CASE REPORT

Occupational asthma due to heated polypropylene

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ABSTRACT: A 35 year-old nonatopic woman was referred to the hospital for possible work-related asthma. She had worked as an operator, at a plant producing polypropylene bags, for the previous four yrs. Her main complaint was a productive cough with dyspnoea and wheezing, as well as rhinitis over the past 3 yrs. She had been absent from work for 6 months on maternity leave, and had improved greatly. She was on a beta,-adrenergic agent and had to take it at least four times daily.

Baseline spirometry whilst at work showed marked airflow obstruction (forced expiratory volume in one second (FEV1) of 43% predicted (pred). After two months away from work FEV1 improved to 89% pred; provocative concentration of histamine causing a 25% fall in FEV1 (PC20) was 3.6 mg·ml-1 (mild airway hyperresponsiveness). Return to work resulted in a marked deterioration in FEV1, and serial peak expiratory flow (PEFR) values. PC20 was 0.11 mg·ml-1 (severe airway hyperresponsiveness) one week after she had returned to work. Specific inhalation challenges with polypropylene heated to 250°C resulted in a late asthmatic reaction. As formaldehyde is one of the degradation products of heating polypropylene, we exposed her to it for up to 2 h, but we elicited no bronchospastic reaction.

We conclude that heated polypropylene should be listed as one of the agents that cause occupational asthma. Eur Respir J., 1994, 7, 415–417.

Occupational history

A 35 year old woman had been employed at a company that manufactures bags, since the age of 15 yrs. She was exposed primarily to cotton and flax. Over the last 4 yrs, she had been an operator in a different department, working on a machine that transforms polypropylene into threads used in the production of bags, through an extrusion process following melting of pellets. Polypropylene, the chemical formula of which is (CH3)2-CH-[CH2-CH (CH3)]n-CH=CH-CH3, is heated to 250°C in the process. According to information obtained from a chemist, heating polypropylene to this temperature results in the release of several degradation products including: aliphatic hydrocarbons (ethylene, butene); aldehydes (principally formaldehyde); and ketones.

Medical history

The subject had no previous history of respiratory problems. She had stopped smoking 10 yrs ago. One year after being transferred to the department in which polypropylene was heated, she started experiencing progressive symptoms of productive cough, dyspnoea and wheezing, which were worse at work and improved when she was away from work. These symptoms deteriorated to such an extent that she experienced nocturnal awakenings due to asthma on a regular basis and had to visit emergency rooms on two occasions because of it. A year and a half before being seen, she left work for 6 months on maternity leave, and her asthmatic condition improved greatly, although she still required inhaled beta,-adrenergic agent on a p.r.n. basis (not daily). Her return to work caused a worsening in her asthmatic condition. She had no personal or familial atopic history (infantile eczema, urticaria, seasonal or perennial rhinitis, migraine). Skin tests performed with a battery of 15 common inhalants were all negative, with a positive control to histamine phosphate (1 mg·ml-1).

Assessment of spirometry, bronchial responsiveness and peak expiratory flow rates

When she was first seen, at a time when she was still at work, spirometry carried out according to accepted standards [1], showed marked airflow obstruction, with a forced expiratory volume in one second (FEV1) value of 1.3 l, corresponding to 43% of the predicted value [2]. She was prescribed inhaled steroids (beclomethasone, 2,000 µg daily) and kept away from work. Two weeks later, her FEV1 had improved to 1.63 l, and bronchial responsiveness to methacholine assessed using a standardized method (output of nebulizer=0.14 ml·min-1) [3] showed significant bronchial hyperresponsiveness (provocative concentration causing a 20% fall in FEV1 (PC20) 0.24 mg·ml-1). She remained away from work. FEV1 further improved to 2.4, 2.7 and 2.5 l and PC20 to 0.7, 3.6 and
3.4 mg·ml⁻¹ when they were assessed 3, 5 and 7 weeks away from work, respectively, at a time when she was still on beclomethasone, 2,000 µg daily. During this period, she was given a mini-Wright peak flow meter to assess her peak expiratory flow rates (PEFR) every 2 h whilst awake. This showed minimal changes (fig. 1). She was then asked to return to work, her medication being unchanged. This resulted in the reappearance of symptoms on the first day of exposure, with a progressive fall in PEFR, that remained constant during the weekend (fig. 1). Her FEV₁, measured after 5 days at work with a two day break at the weekend, dropped to 2.0 l, and PC₂₀ to 0.11 mg·ml⁻¹. She was put on sick leave and continued to assess her PEFR. Values improved progressively, and two months later, there were no significant changes in daily PEFR, FEV₁ had increased to 3.2 l (normal value), and PC₂₀ was 3.1 mg·ml⁻¹ (mild airway hyperresponsiveness).

