Occupational asthma due to unheated polyvinylchloride resin dust

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ABSTRACT Polyvinylchloride (PVC) resins are widely used in industry. Asthma due to the thermal degradation products of PVC are well documented. In this first case of occupational asthma due to unheated PVC resin dust the patient was exposed to PVC resin dust during the mixing of chemicals used for making plastic seals for bottle caps.

Meatwrappers' asthma and asthma due to the thermal degradation products of polyvinylchloride (PVC) are well documented.¹⁻⁶ Pneumoconiosis⁷⁻⁹ and interstitial pneumonitis¹⁰ among workers exposed to PVC dust have been reported. Respiratory symptoms and abnormalities of lung function and of the chest radiograph have been described in surveys of workers exposed to PVC dust.^{11,12} We report the first case of asthma induced by occupational exposure to unheated PVC resin dust.

Case report

A 32 year old man worked for 14 years in a factory manufacturing bottle caps. Each metal cap was lined with a plastic seal on its inner surface. Both the fabrication of the metal caps and the injection moulding process of making plastic seals were carried out in the main production hall of the factory. The PVC resin mixture for the injection moulding process was prepared in a fairly large air conditioned room that was separated from the main production hall. For the past eight years the patient worked in this mixing room where he was exposed to PVC resin dust and other chemicals during mixing.

He started to have episodes of cough and breathlessness about five years after working in the mixing room. Initially, the frequency of symptoms was once in three to four months. During the past year, however, his symptoms had occurred almost daily and he had to depend on a ventolin inhaler for relief. He worked a five day week from 0730 to 1630. His symptoms usually started at about 0200 or 0300. Symptoms improved on weekends and holidays.

There was no associated rhinitis. He had no history of asthma or atopy. His sister had a history of asthma.

OCCUPATIONAL EXPOSURE

The preparation of PVC resin mixture in the mixing room was observed and the exposure dust concentration monitored. Three types of chemicals were blended together to form the mixture: (i) PVC resin, a white powder, (ii) dioctylphthalate or Di-2-ethylhexylphthalate (DOP), a plasticiser in the form of a clear oil liquid, and (iii) a paste containing azodicarbonamide (a plastics blowing or foaming agent), colouring agent and stabilisers. The PVC resin itself was a mixture of three grades of PVC resin: types A, B, and C. Type A was an emulsion resin with no stabilisers added, type B an emulsion resin that had been stabilised against heat and type C a suspension resin with no stabilisers added. The ratio of A:B:C was 3:1:0.7. The paste containing azodicarbonamide and the liquid DOP were first placed in a tank, then five bags (120 kg) of PVC resins were manually poured into the tank. The pouring process was visibly dusty and took about 10 minutes. No local exhaust ventilation was provided.

A quartz crystal microbalance cascade impactor was used to monitor the dust concentrations in the breathing zone of a worker during the pouring of PVC resins. The average concentrations of exposure to the respirable ($< 10 \mu\text{m}$) and non-respirable dust were 0.16 mg/m^3 and 0.37 mg/m^3 respectively. The aerodynamic diameter of the respirable dust ranged from 1 to $9 \mu\text{m}$. The background total dust concentration was 0.02 mg/m^3 . The threshold limit value of PVC is 5 mg/m^3 for respirable dust.¹³

After the pouring of the PVC resins, the liquid mixture was mechanically blended for about 45 minutes. This later process was not dusty and was totally enclosed. The blended mixture was then transported

out of the mixing room to the main production area where it was used to make the plastic seals for the bottle caps.

A total of six batches of PVC resin mixture was made each day over an eight hour work shift by two workers who were confined to the mixing room and did no work in the main production area.

The other worker had no symptoms. There was, however, a further worker who had developed episodes of cough and breathlessness about three years after working in the mixing room. His symptoms occurred two to three hours after starting work and sometimes in the evening after work. He left the factory about five years ago. His symptoms have improved but occasionally he still has asthmatic attacks associated with upper respiratory tract infections. He was not keen to have any medical investigations.

