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PULMONARY FIBROSIS IN AN ALUMINIUM WORKER

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Aluminium dust has never been shown to be harmful to man in Great Britain. This paper reports a fatal case of progressive pulmonary fibrosis in a young man occupationally exposed to a heavy concentration of fine aluminium dust. Clinically, radiologically, and pathologically this case was indistinguishable from cases of aluminium fibrosis of the lung described by Shaver in Canada.

In Great Britain there have been no reports of pulmonary fibrosis due to particulate aluminium. On the contrary, the Medical Research Council's Industrial Pulmonary Diseases Committee in 1936 investigated 50 workers exposed to aluminium dust and were unable to find any evidence that it caused fibrosis of the lung. Hunter, Milton, Perry, and Thompson (1944) studied the health and environment of 97 duralumin propeller grinders and concluded that there was no evidence that it occasioned any disease of the trachea, bronchi, or lungs. Eloquent testimony to its innocent reputation is the fact that coalminers have been deliberately exposed to this dust (*Lancet*, 1956) with the intention of preventing pneumoconiosis. On the other hand, in Germany (Goralewski, 1947) and Canada (Shaver, 1948) cases have occurred which have been recorded in a growing literature which has been summarized by Perry (1955).

Case History

A.G. died, aged 22.*

After leaving school he worked as an agricultural labourer until he was 19. At this age, in March, 1951, he joined a factory making fine aluminium powder and worked in an atmosphere heavily contaminated with fine aluminium dust until disabled by breathlessness in December, 1953. He gave no history of previous illness except that in November, 1951, he had been admitted to hospital for 10 days for treatment of second degree burns received at work. The breathlessness became progressively worse and by March, 1954, he had to walk slowly on the flat, though he was able to sleep comfortably with one pillow. Three months later the breathlessness was such that he spent most of the day in his bedroom; bouts of morning cough, producing occasional scanty mucoid sputum, were very distressing. On the night of August 29 he became worse and the following morning was admitted to hospital desperately ill, cold, shocked, and cyanosed, in a stuporous state feebly gasping for breath. The temperature was 100·4°F., pulse 110/min., respirations 45/min. Blood pressure was 95/70 mm. Hg. He died four hours later. An hour after death I performed bronchoscopy and saw the

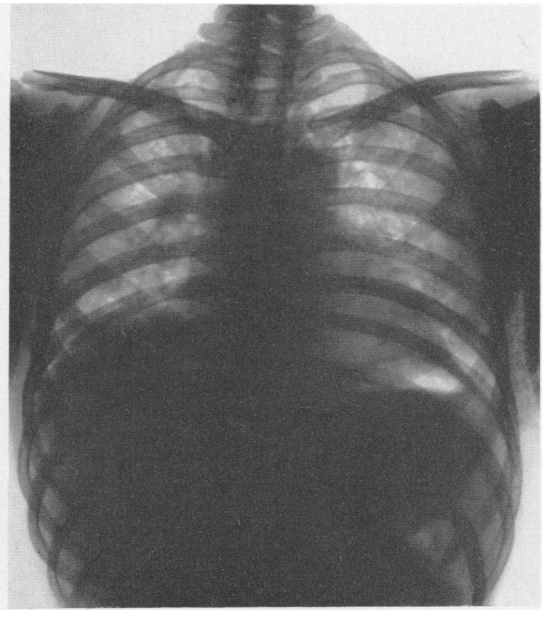

FIG. 1.—Chest radiograph in full inspiration taken on April 7, 1954. Appearances of pulmonary fibrosis: raised diaphragm (on screening respiratory excursion was greatly reduced), mediastinum drawn out, abnormal opacities most marked in the upper zones, trachea curved to the right side.

*Since submitting this report the man who replaced him at work has died of the same disease, and I have found two ambulant cases.

