A CASE OF FARMER'S LUNG

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FARMER'S LUNG WAS FIRST REPORTED in 1932 (Campbell) but only in recent years has it become more widely recognized. It is thought probable that this is the most fully investigated patient to be so far described.

The patient, born on 7 April 1905, was unmarried and lived with his parents, brother and sister. All worked hard on the farm under the somewhat tyrannical direction of the father.

Family history. The father is a senile diabetic and both he and his wife suffer from severe osteoarthritis. The brother and two sisters are all fit. No member of the family has had any respiratory illness nor been affected in any way by mouldy hay.

The environment. The family lived in a small gritstone farmhouse on the edge of a moor and situated over 1,000 feet above sea level. Much of the land tends to be marshy, some fields consisting largely of reeds. Summers are short. Slow growth means a comparatively late hay harvest. Hay is stored in an ill-ventilated barn some distance from the farm and is brought up for winter feeding.

A 1959 sample of the hay was typical of that usually associated with farmer's lung (table I) (Gregory and Lacey 1963). It had undergone spontaneous heating and become mouldy during maturation.

TABLE I

EXAMINATION OF SAMPLE OF MOULDY HAY
(Carried out at Rothamsted Experimental Station)

	Moulds	*Actinomycetes spores*	*Humicola lanuginosa*	*Bacteria*
Visual examination Millions/g. dry weight hay	16.6	78.3	2.87	
From culture Thousands/g. dry weight hay	4.67	24.7		2.28

Notes: pH. 7.5
Aspergillus and penicillium spores in abundance
Actinomycetes isolated at 24° were abundant
Humicola stellata was isolated

