

CASE REPORT

Occupational asthma and the paper recycling industry

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Background	Occupational disease linked to the paper recycling industry has not been well documented. No previously confirmed formal diagnosis of occupational asthma (OA) caused by hydroxylamine has been made.
Methods	We have assessed and performed occupational assessment of eight workers involved in this industry. Two of these were later diagnosed with OA and are reported here.
Results	Both workers developed their respiratory symptoms within 2 years of the first use of the chemical hydroxylamine as part of the 'de-inking' process. Hydroxylamine was used as a substitute for glutaraldehyde on risk grounds, although no prior cases of OA had been found. The two workers had worked at the same plant for 11 and 20 years, respectively. Both gave histories of work-related wheeze, shortness of breath and cough. Both cases performed OASYS peak flow records over a 3-week period and had OASYS II index of 2.85 and 2.67, respectively. Both were redeployed on site to non-exposed areas and subsequently demonstrated improvement in bronchial reactivity. Case 2 subsequently consented to and underwent a blinded, placebo-controlled occupational challenge using hydroxylamine demonstrating a significant isolated late asthmatic response.
Conclusion	We believe that these are the first two confirmed cases of OA caused by hydroxylamine in the paper recycling industry.
Key words	Asthma; glutaraldehyde; hydroxylamine; occupational asthma; paper recycling.

Introduction

Paper recycling is a developing and environmentally needed process. The process is complex and involves the use of a wide array of chemical processes and at stages a veritable 'soup' of paper waste materials, added and extracted compounds and possibly intermediates. One component of this process is the need to chemically remove the ink from waste paper, before proceeding to the production of recycled paper (de-inking).

We report on two cases of occupational asthma (OA) developing in workers exposed in this industry, who first presented shortly after a change of industrial process, to switch from the use of glutaraldehyde as part of the de-inking process by substitution with hydroxylamine [1] (in approximately 2000).

Both cases were investigated by use of spirometry and histamine challenges. By use of a spirometer, the patients' baseline lung function and any subsequent changes were recorded, giving us a good indication of their progress. Histamine challenges involved incremental doses of histamine until bronchoconstriction caused a 20% decrease in the FEV₁. The result is given as a PC20 (provoked concentration) value, the lower the value, the more reactive the airways.

Case 1

In 1991, Mr X (age 30 years) started to work in the paper recycling industry. Initially, he worked in the boiler house where he stated that he had no chemical exposure. In 1993, Mr X changed jobs within the company to work in the actual paper mill as a shift technician where he worked on breakdown maintenance. On this job, he worked in the pulping and pressing areas where he used a filter mask (chemical vapour cartridge) as protection from mixed chemical and fume exposure. In 1998, he changed jobs again and worked as an instrument technician mainly in the de-inking department.

Mr X had no previous history of allergies, asthma or chest problems until 5 years ago (2002) when his wife noticed him

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wheezing in his sleep and also after coming home from work. His wheezing gradually worsened requiring time off work and treatment with antibiotics and steroids intermittently. He reported his symptoms through occupational health and was advised to avoid the relevant areas and subsequently his symptoms improved. Up until 2005, Mr X was well controlled on low-dose inhaled steroids; however, during that month, he suffered from an asthma attack at work and he was subsequently admitted to hospital. This attack prompted his referral for assessment. At his first consultation, he had mildly obstructive spirometry and a histamine challenge demonstrating moderate to severe bronchial hyperactivity with a PC20 value of 1.25 mg/ml. Occupational peak flow records were undertaken and were analysed with the OASYS II package, resulting in an OASYS index of 2.85 (threshold index of 2.5), suggesting OA. Mr X's job was subsequently modified and he avoided all exposure to the de-inking process where hydroxylamine was in use. This led to both a symptomatic and bronchial reactivity improvement of nearly two-doubling doses (4.5 mg/ml histamine), without a change in maintenance asthma therapy.

Case 2

Mr Y was a hired electrician when he joined the paper recycling mill in 1980 (aged 31) initially as a contractor before being employed full time (1983). Throughout his employment, he worked in many areas but concentrated his time in the de-inking area.

Mr Y is a lifelong non-smoker and had never had problems with hay fever, asthma or eczema. In 2003, he suffered frequent respiratory problems leading to his general practitioner prescribing four courses of antibiotics within

13 months and the initiation of low-dose inhaled steroids. At the end of 2003, Mr Y changed roles (for non-health reasons) within the plant and coincidentally noticed improvement in his symptoms and need for antibiotics (once in 18 months). He had one short period of re-exposure that resulted in a significant exacerbation of his asthma requiring 2 weeks sickness absence. After 18 months, he had continued on low-dose inhaled steroids and was largely asymptomatic. He continued to be required to return to the de-inking area and repeatedly noticed short periods of deterioration.

