Hypersensitivity pneumonitis in a mouldy house

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ABSTRACT: Two cases of hypersensitivity pneumonitis, one confirmed by histopathology, are described. The patients were members of different families and developed the disease as occupants, at separate times, of the same inner-city dwelling. We believe the disorder resulted from exposure to thermophilic microorganisms prevalent in their domestic environment. Both patients recovered after moving from this place of residence. The need for greater awareness of this variety of hypersensitivity pneumonitis is stressed.

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HYPERSENSITIVITY pneumonitis may occur after exposure to a wide variety of organic antigens from sources that include mouldy hay, mouldy bagasse, different kinds of birds, grain and silage, and, less frequently, other occupational exposure.[1] More recently, a mini-epidemic of domiciliary hypersensitivity pneumonitis has been described in a family exposed to the organism *Bacillus subtilis* in decaying wood dust released from a bathroom during remodelling[2] and hypersensitivity pneumonitis has also been diagnosed in people living in old, damp and mouldy inner-city houses.[3] We report two further cases occurring in different families occupying the same dwelling at different times in order to emphasise the need for greater awareness of the domiciliary form of hypersensitivity pneumonitis. Otherwise this may be diagnosed wrongly as an infective respiratory illness.

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Case 1

A 23-year-old non-smoking housewife sought treatment in November, 1979, after she had suffered a non-productive cough, dyspnoea and fatigue for six weeks. Three months previously, she had moved to the ground floor of an old, damp and dusty inner-city dwelling.

On examination, the patient was afebrile, in respiratory distress, and tachypnoeic at rest. There was reduced chest expansion. On chest auscultation, fine bibasilar crepitations which did not clear with coughing were audible. The results of spirometry were a forced expiratory volume in one second (FEV_1) of 0.9 L (predicted FEV_1, 2.2 L) and a forced vital capacity (FVC) of 1.1 L (predicted FVC, 2.8 L). There was moderate reduction in residual volume (0.87 L; predicted 1.14 L) and total lung capacity (2.01 L; predicted, 4.03 L).

Analysis of arterial blood gases showed hypoxaemia and alveolar hyperventilation (PaO_2 9.46 kPa; $PaCO_2$, 4.27 kPa; pH 7.45). The patient's lung diffusing capacity for carbon monoxide could not be measured due to her respiratory distress and inability to hold her breath. Chest X-ray examination showed bilateral fine mottling (Figure 1a). Serum precipitin tests for aspergillus and thermophilic organisms, by double diffusion using commercial antigens (Hollister Stier), returned negative results.

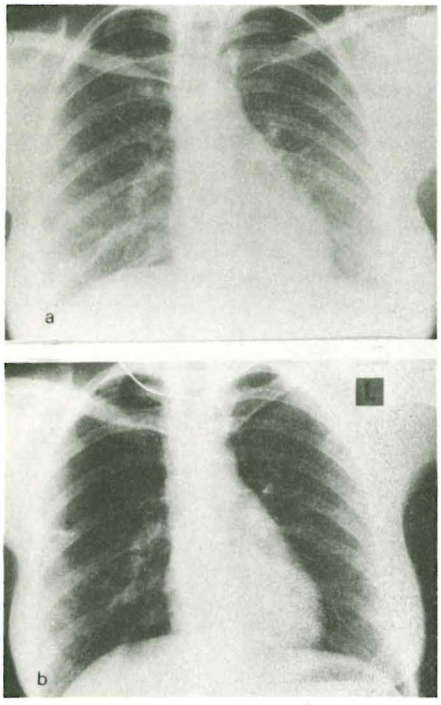

FIGURE 1: Chest X-ray films of Patient 1: (a) at the time of presentation (above); and (b) five weeks later (below) when symptoms had resolved. There has been complete resolution of the bilateral infiltrates.