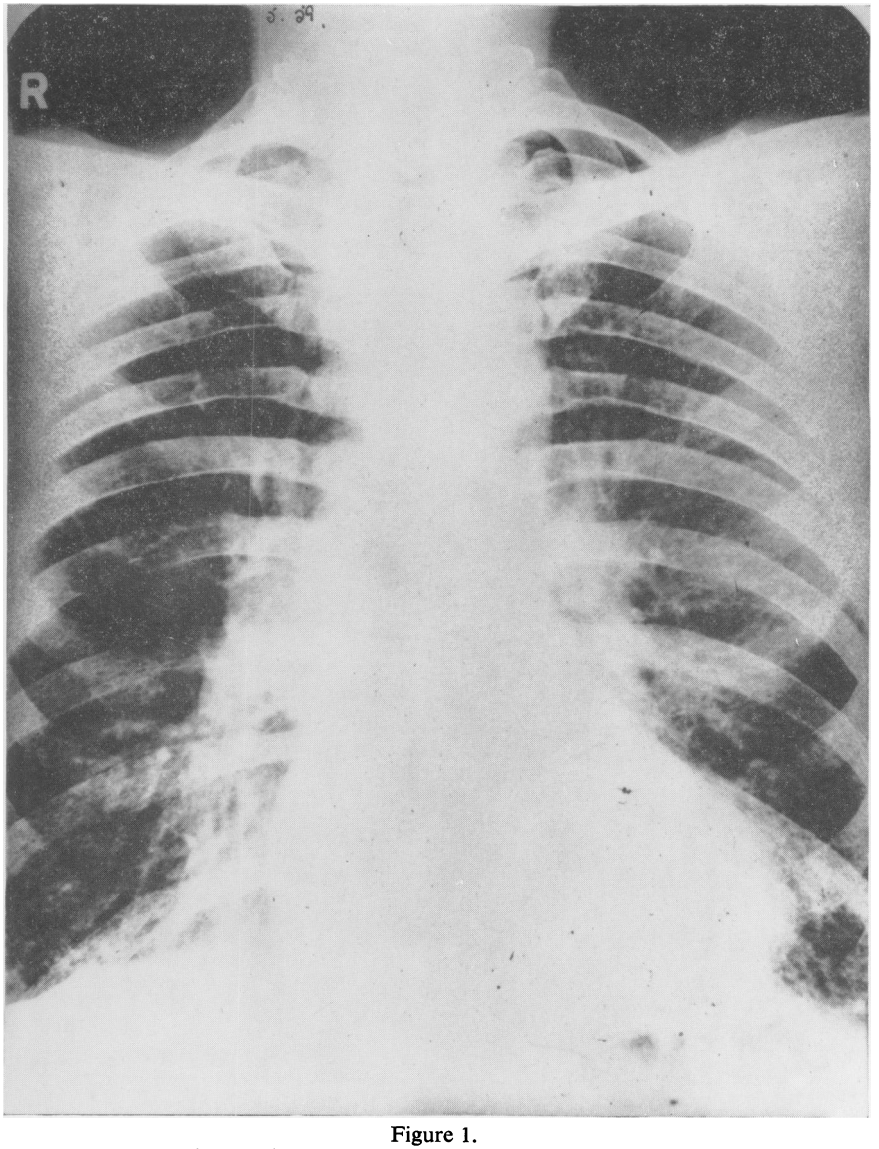

Figure 1.
Chest radiograph showing the extensive fine mottling

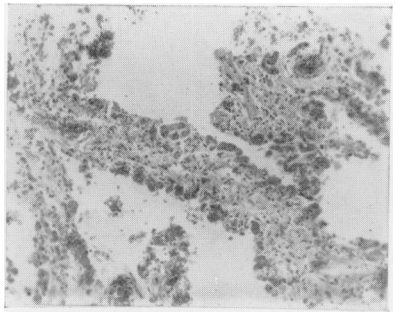

Figure 2.
Lung Section. Dilated capillaries on the surface of the thickened fibrotic alveolar wall (H. and E. × 150)

Figure 3.
Lung Section. Laminated concretions showing calcification giant cell reaction and exudation of lymphocytes. (H. and E. × 250)

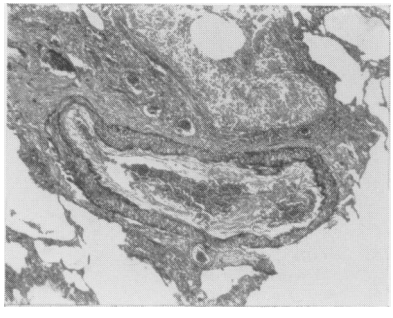

Figure 4.
Lung Section. Focal collection of laminated concretions with surrounding lymphocytes. (H. and E. × 250)

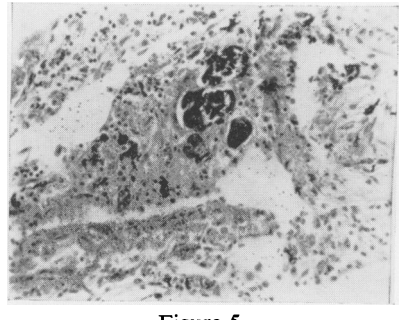

Figure 5.
Lung Section. Focal collection of calcified concretions with surrounding lymphocytes. (H. and E. × 250)

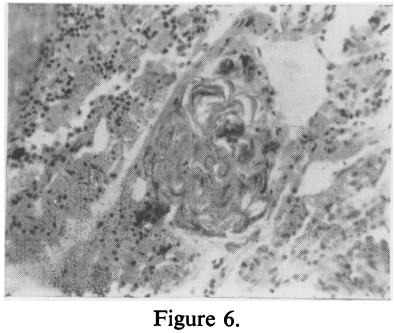

Figure 6.
Lung Section. ' Muscular pulmonary artery ' showing medial hypertrophy and organized recanalized thrombus. (Elastic/Van Gieson × 60)

