

CASE REPORT

Occupational asthma induced by tall oil in the rubber tyre industry

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Summary

A worker in the rubber tyre industry is described with occupational asthma from exposure to a solution of tall oil, a pine resin, confirmed by specific inhalation challenge. This supports studies of contact dermatitis which have suggested abietic and dehydroabietic acid oxidants to be the cause of colophony induced allergic reactions.

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Introduction

When colophony is heated in temperatures above 250°C, resin oil composed of abietic acid is formed and has been suggested to be the main active agent in colophony induced asthma. Oxidation products of abietic acid and dehydroabietic acid have been reported to be contact allergens [1–3]. Tall oil resin is a pine resin of abietic, and dehydroabietic acid, and contact allergy has been reported with exposure to resin of tall oil in sand cores [4]. Occupational asthma has been well described secondary to fumes from heated colophony resin in soldering flux [5–7]. However, there has only been one previous patient described with occupational asthma due to a pine oil preparation [8]. This was present in emulsified oil mist from a coolant used by a toolsetter. The patient described here developed occupational asthma in the rubber tyre industry with exposure to heated crude tall oil used as an adhesive.

Case report

The subject was a 55-year-old man who had worked for a company manufacturing rubber tyres for 32 years. For the last 20 years, he had worked as a cushion mill operator, applying a hot rubber coating to rubber strips. Two years prior to assessment, the chemicals in his work were changed to the use of 'Pamtac 1500'; crude tall oil, a pine resin of abietic and dehydroabietic acids, heated to 100°C. One year later, he developed symptoms of rhinitis immediately on starting work and also developed exercise

induced asthma. He received no medication but three months later required hospital admission for asthma. Symptoms cleared and he took no regular medications. Three weeks later he returned to work and symptoms recurred after 5 days, requiring further hospital admission. He took regular bronchodilators and inhaled steroids following that admission and felt well off work. He had a past history of rhinitis for several years and a nasal polypectomy 6 months before the onset of asthma symptoms but no history of ASA sensitivity. He was referred by the Ontario Workers' Compensation Board for an assessment 7 months after leaving work. Physical examination at that time was normal except for mild nasal mucosal swelling. Skin prick tests to a group of 16 common allergen extracts (Bencard, Mississauga, Ontario) revealed no significant weal and flare responses. On pulmonary function testing, airflow rates, lung volumes, and diffusing capacity were normal, except for a mild reduction in flow rates at 25% of vital capacity (25% of predicted). There was no change following a bronchodilator. Methacholine challenge by the method of Juniper and colleagues [9] showed mild bronchial hyper-responsiveness with a PC₂₀ (provocation dose producing a 20% drop in forced expiratory volume in one second [FEV₁] of 2 mg/ml). Arrangements were made for him to return to work, recording peak flow rates at home and at work. Peak flow rates at work fell from levels above 400 down to 260 l/min (Fig. 1). After two days at work, his FEV₁ had fallen from 4.5 l to 1.2 l and repeat methacholine challenge could not be performed. Over the next two weeks off work, peak flow rates returned to above 400 l/min and the FEV₁ went up to 4.3 litres. Single-blind inhalation challenge testing was performed in a chamber with Pamtac 1500 heated in a glass beaker to 100°C using

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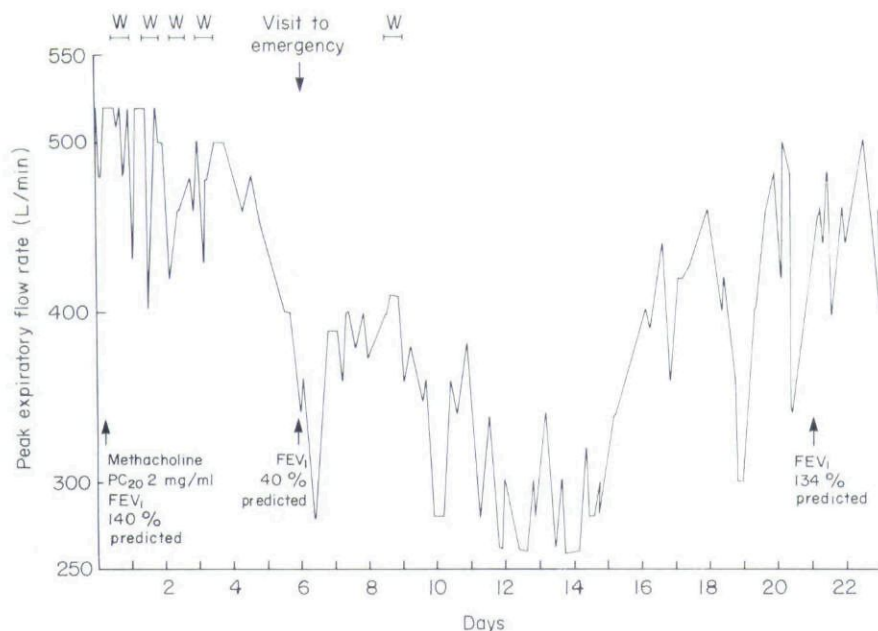


Fig. 1. Peak flow rates (l/min) before and after return to work. W, work day.