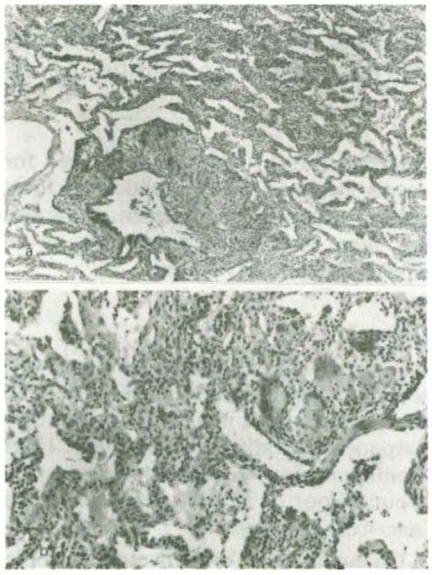

FIGURE 2: Histopathological findings in the lung biopsy taken from Patient 1. Above (a): There is a pronounced, diffuse inflammatory reaction involving the alveolar walls. A non-caseating giant-cell granulomatous reaction is prominent around a bronchiole. The cellular nature of the reaction in the interstitium is evident (haematoxylin-eosin stain; magnification x 25). Below (b): The expansion of the alveolar walls by oedema and cellular infiltrate, the non-caseating granulomas with giant cells and the lack of fibrosis are shown at this higher magnification. Cells are predominantly lymphocytes and macrophages (haematoxylin-eosin stain; magnification x 68).

The patient underwent an open lung biopsy after transbronchial biopsy through a fibreoptic bronchoscope did not help in arriving at a diagnosis. Histologically, the lung biopsy specimen was compatible with acute hypersensitivity pneumonitis with marked lymphocytic, plasma-cell, and mononuclear-cell infiltration (Figure 2a) and numerous non-caseating granulomas within the interstitial spaces (Figure 2b). The patient made a complete recovery after seven days of corticosteroid therapy and a change of abode. This move had been planned before the patient became ill and was not made on medical advice. Spirometric values of the patient two months later included a FEV_1 of 2.5 L and FVC of 2.75 L. Chest X-ray examination results were normal (Figure 1b).

Case 2

A 6-year-old girl, with presenting symptoms of dyspnoea, loss of energy, anorexia and 2-kg weight loss of 10 weeks' duration, attended for treatment in January 1982. Eleven months before, she and her parents had moved to the house formerly occupied by the patient in Case 1.

On examination, the child weighed 19 kg. She had difficulty in breathing and there were signs of subcostal and intercostal recession, tachypnoea, reduced chest

expansion, and tachycardia. There were no crepitations audible on chest auscultation; minimal exercise in the ward caused obvious cyanosis of the patient. Spirometric values were an FEV_1 of 0.5 L (predicted, 1.1 L) and FVC of 0.55 L (predicted, 1.4 L). The patient showed signs of hypoxaemia and alveolar hyperventilation at rest (PaO_2, 8.53 kPa; $PaCO_2$, 4 kPa; pH 7.44). Chest X-ray examination showed bilateral haziness (Figure 3).

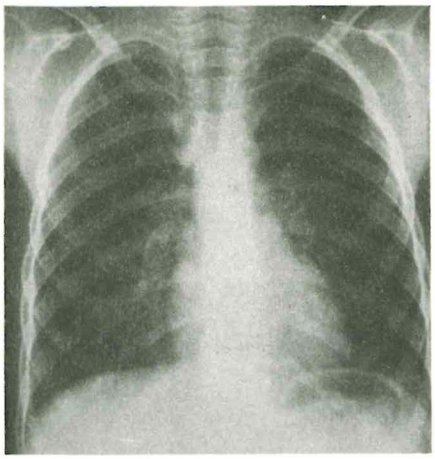

FIGURE 3: Chest X-ray film of patient 2 at the time of presentation showing diffuse bilateral infiltrates.

When it became apparent that the two patients had shared a common residence, investigations were carried out there. Abundant quantities of dust and mould were found behind torn wallpapers and beneath worn carpets. Fungi of the *Aspergillus, Penicillium* and *Cladosporium* species were isolated from samples obtained from the walls and floor, but thermophilic actinomycetes were not isolated. No attempt was made to extract antigens from the samples for purposes of skin testing, bronchoprovocation or specific serum precipitin estimation. Testing for serum precipitins for aspergillus and thermophilic organisms, using commercially prepared antigens, gave negative results.

