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Occupational asthma due to indirect exposure to lauryl dimethyl benzyl ammonium chloride used in a floor cleaner

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Abstract

The case is described of a 44 year old pharmacist who developed severe occupational asthma threatening his continued employment, confirmed by serial measurement of peak expiratory flow at home and work. The cause was found to be the cleaning agent used in his office when it was unoccupied. Bronchial challenge testing identified the specific agent to which he was sensitised as lauryl dimethyl benzyl ammonium chloride, a constituent of the floor cleaner. Substitution of this floor cleaner by a simple detergent cleaner led to a substantial improvement in his asthma, confirmed by repeated serial peak flow measurements.

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Several cleaning agents and biocides have been described as causes of occupational asthma including chlorhexidine, glutaraldehyde, formaldehyde, isothiozolinones, and chloramine T.5 All cases described so far have been in subjects using these chemicals. It is possible that the protein denaturing properties of these chemicals is the method by which sensitisation occurs, creating neoantigens from human proteins; their chemical dissimilarity would favour such a mechanism.

Case report

A 44 year old hospital pharmacist had his first attack of asthma at the age of 22 following a spill of chloroxylenol on the floor. The attack was associated with cyanosis and lasted a few hours. He noticed that symptoms returned on contact with trichlorophenol. His asthma went into remission until one year after moving to a new pharmacy containing a cleanroom manufacturing area. It became progressively worse over the next six years requiring several periods off work. He noticed that it became more severe as the working week progressed and improved at weekends and holidays. Thursdays were often his worst day, despite working similar hours on Fridays. It would start noticeably on the second working day after about three hours at work and was associated with running eyes and aching in

the limbs with a sensation of fever, although his temperature remained normal. Symptoms would often wake him from sleep. He had smoked 20 cigarettes daily between the ages of 18 and 27. In his teens he had been a county standard long distance runner.

Examination was normal on a day away from work. Spirometric and gas transfer measurements were all above those predicted in the clinic. Skin prick tests to common environmental allergens were negative, total IgE was $<25\,\mathrm{kU/l}$, and total IgA was reduced at $0.7\,\mathrm{g/l}$. Histamine reactivity on a day away from work was increased at $0.9\,\mu\mathrm{mol}$ (normal >8) by the Yan method. Two hourly recording of peak expiratory flow at home and work showed clear occupational asthma (first two weeks of fig 1).

A cause was sought in his pharmacy. His work was largely in his office with some visits to the wards. The building was air conditioned but the humidifier was not used. There was no handling of bulk ispaghula or enzyme preparations. The floor of the whole pharmacy was cleaned (usually on Wednesdays) with Vantropol, a cleaner containing two biocides. Substitution of this cleaner with a general detergent, initially unknown to him, resulted in substantial improvement in his asthma (second two weeks of fig 1). He was then admitted for bronchial provocation testing, staying in an individual room the floor of which was cleaned with plain water only. Vantropol contains two biocides, one of which is lauryl dimethyl benzyl ammonium chloride, as well as two colourants, a perfume, and two non-ionic surfactants. His-

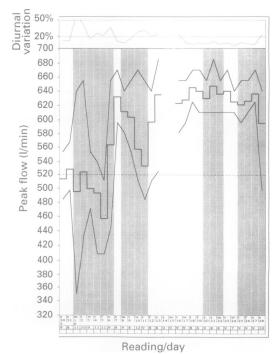


Figure 1 Plots of two hourly peak flows at home and work using the OASYS plotter. The top panel shows durnal variation in peak flow. The centre panel shows daily maximum (top line), daily mean (middle line), and daily minimum (bottom line). Days at work have a shaded background, days away a clear background. At the bottom is the date and number of readings per day. The first section shows the record when the floor cleaner contained lauryl dimethyl benzyl ammonium chloride.

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> tamine reactivity was 6.5 µmol on admission after a period away from exposure. On separate days challenges were carried out by putting 1 ml of a 1:400 dilution in water of each ingredient onto a gauze in front of him for one minute. There was no reaction following exposure to the detergent cleaner. Exposure to Vantropol caused a small reaction at 2-3 hours, followed by chills, wheeze, and breathlessness which were maximal nine hours after the challenge (fig 2). This reaction was reproduced the next day with exposure to lauryl dimethyl benzyl ammonium chloride alone. There was a significant late asthmatic reaction starting at seven hours, with a fall in FEV₁ from 4·1 to 2·3 litres. On the final day all the other constituents including the second biocide were mixed. No reaction was seen following this challenge. Subsequent return to work, with use only of the detergent cleaner in the pharmacy, led to substantial

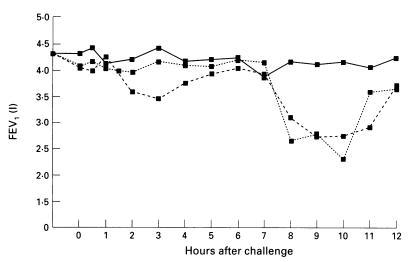


Figure 2 Bronchial provocation testing with one minute exposures to Vantropol, the commercial floor cleaner (----) and its two constituent biocides: lauryl dimethyl benzyl ammonium chloride (.....) and polymeric biguanide hydrochloride including the surfactants, colourants and perfume (-). A reproducible late asthmatic reaction was seen following exposure to the complete cleaner and to lauryl dimethyl benzyl ammonium chloride, but no reaction to the other constituents.

resolution of symptoms. Five months later histamine reactivity was >8 µmol, but one year later it had fallen to 4 µmol. This was associated with the re-emergence of minor symptoms which appeared to be work related.

Discussion

Benzylkonium chloride is the name given to a group of biocides with the general formula alkyl dimethyl benzyl ammonium chloride. The biocide in this case contained the lauryl derivative. There has been one previous report of occupational asthma from exposure to benzylkonium chloride in a laundry worker6 with a dual reaction following challenge which was blocked by sodium cromoglycate. The same material has been implicated in the transient wheeze experienced after nebulisation of ipratropium bromide (Atrovent) from multidose vials.78 The exposure in our patient was indirect as he never handled the substance himself, suggesting that his exposure was very low. This, together with the near normal histamine reactivity before challenge, the very small exposure given, and the pronounced late asthmatic reaction seen, suggests that the mechanism was one of hypersensitivity rather than irritation, although the reactions in asthmatic subjects following nebulisation are usually more characteristic of an irritant mechanism.

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