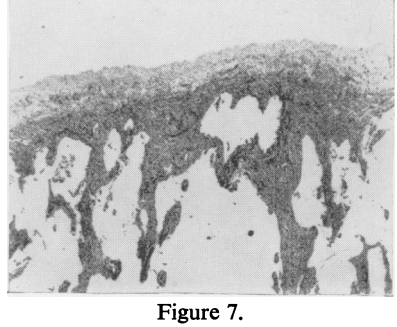

Figure 7.
Lung Section. Pulmonary arteriole showing distinct muscular media with internal and external elastic laminae indicating grade 1 hypertensive pulmonary vascular disease. (Elastic/Van Gieson × 250)

History and course of illness

The first significant illness occurred in 1938 when the patient was aged 33 years. He complained of cough, breathlessness and swelling of the ankles, which was thought to be of cardiac origin. In March 1954 an influenzal illness was followed by a persistent cough, breathlessness, headache, general stiffness and precordial pain on exertion. There was no significant cardiorespiratory abnormality but this illness kept him off farm work for seven months.

The following March a pyrexial illness occurred with cough, dyspnoea on exertion and cyanosis. Bilateral basal crepitations appeared for two months. An x-ray of the chest at that time was not reported as showing signs of farmer's lung. Early in 1956, 1957 and 1958 there were similar recurrences. February 1959, however, saw the onset of the most severe attack so far. Crepitations were present at the left base but extended over the whole of the right lower and midzones. An x-ray showed extensive fine mottling in both lung fields consistent with farmer's lung (figure 1). The ESR was 5 mm./hour, white cell count 6,100, polymorphs 67 per cent, lymphocytes 31 per cent, monocytes 2 per cent. This illness persisted until October.

In November 1959 the patient was deliberately exposed to mouldy hay and the peak expiratory flow rate measured (table II). This showed a reduction after exposure. By the evening the patient was dyspnoeic with a tight chest and coughing a little white tenacious sputum. These symptoms were more severe than would be expected with this degree of fall in peak flow rate.

TABLE II

PEAK EXPIRATORY FLOW RATE (WRIGHT) IN LITRES PER MINUTE
(Average of five readings)

Hay exposure	*Before*	*After*
Good hay	420 (Predicted normal range for patient 350-550)	470
Mouldy hay	410	360

The usual symptoms recurred in February 1960, two hours after handling mouldy hay. By March, when he was admitted to the Queen Elizabeth Hospital, Birmingham, he had marked signs of congestive cardiac failure. There was poor air entry in the upper zones, scattered rhonchi in the right lung and crepitations at the right axilla. Full investigation of pulmonary function was carried out (see below) after treating the heart failure. Partial recovery was followed by slow deterioration and the patient died in October 1963.

During the final illness the Pneumoconiosis Medical Panel recognized this as an industrial disease and assessed disability at 70 per cent from November 1959.

Investigations

These were carried out during March 1960.

The white cell count was 7,800, including 0.5 per cent eosinophils only.

Chest x-ray showed diffuse reticulation. Mantoux and Kveim tests were negative as also were skin tests to cereals, aspergillus, grasses, pollen and mixed moulds. Pulmonary function results are shown in tables III and IV. The vital capacity was virtually normal. The residual volume was greater than predicted but the total lung capacity was normal. The ventilatory function results showed no evidence of significant airway outlook and keep up to date with new ideas and progressive techniques obstruction, as is usual in farmer's lung (Bishop, Melnick, and Raine 1963). As expected the diffusing capacity was considerably reduced and out of proportion to the total lung capacity. The ventilation-perfusion relationships suggest that blood flow was distributed to the alveoli less evenly than normal. On cardiac catheterization a severe pulmonary hypertension was found during exercise. Arterial hypoxaemia may have contributed to this rise (Bishop, Melnick, and Raine 1963). The pulmonary wedge pressure was normal. The cardiac output response was normal at rest and slightly reduced on exercise.

