Thorax 1986;**41**:68–69

Farmer's lung after *Mycoplasma pneumoniae* infection

CLIVE McGAVIN

From Plymouth Chest Clinic, Plymouth

Farmer's lung is a hypersensitivity reaction to fungal spores in mouldy hay, most commonly *Micropolyspora faeni*. The type of immunological reaction is debated[1] but a high dose of antigen is necessary and the inflammation may result from activation of the alternative pathway of complement, linked to a type 3 response. Detection of serum precipitins to fungal antigens is used as a diagnostic aid but it is unclear why up to 18% of men with sufficient exposure to antigen and with the serum precipitins do not develop symptoms.[2] Two Cornish men are described who had long term exposure to dusty hay but who developed symptoms of farmer's lung only after infection with *Mycoplasma pneumoniae*.

Case reports

CASE 1

A 31 year old non-smoking farmer presented in January 1979 with an acute febrile illness associated with cough, sputum, headache, and extensive lung crackles. He had had no symptoms of farmer's lung before 1979 despite years of handling hay. At presentation his chest radiograph showed bilateral consolidation and the titre of antibody to *Myco pneumoniae* in the complement fixation test[3] was 1/320. After one month, during which he had avoided hay and taken tetracycline, he was still unwell and breathless. The *Myco pneumoniae* antibody titre in the complement fixation test had risen to 1/1280 and the precipitin test for *M faeni* measured by a double diffusion technique[4] gave a strongly positive result. He was admitted to hospital and treated with prednisolone and erythromycin. He improved over a fortnight and was discharged from hospital. One month after discharge, while still taking prednisolone 10 mg daily, he spent a day working in a barn in which the winter's hay had been stored. That evening he developed fever, breathlessness, and headache and was readmitted with a diagnosis of acute farmer's lung. Again he improved gradually with increased doses of corticosteroids. By August 1979 the drugs had been withdrawn, the chest radiograph was normal, but the single breath carbon monoxide transfer factor (TLCO) was 76% predicted. He has since avoided contact with hay and remained symptom free. In April 1981 a complement fixation test for *Myco pneumoniae* showed a titre of only 1/40 but the result of the *M faeni* precipitin test remained strongly positive.

CASE 2

This 40 year old farmer had also handled hay for many years with impunity; in particular, the harvest of 1980 had produced dusty hay but he did not develop symptoms of hypersensitivity in the following winter. In December 1981 he developed pneumonia. The *Myco pneumoniae* complement fixation test gave a positive result with a titre of 1/1280 and the result of the precipitin test against *M faeni* was strongly positive. Despite treatment with tetracycline and corticosteroids and advice to avoid hay he remained breathless for several months. In May 1982 his *Myco pneumoniae* antibody titre had fallen to 1/80. By May 1983 he had recovered fully with no residual lung crackles and his carbon monoxide transfer factor (TLCO) was 70% of the predicted value. In December he had a brief exposure to dusty hay while opening a new rick containing mouldy bales. That night he developed symptoms of acute farmer's lung. When seen eight weeks later he was still breathless with extensive lung crackles. The *Myco pneumoniae* antibody titre in the complement fixation test was 1/160 and precipitins against *M faeni* remained strongly positive. Again he made a slow recovery over several months.

Discussion

These cases have features in common. Neither man had symptoms of farmer's lung before the infection with *Myco pneumoniae* despite years of handling hay; it is reasonable to suggest that the precipitins against *M faeni* were present before the mycoplasma infection because they remained strongly positive subsequently while the *Myco pneumoniae* antibody titre in the complement fixation test waned. Both men had acute respiratory illnesses with clinical and serological features of *Myco pneumoniae* infection but, interestingly, the delayed recovery over several months was typical of neither mycoplasma infection nor acute farmer's lung. Both men developed acute respiratory illnesses when exposed to hay dust after the mycoplasma infections; these illnesses more closely resembled acute farmer's lung than pulmonary mycotoxicosis[5] in that both men had precipitins to *M faeni* and neither had had a heavy exposure to spores in a confined space such as a silo.

In 1975 Davies *et al*[6] reported the development of IgG antibodies against *M faeni* in two patients with severe *Myco pneumoniae* infection without exposure to mouldy hay. They described a polysaccharide surface antigen common to the *Mycoplasma* and the *Micropolyspora* spores. Possibly the presence of anti-*Mycoplasma* antibodies in the two farmers caused an enhanced reaction to fungal spores by the activation of the alternative pathway of complement. It is also possible that the mycoplasma infection produced a non-specific increase in levels of complement and acute phase proteins in the lung, and hence a non-specifically enhanced reaction to subsequent inhalation of fungal antigen.

I acknowledge the advice of Dr Alex Ferguson and Dr Brian Davies and the secretarial assistance of Miss Joan Mitchell.