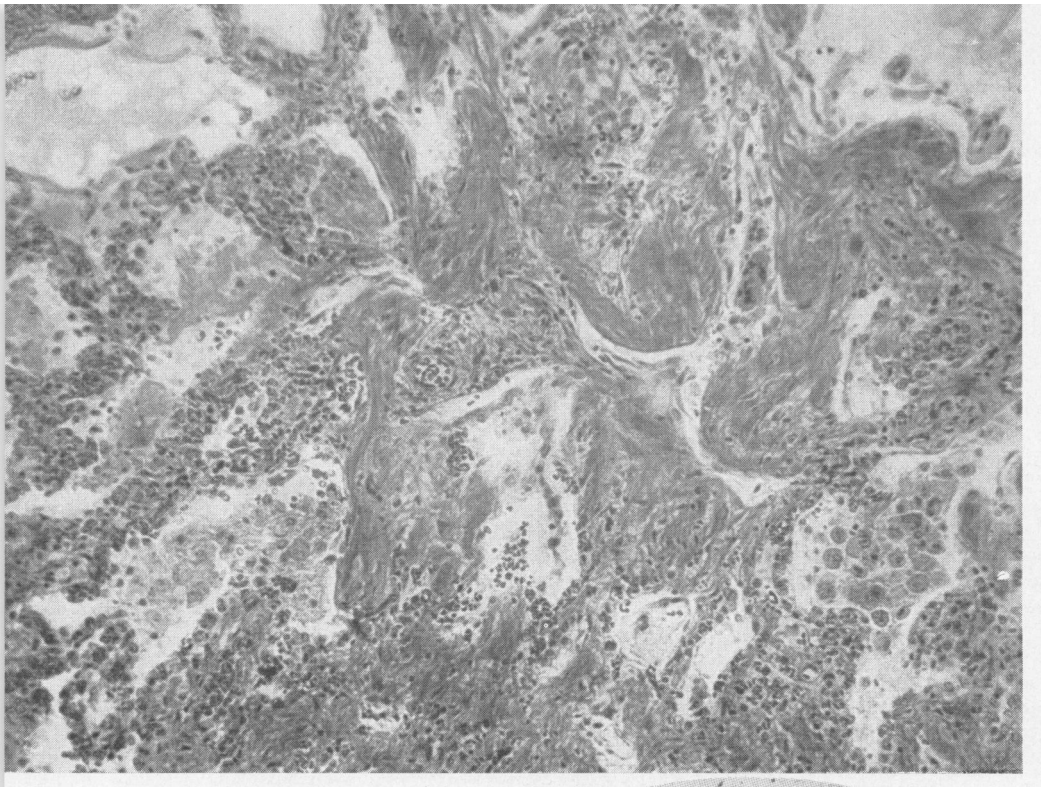

FIG. 2.—Section of lung showing fibrosis. (van Giesen) × 400.

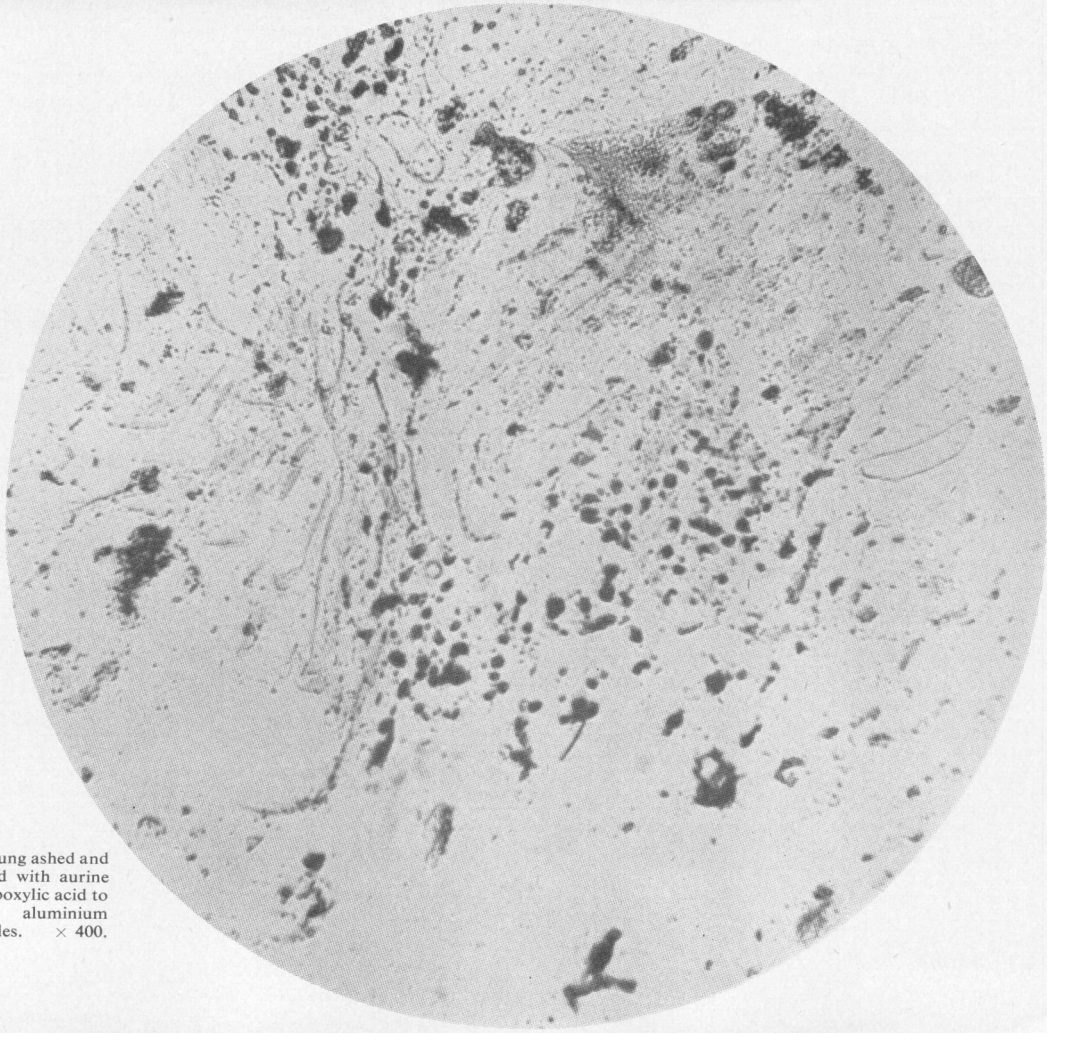

FIG. 3.—Lung ashed and stained with aurine tricarboxylic acid to show aluminium particles. × 400.

mucous membrane pale and glistening and normal throughout; no secretions were seen.

