consultation of the records and previous histological sections of material from the same mine has revealed two additional cases, both of which showed lobar pneumonia. There was very little connective tissue increase, but the unusual structures (Fig. 8) and refractile crystalline particles were present. No history of length of service in the mill was obtained with either of these cases.

Further study of the golden yellow bodies showed a variety of shapes, but the most common forms had rounded or club-shaped ends and a segmented body tapering to a finely pointed tail. They were non-refractile to polarized light, soluble in strong acids, and, on raising to a red heat, turned black and tended to lose their outline. In sections treated with hot dilute hydrochloric acid and potassium ferrocyanide they gave a well-marked Prussian blue reaction. No pigment except these structures giving the iron reaction could be detected in the sections examined. Strong hydrochloric acid, having been tested for the presence of iron and found negative, was used to dissolve out iron from fresh sections. After treating the sections the hydrochloric acid gave a very distinct red-pink colour with potassium thiocyanate, and the red-pink colour disappeared on the addition of a solution of mercuric chloride. The sections were re-examined and showed that the majority of the golden yellow bodies had been dissolved out. From these tests it was concluded that the structures contained a large percentage of iron.

As controls, sections of lungs from a large number of miners on the Rand who had died from silicosis and tuberculosis were examined. Bodies such as have been described above were found in none of these, nor was the Prussian blue reaction or other test for iron positive except in those cases where the pigment was obviously of haematogenous origin. A single case of a miner was also investigated; he had worked for twenty-eight years in the haematite mines in the North of England, and subsequently for seven and a quarter years in the gold mines of the Rand, South Africa. The cause of death was carcinoma of the gall-bladder. Macroscopically the pulmonary root glands were enlarged, pigmented, and fibrosed. The pigment was dark rust-coloured and gave a marked Prussian blue reaction with dilute hydrochloric acid and potassium ferrocyanide. Occasional rust-coloured subpleural islets were visible. On section the lungs showed a slight diffuse fibrosis, and a few large areas of fibrosis. The whole lung gave a marked iron reaction. Histological sections of the large areas showed moderately well defined, but irregularly shaped, masses of well-formed acellular fibrous tissue (Fig. 9). The alveolar walls were slightly thickened, and there was a moderate degree of connective tissue increase round the blood vessels and bronchi. A large quantity of pigment of a reddish-brown colour was contained in phagocytic cells and lying free in alveoli, alveolar walls, and between the fibres of the newly formed fibrous tissue. The greater part of this pigment gave the iron reaction. In addition, there was another variety of pigment, in much smaller quantity, in the form of crystalline refractile particles. The latter was intimately mixed with the iron-containing dust. This lung was especially examined to determine, if possible, whether the same peculiar bodies were being formed from a deposit of ferric iron dust as in the case of an asbestos dust which contained both ferrous and ferric iron. A careful and thorough search was made, but no particles with a similar appearance were detected (Fig. 10). At the time of the patient's death this case of ironstone phthisis was complicated by underground work in the gold mines on the Rand, but in 1919 it simulated, by *x*-ray photography and physical examination, an early case of silicosis,

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and confirms the findings of Sir Kenneth Goadby[1] and Dr Cronin.[2]

In addition to the human lungs showing asbestosis, Dr. Mavrogordato of the South African Institute for Medical Research has supplied me with the lungs of a guinea-pig which died in December, 1927, from causes other than asbestosis. This animal was exposed to an asbestos dust atmosphere experimentally. The length of exposure was two hours a day on each of fifty days. The first exposure took place on February 4th, 1925, and the last on April 1st of the same year. The asbestiform compound used for the experiments was a chrysotile[3] obtained from the mine in Southern Rhodesia. Histological sections showed a slight generalized fibrosis and an increase in pigment, but the interesting feature was the presence of the golden yellow bodies (Fig. 11), similar to those seen in the lungs of human pulmonary asbestosis.

A comparison between the human cases and the experimental animal showed that the fibrosis was more rapid and extensive in the human cases than in the experimental animal. This is readily explained by the difficulty in reducing the tough asbestos fibre to a uniformly fine powder, and to the presence of a comparatively small proportion of the rock dust which is usually associated with asbestiform compounds. For "dusting" animals a limited amount of dust is available, and of this only a small proportion contains particles of sufficiently small dimensions to permit of their reaching the lung alveoli. In silicosis[4] the size of the majority of the particles which reach the alveoli is between 1μ and 3μ; in the haematite lung, mentioned above, the particles appear larger, but even here are much smaller than the greater number of those prepared from asbestos for "dusting" experiments. In the working mill the conditions are very different from an asbestos dust atmosphere produced experimentally. Fine dust is continuously reaching the atmosphere, and only the very fine particles remain suspended for any length of time. These gradually increase in numbers until a maximum concentration is reached, then remain more or less constant during working hours. Thus it will be seen that, in order to produce changes in experimental animals showing the same degree of fibrosis in the same length of time, an atmosphere approximating that of the working mill will have to be obtained.