SERIAL MEASUREMENTS OF PEAK FLOW AT HOME AND WORK

Peak expiratory flow rate was measured every three hours from waking to sleeping over a three week period (fig 1). The mean peak flow rate improved during the weeks at home and deteriorated during the working week. The fall in minimum peak flow rate on one day in each of the two weeks at home probably suggests some non-specific bronchial hyperreactivity.

BRONCHIAL PROVOCATION TESTING

Bronchial provocation testing was carried out on an inpatient basis. The patient had been transferred from the mixing room about six weeks before. On the first day no provocation was carried out and a baseline peak flow rate obtained. On the second day provocation was carried out by the worker using a spoon to transfer lactose powder between two compartments of

Fig 1 ■ Daily maximum, △ mean, and ● minimum peak expiratory flow.

Fig 2 Peak expiratory flow baseline (■ day 1) and after exposure to lactose (△ day 2) and to PVC resin dust at room temperature (● day 3). Arrow indicates time of exposure on days 2 and 3.

a tray for 20 minutes. The average concentrations of exposure to lactose dust were 0.13 mg/m^3 for respirable dust and 0.48 mg/m^3 for non-respirable dust. The diurnal variation in the peak flow rate was 18%. On the third day provocation was carried out with PVC resin dust for 20 minutes using the same procedure. The average concentrations of exposure to the PVC resin dust during the provocation were 0.12 mg/m^3 for respirable dust and 0.20 mg/m^3 for non-respirable dust. The peak flow rate started to fall about nine hours after exposure and reached a maximum reduction of about 58% of the baseline 16 hours after exposure. Nebulised ventolin was administered and the peak flow improved after that (fig 2). No provocation test was done with the liquid DOP and the paste containing azodicarbonamide.

OTHER INVESTIGATIONS

Chest x ray examination did not show any evidence of pneumoconiosis. The patient had non-specific bronchial hyperreactivity as assessed by histamine inhalation challenge, the $\text{PD}_{20} \text{ FEV}_1$ being $0.095 \text{ } \mu\text{mol}$. Skin prick testing to common environmental allergens was positive to house dust mite, feathers, and cat fur.

Discussion

Our patient had occupational asthma from exposure to PVC resin dust. Based on the history, another worker who had already left the factory probably also had occupational asthma from the PVC resins. Our patient was also exposed to DOP and azodicarbonamide. Azodicarbonamide dust is known to induce asthma.^{6,14,15} So far DOP and other phthalate esters

have not been identified as inducing asthma.^{6,16} The azodicarbonamide was in paste form and the DOP in liquid form. As the process only involved mixing the constituents at room temperature it was unlikely that any fume, dust, or vapour would be generated from these two chemicals. On the other hand, the pouring of PVC resin was visibly dusty. Thus we suspected that the PVC resin dust was the most likely causative agent. This was confirmed by the bronchial provocation test.

PVC resins are produced by four basic processes: suspension, emulsion, bulk, and solution polymerisation. PVC suspension resins are usually dust free and granular. PVC emulsion resins are small particles containing little free monomer.¹⁷ PVC resins are generally used with admixtures of stabilisers (to retard degradation), lubricants, plasticisers (to increase the flexibility of the final plastic), blowing agents, and pigments depending on the specific requirements.¹³ The PVC resin used in our case was a mixture of the suspension and emulsion types. The plasticiser, blowing agent, and pigment were only added during the mixing process. According to the supplier, the PVC resin was 98.5% pure. Only one of the three grades was stabilised against heat. We do not know the type of stabiliser used but commonly used stabilisers include lead soaps and salts, barium cadmium soaps, and cadmium zinc soaps.¹⁷ These are not known to cause asthma. They are also not likely to cause asthma.

The most likely cause of the occupational asthma, we believe, is the PVC dust itself. This case differs from the asthma caused by the degradation products of PVC. The combustion of PVC causes the formation of highly irritant substances such as hydrogen chloride and phosgene.¹³ Emissions from hot wire cutting of PVC packaging film include hydrogen chloride and dioctyl adipate.⁴ Our patient was exposed only to PVC resin dust at room temperature.

PVC resins are widely used in industry. PVC resin dust has for many years been considered a nuisance. Circumstances of exposure may vary depending on the type of PVC resin and the method of use. This case has shown that PVC resin dust may cause asthma and steps should be taken to reduce occupational exposure to the dust.

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