At the first assessment, Mr Y had a relatively normal lung function with some mild airways reactivity to histamine (PC20: 6 mg/ml histamine). Subsequent OASYS from his occupational peak flows gave an OASYS index of 2.67 which was suggestive of a diagnosis of OA.

Mr Y consented to a blinded placebo-controlled-specific occupational challenge. The control solution was inactivated glutaraldehyde; the active exposure was with hydroxylamine. The challenge was performed as follows:

- Day 1—baseline FEV₁ hourly and histamine challenge;
- Day 2—placebo challenge 5 min painting;
- Day 3—low-dose challenge hydroxylamine 5 min painting;
- Day 4—aerosolization of hydroxylamine to challenge chamber exposure 3 min;
- Day 5—histamine challenge.

The FEV₁ response to each challenge is shown in Figure 1. The challenge produced a 16% (compared to placebo) late asthmatic response maximum at 6 h. Bronchial hyperactivity to histamine did not change from pre-challenge to Day 4 (first day post-high-dose challenge).

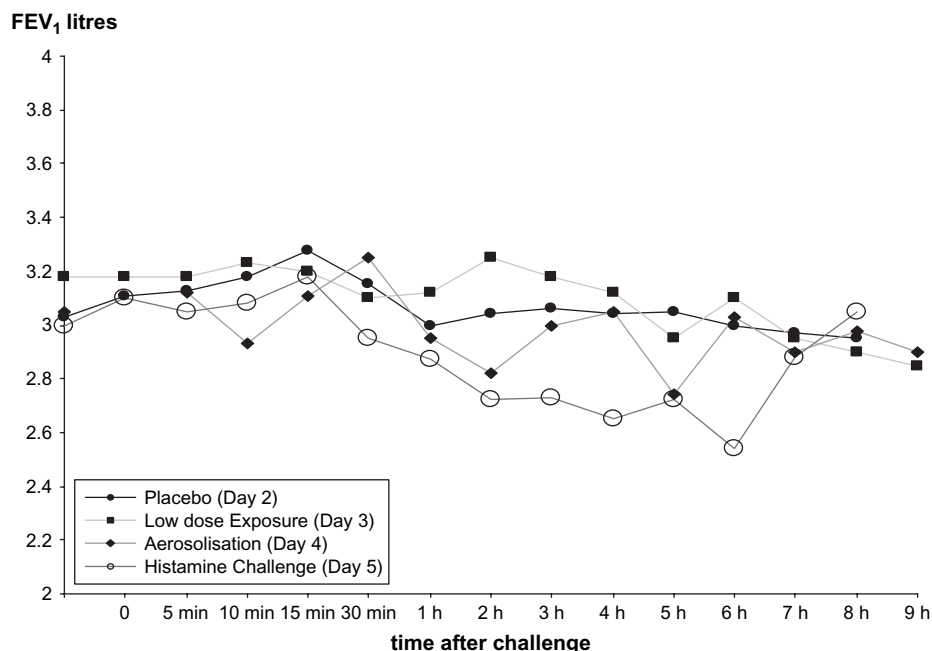


Figure 1. Mr Y's occupational challenge.

The occupational challenge resulted in a symptomatic deterioration that lasted for 2–3 weeks and required oral prednisolone to settle.

Discussion

These cases represent a cautionary tale as the index cases, followed on from the replacement of glutaraldehyde with hydroxylamine. This decision was made following risk assessments based on the literature of the sensitizing potential of glutaraldehyde, seen largely within the medical setting. No prior cases of occupational disease had been witnessed at the plant despite its paper recycling activity for ≥ 20 years prior to the switch from glutaraldehyde to hydroxylamine. Since the first two cases were found, one of two further refer-

als from the same industry site has also been provisionally diagnosed with OA.

OA to hydroxylamine has only been poorly described previously and we believe that this is the first case report, involving well-documented evidence of OA occurring as a result of exposure in the paper recycling industry.

Conflicts of interest

None declared.

Reference

1. *Summary Record Commission Working Group on the Classification and Labelling of Dangerous Substances, Meeting on Sensitizers*. Ispra, Italy: ECB, 1994; 8–9.