The amount of fibrosis in two of the human cases (Cases I and II) was quite definite, and, if due to the presence of asbestos dust, the initial rate of production was rapid when compared with present-day non-infective silicosis on the Rand. It is difficult to state a definite time for the production of an appreciable degree of fibrosis in pure non-infective silicosis, but modern observation tends to show that it is in the neighbourhood of ten years. In Case I a moderately marked fibrosis had taken place after one year of work in the mill, but this was complicated by tuberculosis. It is known that the rate of fibrous tissue production is very much greater in dust diseases complicated by infections, but even allowing for this the connective tissue increase in Case I was rapid. In Case II there was a still more definite fibrosis after two years of work in the mill with no evidence of tuberculosis.

Before this work was completed Drs. Cooke,[5] Stuart McDonald, and Oliver[7] published their papers dealing with pulmonary asbestosis, and previous to that a short article on the same subject appeared in the *Lancet*. The similarity between the case described and ours was suspected when the first article appeared, and our work goes far to confirm the findings. As in those recorded here, the asbestos dust responsible for the changes in the lungs in their case was a chrysotile. Whether other asbestiform compounds are capable of producing these changes it is impossible to say, as there appears to be no record of any such cases in the literature.

In the Union of South Africa[8] and Rhodesia there are many asbestos mines, from some of which chrysotile is obtained, but two other interesting varieties are found—namely, crocidolite and amosite. Amosite is a comparatively recent discovery, the chemistry of which has not been completely worked out, but it appears to be somewhat similar to crocidolite in composition. Both these compounds contain a large percentage of ferrous iron. Analysis of four samples of amosite[8] showed between 32 and 44 per cent. of FeO, and eight samples of crocidolite[8] between 16.5 and 40.5 per cent. Up to the present there has been no examination of *post-mortem* material from cases of death among those working in mills where these minerals are treated. No such material has been made available.

Chrysotile or serpentine asbestos usually contains 2 to 3 per cent. of FeO isomorphously replacing magnesia. The analysis of Dr Cooke's case showed 3 per cent. of FeO, and the Rhodesian mineral, according to Mr. A. L. Hall,[3] 2.44 per cent. Dr. McCrae of the Government Chemical Laboratory, Johannesburg, analysed the FeO content of the asbestos used by Dr. Mavrogordato in his animal experiments, and found a much lower percentage—namely, 0.45. Apparently even with very small percentages of FeO the golden yellow bodies are formed in the lungs.

With regard to the unusual structure and chemical nature of the golden yellow bodies found in the lungs in pulmonary asbestosis, until many more cases have been examined and more experimental work has been done very little can be stated. There is no doubt that they are in some way associated with the asbestos, or, probably, more particularly with the FeO content, either of the asbestos itself or of the dust of the mill. This dust contains a much higher percentage of iron, which may be derived partly from the lode from which the asbestos is mined.

Three possibilities regarding the formation of the golden yellow bodies are worthy of mention:

(1) In the form of a gel, as suggested by Dr. Stuart McDonald.

(2) As particles of ferruginous quartz formed in the lungs under conditions similar to weathering, or ferruginous quartz changed in composition as a result of combination with constituents of the body fluids. In support of this it may be mentioned that crocidolite, which is an alkali silicate with ferrous iron, is especially liable to decomposition when exposed to weathering. Sodium is removed, the iron oxidized and hydrated to form limonite, and silica set free; there then results a ferruginous quartz which possesses the finely fibrous structure of the original mineral. It is extremely hard, and coloured a rich golden yellow. It is possible that a change such as this occurs with the small quantity of ferrous iron associated with chrysotile, and the colour of the bodies in the lungs is very suggestive.

(3) Phagocytosis of these structures is a very prominent feature in the lungs, and it was thought that the action of these cells may have been responsible, first, for some change in chemical composition, then a building up and moulding into the various shapes seen.

The fibrous tissue change in the lungs of the above-mentioned cases of workers in asbestos mills is probably the result of a reaction on the parts of the tissues to both the golden yellow bodies and a small quantity of silica.

Sufficient cases of pulmonary asbestosis have not been recorded to base an opinion upon the liability to secondary infection by specific inflammatory processes such as tuberculosis, but it is very suggestive that two of those now recorded, which have been examined histologically, have shown tuberculosis complicating the changes produced by an asbestos dust occupation of the lungs.

Besides these cases there is still further evidence in favour of tuberculosis being a complicating factor.

Dr. H. M. Murray reported a fatal case in the *Charing Cross Hospital Gazette* in 1900; and in the United States of America, from one source,[9] during the period 1907 to 1914 there were 13 deaths, 3 of which were from tuberculosis.

In 1910 Dr. Collis[10] reported on the relationship of asbestos dust to pulmonary tuberculosis, and found that 5 deaths from phthisis had occurred in five years amongst a staff of less than forty workers employed at a factory where asbestos is woven.

In conclusion, I wish to record my thanks to Dr. Mavrogordato for data and material from an experimental animal; to Dr. Irvine, chairman of the Miners' Phthisis Medical Bureau, Johannesburg, for material from silicotic cases and from the lungs of a haematite miner; to Dr. McCrae for his analysis of the FeO content of a sample of asbestos; and to Mr. P. Longmore for his valuable assistance in preparing the microphotographs.

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