

Occupational asthma due to hexachlorophene

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Low molecular weight chemicals may induce occupational asthma by acting as haptens.^{1,2} Sterilising agents of low molecular weight containing halogens have been reported to cause occupational asthma.³ Here we report a case of occupational asthma due to inhaled hexachlorophene (molecular weight 406.9), which is used in medical practice as a powder or solution for topical disinfection. In the patient we describe asthmatic symptoms were provoked by hexachlorophene powder and the asthmatic attacks were reproduced by inhalation challenge. To obtain further information about the pathogenesis of the reaction we measured serum neutrophil chemotactic activity before and after challenge.

Case report and investigations

The patient was a 43 year old children's nurse who had been working in contact with hexachlorophene for 15 years. She had had respiratory symptoms for about 10 years. Initially her symptoms were of rhinitis but for the last two years she had had frequent attacks of asthma, which were sometimes very severe. She had noticed that her symptoms of chest tightness and breathlessness began some minutes after starting work with hexachlorophene and that she was free of symptoms at weekends and while on holiday. She smoked four cigarettes daily. She was non-atopic as judged by negative skinprick test responses to common allergens and a total serum IgE level of 73 U/ml as measured by the PRIST technique (Phadebas). Skin testing with a 1% solution of hexachlorophene also produced a negative result; the blood eosinophil count was normal. The results of routine blood tests and the chest radiograph were normal. Non-specific bronchial reactivity to five breaths of acetylcholine (100 µg/ml) inhaled from an ultrasonic nebuliser was shown to be increased.

Inhalation challenge tests using the technique of Pepys and Hutchcroft⁴ were performed, airways resistance being measured in a constant volume whole body plethysmograph. On three separate occasions the patient inhaled undiluted hexachlorophene powder for 20 minutes and on a subsequent occasion the test was repeated 20 minutes after inhalation of 40 mg sodium cromoglycate. On other days we assessed the spontaneous diurnal variation in airway resistance⁵ and, as a control, an inhalation challenge was performed with lactose in double blind fashion. We also performed an inhalation challenge with hexachlorophene in an asthmatic who had not previously been exposed.

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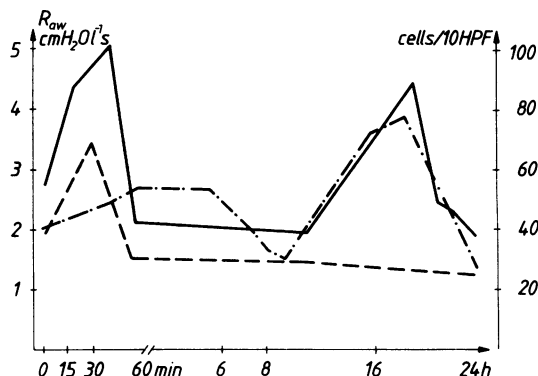


Fig 1 Changes in airway resistance after hexachlorophene challenge (—) and control challenge (---). The broken line (- - -) shows the profile of neutrophil chemotactic activity after hexachlorophene challenge. Raw—airways resistance; HPF—high power fields.

For the measurement of serum neutrophil chemotactic activity⁶ a modified Boyden chamber⁷ with two compartments separated by nitrocellulose filter (pore size 8 µm) was used, and after incubation for 90 minutes the filters were removed and stained by a standard technique. The cells were counted on the lower surface of the filter in 10 microscopic fields chosen at random.

Figure 1 shows a representative example of the changes in airways resistance after hexachlorophene and control

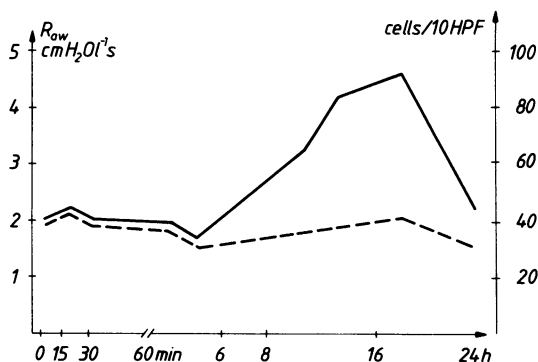


Fig 2 Effect of pretreatment with sodium cromoglycate on airway resistance (—) and neutrophil chemotactic activity (- - -) after hexachlorophene challenge. Abbreviations as in figure 1.

challenges, together with changes in neutrophil chemotactic activity after hexachlorophene challenge. On the control day there was the expected rise in airways resistance in the early morning and this was similar on the hexachlorophene day. In addition, after exposure to hexachlorophene, but not to lactose, there was an immediate asthmatic reaction, which mimicked the subjects' usual symptoms and was reproducible on the different study days. The rise in airways resistance was mirrored by a corresponding rise in neutrophil chemotactic activity. After pretreatment with sodium cromoglycate (fig 2) the immediate rise in both airways resistance and neutrophil chemotactic activity was abolished. The late rise in airways resistance was similar to that on the control days and was attributable to spontaneous diurnal variation.

The non-exposed asthmatic subject we studied had a similar degree of hyperreactivity to acetylcholine as our patient and did not react to bronchial challenge with hexachlorophene.

Discussion

We have described occupational asthma due to hexachlorophene in a children's nurse. This agent is widely used as a topical disinfectant but other similarly exposed nurses had no significant respiratory symptoms. In such cases it is important to take account of circadian variation in airway function,⁵ which accounted for the apparent late reaction in our case. The immediate airway reaction was blocked by pretreatment with sodium cromoglycate and this, together with the demonstrated increase in neutrophil chemotactic activity, which was also blocked by sodium cromoglycate, suggests that the mechanism may be mast cell dependent. One other asthmatic subject showed no evidence of asthma

provoked by hexachlorophene when similarly challenged. We do not, however, have any information on neutrophil chemotactic activity in non-exposed asthmatic subjects after challenge or in non-asthmatic normal subjects regularly exposed to hexachlorophene.

Hexachlorophene is in frequent use in hospitals as an antiseptic in both lotion and powder form. The powder has the theoretical advantage of achieving a similar antiseptic effect with less skin absorption but the findings in the patient described here suggest that this needs to be balanced against its possible role as a cause of occupational asthma.

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References

- ¹ Pepys J. Occupational asthma: an overview. *J Occup Med* 1982; **24**:534-8.
- ² Butcher BT. Pulmonary reactions to inhaled low molecular weight chemicals. *Eur J Respir Dis* 1982; **63**, suppl 123:13-6.
- ³ Bourne MS, Flindt MLH, Miles-Walker J. Asthma due to industrial use of chloramine. *Br Med J* 1979; **iii**:10-2.
- ⁴ Pepys J, Hutchcroft BJ. Bronchial provocation tests in etiologic diagnosis and analysis of asthma. *Am Rev Respir Dis* 1975; **112**:829-40.
- ⁵ Burge PS. Single and serial measurements of lung function in the diagnosis of occupational asthma. *Eur J Respir Dis* 1982; **63**, suppl 123:47-59.
- ⁶ Nagy L, Tak LH, Kay AB. Neutrophil chemotactic activity in antigen induced late asthmatic reaction. *N Engl J Med* 1982; **306**:499-501.
- ⁷ Kay AB. Studies on eosinophil leucocyte migration. *Clin Exp Immunol* 1978; **61**:54-62.