

have reported values of 25-hydroxycholecalciferol of over 749 nmol/l (300 ng/ml) in vitamin D intoxication, and the value in the present case was even greater. It is not clear how much calciferol was being ingested, but the lack of response of the hypercalcaemia to treatment with prednisolone could have been due to consumption of a very high dose.

¹ Asher, R, *Lancet*, 1951, 1, 339.

² Addison, G M, *et al*, *Journal of Endocrinology*, 1971, 49, 521.

³ Preece, M A, *et al*, *Clinica Chimica Acta*, 1974, 54, 235.

⁴ O'Riordan, J L H, *et al*, *Clinical Endocrinology*, 1972, 1, 149.

⁵ Preece, M A, *et al*, *Quarterly Journal of Medicine*, 1975, 44, 575.

Department of Metabolism and Endocrinology, The London Hospital Medical College, London E1

P E BELCHETZ, MSc, MRCP, lecturer (present address: MRC Clinical Research Centre, Northwick Park Hospital, Harrow, Middlesex)
R D COHEN, MD, FRCP, professor of metabolic medicine

The Medical Unit, The Middlesex Hospital, London W1

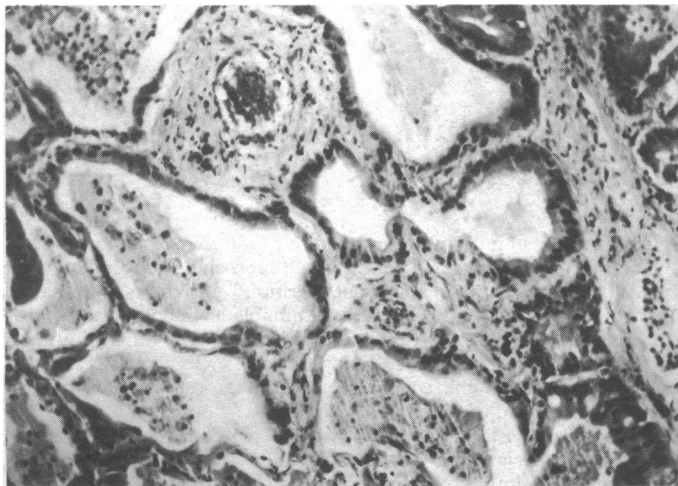
J L H O'RIORDAN, DM, FRCP, deputy director
S TOMLINSON, MB, MRCP, MRC clinical research fellow

Aflatoxin inhalation and alveolar cell carcinoma

Aflatoxin, a toxic metabolite of the mould *Aspergillus flavus*, is one of the most potent carcinogens known.¹ Epidemiological studies in tropical areas have shown that the high incidence of primary liver cancer in their populations may be related to the ingestion of contaminated food.^{2,3} This paper reports a case of pulmonary adenomatosis due possibly to inhalation of aflatoxin.

Case report

A chemical engineer aged 68 worked for three months on a method of sterilising Brazilian peanutmeal which was contaminated by the mould *Aspergillus flavus*. Three months after finishing this work he became ill with high fever and began to expectorate thick, white sputum. X-ray examination showed cavitation in the left lower lobe of the lung. At first the process was considered to be due to tuberculosis, and later to mycotic disease. After two months further lesions developed in both lungs. The condition of the patient became worse and he died 11 months after the onset of his illness. Necropsy showed enlarged, heavy lungs diffusely infiltrated with firm yellow-white or reddish lesions.



Section of lung. Interalveolar septa well preserved. Alveoli lined with high cylindrical or cubical epithelium. (Haematoxylin and eosin. $\times 95$.)

Histological examination (see fig) showed bands of fibrous tissue in the parenchyma. The alveoli were lined with high cylindrical or cubical epithelium with giant multinucleated cells, some containing mucous vacuoles in their plasma. The interalveolar septa were well preserved. Mitotic figures were rare. The picture was that of pulmonary adenomatosis. No metastases or tumours in other organs were found. Bacteriological examination was negative. A sample of lung tissue was taken for chemical investigation. Thin-layer chromatography of the extract showed a blue fluorescent spot in 365 nm UV light similar to that of a commercial sample of aflatoxin B₁ (Calbiochem, California), the same colour change as standard B₁ when treated with 50% H₂SO₄, and an RF value identical to that of the commercial aflatoxin sample B₁. A colleague of this patient who had been doing the same work had died three years before of pulmonary adenomatosis, but no chemical investigations were done in his case.

Discussion

The pathogenesis and aetiology of primary pulmonary adenomatosis is not clear. It often develops at the site of fibrous scars of inflammatory origin or in a lung chronically damaged from other causes. Eversole and Rienhoff⁴ suggested that it is due to congenital malformations, while Spencer⁵ thought it was caused by the inhalation of irritant substances together with other factors that lead to chronic damage to the lung. It seems that men who inhale toxic mould during their work might well be at risk.

¹ Butler, W H, Greenblatt, M, and Lijinsky, M, *Cancer Research*, 1969, 29, 2206.

² Hutt, A M, Wogan, G N, and Davidson, C S, *Gastroenterology*, 1972, 62, 1094.

³ Peers, F G, Linsell, C A, *British Journal of Cancer*, 1973, 27, 473.

⁴ Eversole, S L, and Rienhoff, W F, *Journal of Thoracic and Cardiovascular Surgery*, 1959, 37, 750.

⁵ Spencer, H, *Pathology of the Lung*, p 660. London, Pergamon Press, 1963.

Department of Pathology, Charles University, Hradec Králové, Czechoslovakia
IVANA DVOŘÁČKOVÁ, MD

Pulmonary asbestosis and autoimmunity

Experimental animals exposed to silica particles form rheumatoid factor.¹ Rheumatoid factor and antinuclear antibodies have also been found in persons exposed to asbestos and in patients with pulmonary asbestosis.² Autoimmunity might therefore be implicated in causing the pulmonary lesions of asbestosis. The inhaled asbestos fibres would cause cellular damage, tissue antigens would be released, and autoimmune antibodies formed. Furthermore, HLA-27 antigen, known to be associated with diseases of possible autoimmune aetiology,³ is often present in patients with asbestosis.

Antophyllite, a relatively rare kind of asbestos, is mined in Finland. The incidence of pulmonary tuberculosis and of pulmonary carcinoma is high,⁴ and they occur not only in the factory workers but also in people living close to the mines.⁵ If autoimmune mechanisms played a part in the pathogenesis of their disease we would expect to find rheumatoid factor or antinuclear antibodies in their sera. We therefore examined the sera of all the quarry and fibre mill workers for the presence of rheumatoid factor and antinuclear antibodies.

Methods and results

In all, 66 sera were studied from men who had been working with asbestos for 2 to 38 years, the average being 15.2 years. None had severe asbestosis. We used the Waaler-Rose test for the rheumatoid factor and the fluorescent antibody test for antinuclear antibodies, as used routinely in our laboratory. Both positive and negative control sera were included in all test series.

A Waaler-Rose titre $>1/32$ was found in seven (10.7%) and a titre of $>1/64$ in three (4.5%) of the 66 sera. Antinuclear antibodies were seen in only one serum. The titre with FITC conjugated antihuman immunoglobulin was 1/80 and with antihuman IgG 1/10. These findings are the same as those in the normal healthy population.