Address for reprint requests: Dr Clive McGavin, Plymouth Chest Clinic, Beaumont Park, Plymouth PL4 9BQ.

Accepted 30 August 1985

References

1 Emanuel DA, Kryda MJ. Farmer's lung disease. *Clin Rev Allergy* 1983;1:509–32.
2 Pepys J, Jenkins PA. Precipitin (FLH) tests in farmer's lung. *Thorax* 1965;20:21–35.
3 Bradstreet CMP, Taylor CED. Technique of complement fixation test applicable to the diagnosis of virus disease. *Monthly Bulletin of the Ministry of Health and PHLS* 1962;21:96.
4 Mackenzie DWR, Proctor AGJ, Philpot CM. *Basic serodiagnostic methods for disease caused by fungi and actinomycetes*. London: HMSO, 1980. (PHLS monograph No 12.)
5 Emanuel DA, Wenzel FJ, Lawton BR. Pulmonary mycotoxicosis. *Chest* 1975;67:293–7.
6 Davies BH, Edwards JH, Seaton A. Cross-reacting antibodies to *Micropolyspora faeni* in *Mycoplasma pneumoniae* infection. *Clin Allergy* 1975;5:217–24.

Book notices

Clinical Tests of Respiratory Function. GJ Gibson. (Pp 334; £30.) Macmillan Press. 1984.

Physicians interested in the better practice of modern respiratory medicine now have a plethora of textbooks from which to make a selection for their departmental or hospital libraries or for the more immediately accessible personal bookshelf. *Clinical Tests of Respiratory Function* is a welcome addition, with its greater emphasis on clinical application and lung function complements, without displacing the more laboratory based text of John Cotes. The author correctly observes that tests of respiratory function are not fully integrated into clinical medicine. The requirements for thoracic medicine published in *Thorax* (1984;39:400) recommended that inpatients and outpatients should have ready access to a respiratory physiology laboratory staffed by trained technicians. Such a book as this will be invaluable to those doctors concerned in establishing these departments. The text, after a brief description of the apparatus, is divided into three main sections describing, firstly, the theoretical and practical aspects of the commonly applied tests of mechanical and gas exchange function, respiratory control and exercise; secondly, functional alterations in conditions traditionally encompassed by the respiratory physician, such as airway, alveolar, and pleural diseases; and, thirdly, respiratory impairment in diseases of other systems, such as renal, hepatic, metabolic, and connective tissue disorders. I believe that this book will be invaluable to respiratory physicians with either a mainstream or a more peripheral interest in the practical application of clinical respiratory physiology. The text merits better quality paper and printing, a message which might usefully be conveyed to the publisher in time for the second edition.—RMC

Esophageal Disorders—Pathophysiology and Therapy. Tom R DeMeester, David B Skinner. (Pp 655; $13.) Raven Press. 1985.

This is a multiauthor publication appearing some two years after the Second International Symposium on Oesophageal Disorders held in Chicago in 1983. The papers are generally well written but the presentation of data, the tables, and the graphics are at times of poor construction and fail to give credit to the fine text. The main topics covered in this book include oesophageal physiology, in particular a definitive histological study of nerve and muscle from James Christensen; radionuclide imaging; reflux and its complications; carcinoma; and the functional oesophageal disorders. There is little emphasis on oesophageal varices and atresia. Nearly one third of this 655 page book is devoted to 24 hour pH monitoring. There is far too much overlap, with a grand total of 28 chapters adding little to the previously published works of Johnson and DeMeester. Although this section suffers in particular from the time lag between conference and publication, for those wishing to undertake pH monitoring it is a comprehensive and valuable guide to the techniques and equipment available.—IPA

Respiratory Care. Edited by George G Burton and John E Hodgkin. 2nd ed. (Pp 1071; £47.) JB Lippincott Company. 1984.

Fifty four authors, most medically qualified, contribute to this comprehensive and readable textbook for American respiratory care technicians. Section 1 deals with the evolution of that profession, its training programmes, competency assurance procedures, ethics, and relationships with other professions. The chapter on legal implications of respiratory care has a six page glossary of legal terms, and there is a chapter on contemporary issues in health ethics. Section 2, on the rational basis of respiratory therapy techniques, includes conventional accounts of lung physiology, lung function testing, clinical examination, and interpretation of chest radiographs, and highly technical chapters on medical gases (manufacture, storage, transport) and aerosols (28 equations on the relevant physics). The account of oxygen therapy does not mention its eupnogenic effect. Therapeutics have a transatlantic slant—for example "The basic drug in managing chronic asthma is theophylline." Intermittent positive pressure breathing is discussed sceptically and admitted to be "the ideal placebo." Section 3 deals with respiratory care in critical illness. The book is too long and detailed for medical students, nurses, and physiotherapists, and too superficial in its clinical aspects for chest doctors. In short, it is an excellent book aimed at a readership which does not really exist in the United Kingdom.—CS