

Asthma from plexiglas powders

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Summary

A patient with severe asthma induced by the inhalation of plexiglas dusts is reported. Inhalation testing against various industrial products to which he was exposed at work, showed a specific and reproducible asthmatic reaction to plexiglas dusts. Complete pulmonary investigations after positive challenge excluded associated alveolitis and demonstrated an immediate and a late obstructive response. The patient was not atopic and his clinical history did not suggest occupational disease due to chronic exposure. Reduction of the plexiglas dust exposure resulted in progressive improvement.

Introduction

Immediate and late asthmatic reactions may appear during the synthesis of resins. They are generally precipitated by the inhalation of component powders or fumes liberated when resins are converted to hard solids by the addition of curing agents or hardeners (Fawcett, Taylor & Pepys, 1977; Chester *et al.*, 1977; Dernehl, 1973; Malten & Zielhuis, 1964; Petit, Troquet & Melon, 1961; Maccia *et al.*, 1976; Pepys, Pickering & Terry, 1972; Peters & Murphy, 1971). After polymerization, the materials are considered harmless although they might become toxic when altered by high temperatures (Sokol, Aelony & Beall, 1973; Williams & Smith, 1972; Paisley, 1969; Pepys *et al.*, 1972). The non-toxicity of the finished product is due to its physical characteristics (hard, non-volatile) and from inactivation of the chemical components by the incorporation of macromolecules.

We report here an observation suggesting that plexiglas, a polymerized acrylic resin, can induce severe asthmatic reactions when converted into airborne dusts.

Case report

A 33-year-old man was seen for the first time in June 1976. For one month, he had

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suffered from a dry cough, rhinitis and severe asthma mostly in the evening, at home. There was no family history of allergic disease. His smoking habits, working day exacerbations or week-end improvements did not suggest an obvious causal factor. On examination, he had obstructive rhinitis with polyposis, wheezing and severe breathlessness. The chest radiograph was normal, respiratory function investigations showed normal diffusion and marked obstructive airways disease, reversible after inhalation of sympathomimetic amines. Laboratory results were as follows: eosinophilia of 747 cells/mm^3 , normal IgA, IgG and IgM levels, total haemolytic complement, C_3 , C_4 and α_1 -antitrypsin, low IgE levels— 4 U/ml (PRIST), negative specific IgE (RAST) with classical allergens and no precipitins to *Aspergillus fumigatus* or *Candida albicans*. Skin tests (Bencard) were also negative. Numerous eosinophils were found in the sputum.

During the following 6 months, the patient was treated with sympathomimetic amines, theophylline, DSCG, corticoids (intermittent therapy, 5–30 mg of Prednisolone per day) and home rest. Nevertheless, he had to be hospitalized four times in severe crises responding only to high doses of Prednisolone. Although his previous history was not helpful, occupational asthma was investigated. For 6 months before the onset of asthma, the patient worked in a factory manufacturing electrical equipment. He was employed on various jobs such as cutting plexiglas plates, manipulating phosphorescent products, soldering aluminium wires and spraying epoxy resin paints.

Furthermore, on a home visit, we learned that the patient was living in the caretaker lodge at the factory. Dustbins containing plexiglas dusts were collected in a courtyard which separated the basement flat from the factory. Consequently, occupational inhalation challenge tests were undertaken in the presence of the various industrial substances to which he was exposed.

Occupational challenge

The studies of lung function, which included spirometry, determination of airway resistances and thoracic gas volumes in a constant volume–total body plethysmograph were performed for each challenge test. An exercise test was also performed. The test exposures were carried out as described by Pepys (1975). Leucocytosis and temperature were measured three times per day (09.00, 12.00 and 18.00 hours) and chest X-rays were taken half an hour and 7 hr after a positive challenge.

The patient was hospitalized for 15 days before provocation. Daily baseline readings of FEV_1 and FVC and airway resistances were determined in the absence of drug therapy. The challenges, which consisted of shaking powers or shaving dust, and painting with epoxy resin or TDI varnish, were practised for 30 minutes in an isolated room of 3 m^3 . Each test was stopped immediately respiratory distress occurred. Similarly he was exposed to fumes liberated in the process of soldering as carried out in his occupation.

Results

No significant fluctuations of respiratory function occurred during the observation days or after exercise, soldering with aluminium wires, painting with epoxy resin and manipulating phosphorescent powder tests (Fig. 1).