The child made a spontaneous recovery in hospital. Two weeks after admission chest X-ray examination results were normal. Spirometric values at this time included FEV_1 of 1.0 L and FVC of 1.4 L. Analysis of arterial blood gases showed PaO_2, 11.6 kPa; $PaCO_2$, of 4.67 kPa; and pH 7.38. The family moved from the offending environment and the child remains well.

Discussion

The clinical presentations, radiological and pulmonary function abnormalities, and subsequent course of both patients are consistent with the diagnosis of hypersensitivity pneumonitis. In addition, histological findings confirmed the diagnosis in one patient. Since both patients shared a common address, although at different times, and since both have remained well after a move from the house, it is reasonable to suggest that antigens in their common domestic environment were responsible for their illnesses. The house was old and damp with abundant quantities of dust and mould behind wallpapers and under carpets. Although species of common fungi were grown on culture of the samples obtained from the house, it was not possible to identify the causative agent(s) from these limited studies. Testing for serum precipitins with commercial preparations of aspergillus and thermophilic antigens gave no results in either patient. However, in a previous study,[2] exhaustive tests with carefully prepared extracts of suspected material did not produce serum precipitin responses in affected household members, even in the presence of positive skin and bronchoprovocation responses to these materials. Thus, failure to obtain a serum precipitin reaction with commercial antigen preparations in the two patients described here does not rule out the diagnosis of hypersensitivity pneumonitis due to common domestic antigen exposure. Further mycological identification and antigen extraction for serum precipitin estimation, bronchoprovocation and other studies should be undertaken if necessary to establish the diagnosis and if technically feasible.[4,5] In the cases reported here, these tests were not deemed necessary for diagnosis or management.

As with other forms of the disease, the prevalence of hypersensitivity pneumonitis due to domiciliary exposure to organic dusts is not known. Association of the disease with the use of recirculating airconditioning units and home humidifiers is well recognised,[6,7] but it has been suggested recently that domiciliary exposure under other circumstances, such as exposure to moulds proliferating on damp walls and floorboards is an increasingly frequent cause of disease in inner-city dwellers.[3] The cases described here support this suggestion. Since the symptoms of hypersensitivity pneumonitis are not specific and may lead to mistaken diagnosis of respiratory infection, and since removal of the patient from the offending environment is frequently necessary for effective treatment, awareness of this form of the disease is needed to ensure accurate diagnosis and proper management of the patient and other family members who may have unsuspected disease.

Acknowledgement

Mrs J. Peate assisted in the preparation of the manuscript.

Addendum

The owners of the house were informed of the health hazard, and subsequently renovated the dwelling. To our knowledge, there have been no further outbreaks of illness in residents of the house.

References

1. Karr RM, Salvaggio JE. Infiltrative hypersensitivity disease of the lung. In: Parker CW, ed. Clinical Immunology. Philadelphia: WB Saunders, 1980: 1336-1350.
2. Johnson CL, Bernstein IL, Gallagher JS *et al.* Familial hypersensitivity pneumonitis induced by *Bacillus subtilis*. *Am Rev Respir Dis* 1980; 122: 339-348.
3. Lee LH. Hypersensitivity pneumonitis. *Aust NZ J Med* 1981; 11: 299-301.
4. DoPico GA, Reddan WG, Chmelik F *et al.* The value of precipitating antibodies in screening for hypersensitivity pneumonitis. *Am Rev Respir Dis* 1976; 113: 451-455.
5. Fink JN. The use of bronchoprovocation in the diagnosis of hypersensitivity pneumonitis. *J Allergy Clin Immunol* 1980; 6: 590-591.
6. Tourville DR, Weiss WI, Wertlake PT *et al.* Hypersensitivity pneumonitis due to contamination of home humidifier. *J Allergy Clin Immunol* 1971; 49: 245-251.
7. Fink JN, Banaszak EF, Baroriak JJ *et al.* Interstitial lung disease due to contamination of forced air systems. *Ann Intern Med* 1976; 84: 406-413.

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