TABLE III

PULMONARY FUNCTION

		Percentage of the predicted normal value for the patient
Vital capacity	3.90 litres	*88*
Residual volume	3.30 litres	*130*
Total lung capacity	7.20 litres	*103*
Maximum breathing capacity ..	81 l/min	*57*
Forced expiratory volume (predicted normal range at 1.0 seconds (F.E.V. 1.0)	2.39–4.47	
F.E.V. 1.0 as percentage of forced vital capacity	55	
Maximum mid expiratory flow rate ..	44 l/min	
Diffusing capacity for carbon monoxide (rest)	8.8 ml/min/mm Hg	*41*
Diffusing capacity for oxygen (exercise)	16 ml/min/mm Hg	*38*
		Predicted normal
Difference in oxygen tension between alveolar gas and arterial blood (breathing air)	19.6 mm Hg	11.2
Physiological dead space as percentage of tidal volume	59.1 per cent	27.5

A serological test in 1961 showed that precipitations were present against extracts of good and mouldy hay.

TABLE IV
PULMONARY FUNCTION—CARDIAC CATHETERIZATION RESULTS

	Rest	*Exercise*
Oxygen uptake ml/min/m^2	157	520
Cardiac index l/min/m^2	3.19	5.40
Mean pressures mm/Hg		
Pulmonary wedge	3	5
Pulmonary arterial	28	68
Arterial haemoglobin oxygen saturation	88.7 per cent	69.1 per cent

Autopsy

There were no emphysematous bullae on the surface of the voluminous lungs. Section showed diffuse panacinar emphysema and the cut surface was firm suggesting fine fibrosis. There were no focal lesions and the bronchi were normal. The heart was grossly enlarged, mainly due to hypertrophy of the right ventricle.

Histological examination showed:

1. Widespread capillary dilatation causing prominent bulging into the alveolar spaces (figure 2).

2. Excess of ferric iron salts due to capillary bleeding and resulting in
   (i) Incrustation of elastic laminae of pulmonary blood vessels with iron salts.
   (ii) Small laminated concretions of iron salts around some of which were granulomatous reactions with foreign body giant cells and epithelioid cells, also focal collections of lymphocytes (figures 3, 4 and 5).

3. The elastic pulmonary arteries (1,000 μ in external diameter) showed atherosclerosis.

4. The muscular arteries (100–1,000 μ) showed increased media thickness with slight intimal fibrosis (some pulmonary arteries showed organized thrombi but there were no ' dilatation lesions ' nor evidence of necrotizing arteritis) (figure 6).

5. The arterioles (100 μ) showed muscularization of the media and slight intimal fibrosis (figure 7).

6. The pulmonary veins showed slight intimal fibrosis.

7. The bronchial arteries were normal.

8. There was some fibrosis of the alveolar walls, the spaces were dilated, the appearance being that of panacinar emphysema. There was swelling of the alveolar pneumocytes forming intra-alveolar macrophages, many containing haemosiderin.

Discussion

This presented many of the typical features of farmer's lung. He, like five others with the disease, lived in that part of this practice adjoining the moors. In contrast, no case has occurred in the lower areas. Once the condition was diagnosed it became clear that previous attacks of chest trouble were due to the same cause. No explanation can be given for the

ankle oedema at the apparent onset of the disease. Certainly this could hardly be heart failure so early in the course. The symptoms recurred with remarkable consistency during February when foddering is at its height. Exposure to mouldy hay demonstrated the typically delayed response.

The histological report was interesting. The granulomatous lesion is well recognized but the cause of the capillary dilatation is obscure. It is tempting to suggest that recurrent local hyperaemia with repeated exposure might be the cause. This would agree with one of Bishop's hypotheses, namely, that farmer's lung may be an Arthus reaction involving the lung parenchyma. Against this theory is lack of recent exposure to mouldy hay when he died in October.

Summary

A case of farmer's lung is described with particular reference to the histological examination of the lungs.

Acknowledgements

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