However, when he handled plexiglas shavings dust, the patient developed an immediate asthmatic reaction complicated with cough and haemoptysis (Fig. 2). The symptoms were reproduced by a second provocation test carried out 2 days later. This challenge induced a greater immediate and a late asthmatic response (Fig. 2) and both

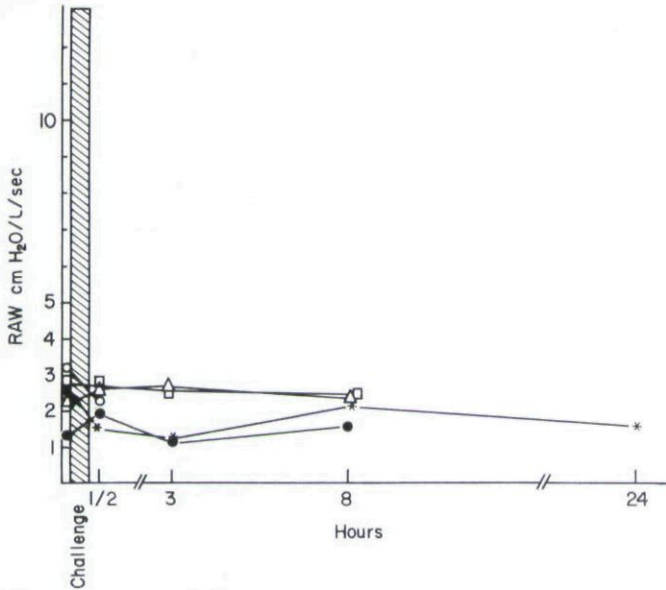


Fig. 1. Challenges with fumes liberated from aluminium wires soldering and epoxy resin painting, phosphorescent powders and exercise test. ○, Exercise; △, aluminium soldering; □, resin epoxy painting; ●, phosphorescent powder; *, spontaneous evolution.

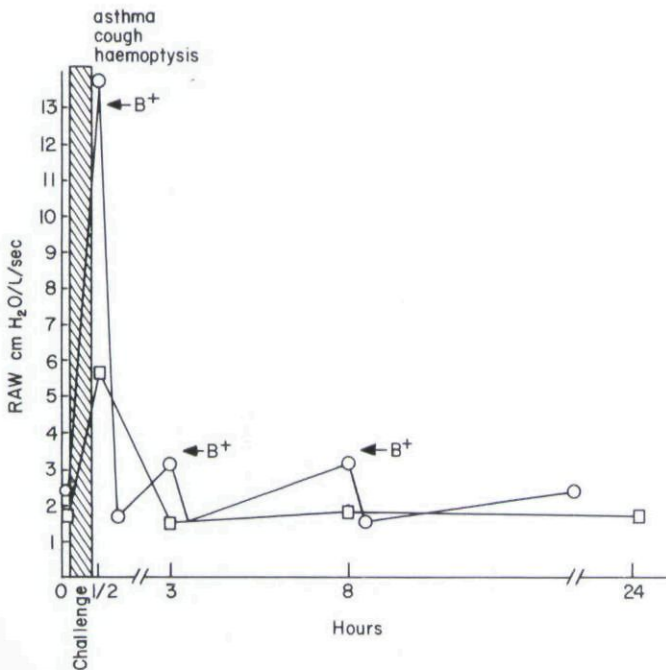


Fig. 2. Immediate and non-immediate asthmatic reactions to challenge with plexiglas dusts. B⁺, two puffs of Fenoterol. □, First test; ○, second test.

Table 1. Evolution of the lung function after the first and the second plexiglas dust exposures.

		Hours after				
	Before	0.5	3	8	24	
First exposure						
FEV ₁ cm ³	3050	1750*	2900	3250	3500	
FVC cm ³	4300	3500	4250	4500	4350	
FEV ₁ /FVC %	71	50	68	72	77	
FRC cm ³	2484	4369	2608	2571	2361	
TLC cm ³	5684	6869	5708	6024	5561	
DLCO cm ³ /min/mmHg (single breath method)	21.43	22.15	19.82	18.45	21.01	
RAW cm H ₂ O/l/sec.	1.6	5.59	1.25	1.84	1.75	
Second exposure						
FEV ₁ cm ³	3100	1350*	2700	3500*	3500	
FVC cm ³	4250	2800	4200	4300		
FEV ₁ /FVC %	73	48	64	86		
FRC cm ³	2487	4700	2162	2383		
TLC cm ³	5587	6350	5560	6283		
DLCO cm ³ /min/mmHg (single breath method)	20.81	23.62	20.68	19.36		
RAW cm H ₂ O/l/sec.	2.35	13.7	3.08	3.1	1.2	

* 80 µg of Fenoterol was administered.