**Specific inhalation challenges**

The subject underwent specific inhalation challenges in the following way: she had a first series of tests in which no significant (<10%) changes in FEV₁ occurred after a control day of nonexposure and after exposure to a control diluant (polyol), nebulized for 30 min at a distance of 1 m from her mouth in a challenge room [4]. The next day, the subject was exposed to heated polypropylene for 4 min. This was done using the same material as that used at work, which, according to the safety data sheet, contained no other contaminants besides polypropylene. There was a maximum fall in FEV₁ of 16% recorded 20 min after exposure ended, with partial recovery to -11% compared to the pre-exposure value. This was followed by a progressive late reaction (maximum fall in FEV₁ of 34% 6 h after exposure ended). Oral temperature increased to 37.5°C (from a baseline value of 36.3°C), but white blood counts (WBC) were normal (6,600 cells·mm⁻³). These tests were repeated one month later. This time the subject was exposed to heated polypropylene for progressive periods of 4, 8 and 20 min (fig. 2). There was a significant late reaction after the last exposure, with a maximum fall in FEV₁ of 31% 7 h after exposure ended. Oral temperature increased to 37.5°C from 36.4°C. WBC was 8,100 cells·mm⁻³ at the time of the maximum reaction and 7,000 cells·mm⁻³ the following morning. There were no significant changes in vital capacity. The next morning, the patient underwent a bronchoscopy with bronchoalveolar lavage. The total number of cells was 11.3×10⁶ (normal value), but neutrophils (9%) and eosinophils (6%) were increased. Lymphocytes were normal (11%).

Heating polypropylene to 250°C results in the release of various degradation products (aliphatic hydrocarbons, aldehydes (mainly formaldehyde), and ketones) [5]. As formaldehyde is recognized as causing occupational asthma [6, 7], we decided to expose the patient to formaldehyde by nebulizing it at a distance of one metre from her mouth in a challenge room. Exposing the patient to formaldehyde for increasing periods up to a maximum of 2 h elicited no significant change in FEV₁ in the hours that followed.

![Fig. 1.](image-url)Fig. 1. – a) Significant fall and increase in daily fluctuations in PEFR after return to work, with fall in FEV₁ and increase in responsiveness to methacholine (fall in PC₂₀). The horizontal lines on the upper panel represent periods spent at work. b) No significant changes in PEFR and improvement in FEV₁ and PC₂₀ after a period away from work. PEFR: peak expiratory flow rate; FEV₁: forced expiratory volume in one second; PC₂₀: provocative concentration producing a 20% fall in FEV₁.
Our subject had an increase in oral temperature at the time of the late reaction. However, this was not accompanied by significant increase in white blood count or by a reduction in vital capacity, which makes the possibility of alveolitis less likely. Bronchoalveolar lavage revealed the presence of increased percentages of eosinophils and neutrophils. Although the increase in eosinophils is typical of late reactions, the increase in neutrophils is not, except, according to some researchers, in the case of toluene diisocyanate, which is a low molecular weight agent [12].

We conclude that one or other of the degradation products of heated polypropylene is a cause of occupational asthma. The prevalence of this new type of occupational asthma remains to be explored.

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References