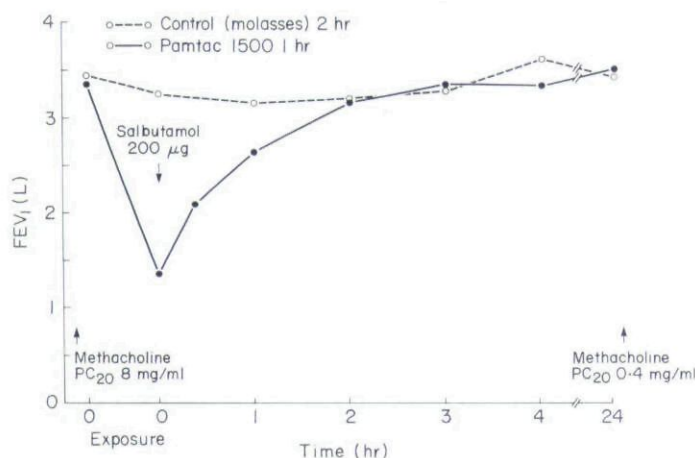


Fig. 2. Single blind challenge testing with a control substance (heated molasses) for 2 hr of exposure and heated Pamtac 1500 for 1 hr of exposure. Methacholine response assessed before challenges and 24 hr after Pamtac 1500 exposure.

molasses as a control substance of similar appearance on a separate day. Pulmonary function remained stable on the control day, but after exposure to Pamtac 1500 for 65 min he became very dyspnoeic with a 60% fall in FEV_1 (Fig. 2). He improved with administration of salbutamol but 24 hr later although his FEV_1 had returned to baseline levels, his methacholine response had increased 20-fold with a PC_{20} of 0.4 mg/ml. There was no

late response by 4 hr in the laboratory nor any difference in peak flow readings up to 24 hr by hourly peak flow readings at home while awake when comparing control and active challenge days. These did show significant variability from 360–480 l/min both days.

Patch skin tests using abietic acid 10%, colophony 20%, and pine tar (pix Liquida) 3% were negative at 48 and 96 hr as were other standard patch tests including rubber components.

Discussion

Workers in the rubber industry are exposed to numerous chemicals and contact dermatitis to many of these has been reported [10,11]. Occupational asthma to latex has been recently reported [12] in a surgical glove manufacturing plant and immediate skin sensitivity documented to some of the other workplace chemicals in the same study. However, occupational asthma induced by heated pine products has not to our knowledge been previously reported in the rubber industry. The only previous report of occupational asthma induced by tall oil resin was to an aerosolized mist of tall oil resin used in a coolant by a toolsetter [8]. Our findings of occupational asthma induced by heated abietic and dehydroabietic acid support the previous reports on skin sensitivity [2,3], suggesting that oxygenation products of these are the main cause of colophony sensitivity. This would be in keeping with the suggestion by Karlberg that pure abietic acid is not

allergenic [13] and supports the concept that oxygenation and other secondary products as in our patient or workers soldering with colophony resin and perhaps with overheating of a coolant may be the sensitizing agents. The negative patch tests to abietic acid and colophony in this patient are not surprising since he had no history of contact dermatitis or a delayed hypersensitivity response to these. Volatile oxygenation products produced during heating of the workplace substance could not be used for skin testing or *in vitro* antibody determination.

The findings in this patient were of occupational asthma as demonstrated by history, peak flow studies at work and off work with accompanying changes in methacholine responsiveness and supported by the specific challenge response. The patient was not kept in hospital after the specific challenge test and therefore a late asthmatic response later than 4 hr after exposure could have occurred. The peak flow readings did not reflect changes greater than on the control day but Berube *et al.* [14] have recently shown that peak flow readings are less sensitive than FEV₁ in assessing late asthmatic responses. An increase in airway responsiveness to methacholine occurring after an occupational challenge is often associated with a late asthmatic response, and this possibility cannot be excluded in this patient. An irritant airway response cannot be excluded since other asthmatics with similar baseline airway responsiveness were not challenged in the same manner. No such asthmatic was present in the workplace and it was not considered ethical to expose other asthmatics in that manner for fear of sensitizing them to a substance which they might subsequently encounter in pine products in daily life. However, the patient did not develop any symptoms of eye or nose irritation during the challenge which might have been expected if irritant levels were reached. In addition the previous report of sensitivity to tall oil resin in a coolant included challenge of a control asthmatic which was negative [8], indicating its capacity to cause specific sensitization.

Therefore tall oil resin should be included among the agents which can cause specific allergic responses and occupational asthma in the rubber industry.

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