Five months before he died I admitted him to hospital for investigation and found a healthy-looking, slightly built young man. Respiratory excursion was poor, and medium crepitations were heard, mainly over the pectoral and sub-pectoral regions. The pulmonary second sound was considerably increased and palpable and there was a systolic thrust over the lower end of the sternum. The plain chest radiograph showed the appearances of pulmonary fibrosis (Fig. 1); the barium swallow looked normal. There was no albumin or sugar in the urine; the deposit looked normal under the microscope and there was no growth on culture. Nineteen specimens of sputum were examined for acid-fast bacilli, with negative results. There were no fungi found in the sputum. The total and differential white cell count was normal on two occasions and the erythrocyte sedimentation rate normal on three occasions. The plasma proteins were normal. The tuberculin test was negative to the 1/100 solution. The Wassermann and Kahn tests were negative. A patch test was carried out, applying the aluminium powder from the factory to his skin for 48 hours, with negative result. He was thought to be suffering from pulmonary fibrosis and cor pulmonale.

The possibility of aluminium fibrosis was considered and the nature of his work more fully investigated. He was engaged, with two other men, on an eight-hour shift rota to attend heavy stamping machines which produced finely divided aluminium powder. His work involved exposure to this dust in varying concentrations, over approximately half of his working day. The range of particle size of this powder is large but the quantity of dust in the atmosphere below 5 μ is of the order of 10 mg. per c.m. In order to facilitate the process the dust is mixed with a small quantity of stearine which may form up to 0·5% of the total weight. This forms a fine film of aluminium stearate on the surface of the particle. It is of interest to note that during the second world war when cases of aluminium fibrosis occurred in Germany they were attributed to the absence of stearine due to war-time shortage. The two other men working on the plant, as well as several others doing similar work on a coarser type of powder, were examined and chest films of the other workers taken. None of them showed any evidence of lung fibrosis.

Pathology

Post-mortem examination showed all the organs, except the lungs and heart, to be normal to the naked eye and on microscopy. The lungs, which were prepared by the Gough technique, were lightly adherent to the chest wall. They were shrunken and on section showed marked generalized fibrosis and interstitial emphysema; the fibrosis was particularly marked in the upper lobes. The heart weighed 16 oz. (450 g.), the enlargement being mainly of the right ventricle. Microscopical examination of the lungs showed the fibrosis to be non-nodular (Fig. 2); numerous jagged particles were lying in the lung tissue (Fig. 3) and these gave histochemical reactions for aluminium. A frozen section showed some very small particles, which stained for lipoid, in the phagocytes in some of the alveoli; no other lipoid crystals or globules could be found. Chemical examination of the lung for aluminium gave the following results: root, 227 parts per million of wet lung estimated as aluminium; apex, 15 parts per million; base, a trace. The average content calculated on dry weight was 640 parts per million estimated as Al_2O_3.

Discussion

Pulmonary fibrosis of gradual onset in a previously fit young man, in whom tuberculosis, syphilis, sarcoidosis, malignant infiltration, lipoid pneumonia, the reticuloses, and the Hamman Rich syndrome can be excluded, is unique in my experience. The occupational disease reported by Shaver followed a clinical, radiological, and morbid anatomical and histological pattern that was characteristic. Wyatt and Riddell (1949) stated that the parenchymal fibrosis of the lungs was unlike anything previously encountered. The cases described by Goralewski followed a similar pattern. The patient described in this paper, except that he never had a spontaneous pneumothorax, bears a close resemblance clinically, radiologically, and pathologically to these cases. I suggest that the pulmonary fibrosis was due to a susceptible subject inhaling high concentrations of fine aluminium dust.

I gratefully acknowledge essential help from Dr. Peter Davies who showed the case to Dr. Shaver and his associates during his N.A.P.T. scholarship tour in Canada; from Dr. George Manning, who did the necropsy, histological and chemical examination, and from Professor R. E. Lane for the occupational history, particulars of dust exposure, and for his advice and encouragement.

References

Goralewski, G. (1947). *Z. ges. inn. Med.*, **2**, 665.
Hunter, D., Milton, R., Perry, K. M. A., and Thompson, D. R. (1944). *Brit. J. industr. Med.*, **1**, 159.
(1956). *Lancet*, **2**, 501 [leading article].
Perry, K. M. A. (1955). *Ibid.*, **2**, 463.
Shaver, C. G. (1948). *Occup. Med.*, **5**, 718.
Wyatt, J. P., and Riddell, A. C. R. (1949). *Amer. J. Path.*, **25**, 447.