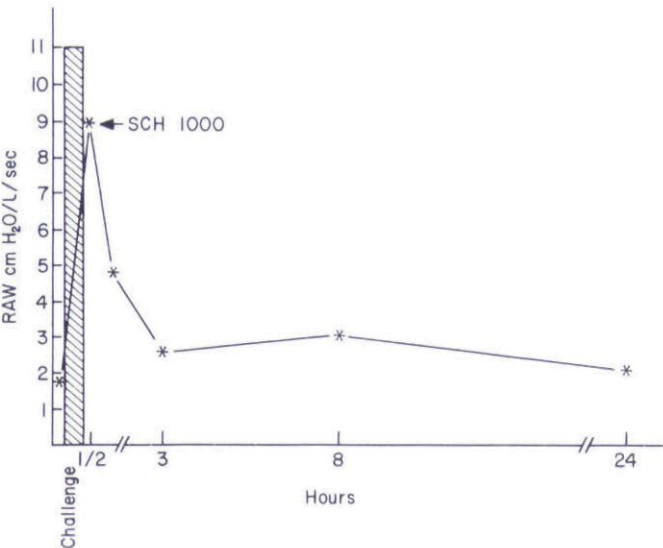


Fig. 3. Immediate asthmatic reaction to challenge with TDI varnish. SCH 1000, one puff.

responded to Fenoterol inhalation (80 μ g). The other functional measurements (Table 1) showed a decrease of FEV₁/FVC ratio while simultaneously the airway resistance increased. There was no alteration of the gas transfer (DLCO). No infiltration was seen on the chest X-ray taken half an hour and 7 hr after the challenge. A slight leucocytosis was also observed 3 hr after plexiglas dust exposure.

Finally, an immediate asthmatic response was obtained after painting with TDI varnish (Fig. 3).

Discussion

Plexiglas is a polyacrylic resin resulting from methylmetacrylate polymerization. The classical synthesis is as follows: methane $\rightarrow$ methylic alcohol + metacrylamide $\rightarrow$ methylmetacrylate $\rightarrow$ polymethylmetacrylate (Plexiglas). Polymethylmetacrylate is translucent, easily manufactured and has dielectric properties. It is used in moulding forms, hard primary products, emulsions and solutions. It is altered at 200°C and at 400°C monomers are collected. The curing agents used for plexiglas synthesis are benzoyl-peroxide and ethyl-peroxide.

In liquid form, the methylmetacrylate monomer is an irritant to the skin and may induce contact dermatitis (Fries, Fisher & Salvati, 1975; Fisher, 1956; Pegum & Medhurst, 1971). The fumes of methylmetacrylate are also irritant to the eyes, the mucosa and the respiratory tract (Lefaux, 1966). When completely polymerized the plastic product is considered harmless (Farri & Morel, 1964), while dust of acrylic emulsion polymer has been shown to be a highly respirable particulate (Cohen, 1974).

Our report shows that airborne forms of plexiglas can induce asthma and the occurrence of immediate and late asthmatic reactions with ocular and nasal participation suggests an allergic process.

The fine shavings dust comes from sawing hard translucent plexiglas plates with electric power saws. The cutting does not increase temperature or liberation of fumes because only small pieces are treated. The patient did not complain of pulmonary distress when he was sawing. So, the possibility of degradation of the plexiglas to monomer form during manipulation is unlikely. The plates of plexiglas did not contain pigments. No liberation of TDI was detected during handling of plexiglas shavings dust (colorimetric method of N.I.O.S.H. Reagent Kit no 78082 limited to 0.01 p.p.m.).

The patient was also shown to have bronchial hyperreactivity to TDI vapours to which he was not exposed. This suggests that he had been sensitized to these products earlier when he used various forms of varnish at work, or, that it is a toxic effect. Indeed, the allergic nature of the bronchial reaction to TDI has been demonstrated at low dose (less than 0.02 p.p.m.) while at high concentration a toxic effect is seen (Butcher *et al.*, 1976).

Occupational asthma is suspected when working day exacerbations, week-end improvements and acute reactions for specific jobs are noted. All of these symptoms were lacking and indeed the patient was often in contact with the substances at work and at home where plexiglas dusts accumulated via a ventilator which aspirated the atmosphere from the factory courtyard into the interior of his home. This continuous inhalation of the plexiglas dusts induced chronic airways obstruction obscuring the acute reaction when sawing plexiglas plates.

Our observations emphasise the importance of occupational challenge tests in severe asthma, when suspected products are handled, even if a typical clinical history is lacking.

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