Occupational asthma due to styrene

J P Hayes, L Lambourn, J A C Hopkirk, S R Durham, A J Newman Taylor

Abstract

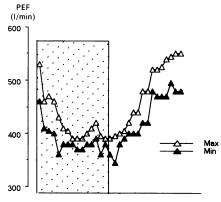
In a patient with asthma who was exposed to styrene serial self recorded measurements of peak expiratory flow showed the asthma to be work related, and inhalation tests with styrene reproducibly provoked a dual asthmatic response and increased responsiveness to inhaled histamine.

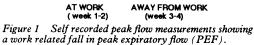
Styrene is a volatile monomer widely used in the production of polymers, copolymers, and reinforced plastics. We report a case of asthma that was shown to be due to exposure to styrene at work.

Case report

A 30 year old RAF air frame technician presented with a 20 month history of chest tightness, wheeze, nocturnal dyspnoea, and decreased exercise tolerance, which he associated with working with fibreglass. In the repair of fibreglass moulds he mixed a polyester resin with an accelerator, which contained a solution of cobalt octoate in styrene, and with an organic peroxidase catalyst. The curing of the polyester is initiated by an accelerator and completed by the catalyst. He had no previous personal or family history of asthma or hayfever. He had smoked 10 cigarettes a day for 10 years and had stopped before the onset of symptoms. He was treated with beclomethasone and salbutamol by inhalation and was relocated at work, with subsequent improvement of his respiratory symptoms. During the following year he was able to discontinue the beclomethasone and had used salbutamol only occasionally for chest tightness.

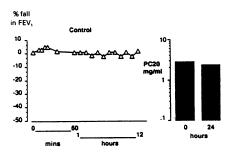
Although he had no direct contact with the



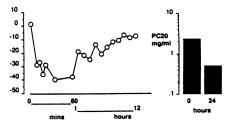


mixing process his symptoms recurred after he returned to the area where he had previously worked. He started inhaling salbutamol and beclamethasone again, and was removed from all contact with the fibreglass process, which was again followed by considerable symptomatic improvement. His forced expiratory volume in one second (FEV₁) and forced vital capacity (FVC) were 3.9 and 4.4 litres and did not improve at this time after inhalation of a bronchodilator. Exercise (five minutes' running) provoked a 19% fall in peak expiratory flow (PEF) from 520 to 420 l/min. He recorded the results of his PEF at two hourly intervals from working to sleeping for two weeks while working with fibreglass and for two weeks while away from work. His PEF deteriorated during the period at work and improved while he was away from work (fig 1). He was referred to the Brompton Hospital for inhalation tests.

Several single blind controlled inhalation tests were undertaken in an enclosed chamber on separate days (fig 2). Before the inhalation tests the dose of histamine causing a 20% fall in FEV₁ (PC₂₀) was 2.75 mg/ml and the variability in his FEV₁ measurements was less than 3% during 12 hours. On the control day







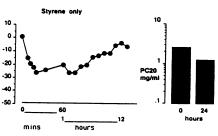


Figure 2 Results of inhalation tests before and after brush painting with 20 ml styrene for one minute, 1%cobalt octoate in styrene for two minutes, and a control substance: percentage fall in FEV₁ and histamine PC₂₀ (concentration causing 20% fall in FEV₁).

Department of Occupational and Environmental Medicine, Royal Brompton and National Heart Hospital, London SW3 6NP J P Hayes L Lambourn S R Durham A J Newman Taylor

RAF Chest Unit, King Edward VII Hospital, Midhurst J A C Hopkirk

Reprint requests to: Dr A J Newman Taylor Accepted 31 January 1991 he painted white spirit on to cardboard for 15 minutes without appreciable change in his FEV_1 or histamine PC₂₀. On the first test day he brush painted the accelerator (1% cobalt octoate in styrene) for two minutes he developed a dual asthmatic response with a decrease in histamine PC₂₀ from 2.3 mg/ml before the test to 0.5 mg/ml 24 hours after the test. Two days later brush painting of styrene alone for one minute provoked a similar dual asthmatic response with a similar reduction in histamine PC₂₀-from 2.7 to 1.3 mg/ml 24 hours after the test. Brush painting with 20 ml pure styrene on another day, which generated an atmospheric styrene concentration of 12 ppm (Gastec colorimeter method, threshold limit value 100 ppm) provoked a similar dual asthmatic response. Mandelic acid, the principle metabolite of styrene,¹ was not detected in the patient's blood or urine, which were sampled immediately and 24 hours after this exposure.

The patient was told of the cause of his asthma and has subsequently avoided exposure to styrene at work. Six months after diagnosis he remains well and uses his bronchodilator inhaler only before strenuous exercise.

Discussion

Styrene or phenylethylene (C₆H₅CHCH₂) was used by this patient for the production and repair of fibreglass moulds. The only previous report of asthma associated with occupational exposure to styrene² described two patients in whom inhalation tests with styrene provoked immediate asthmatic response. an The authors therefore were not able to distinguish whether an irritant or a hypersensitivity response was the cause of the asthma. The findings in this case are consistent with asthma as a manifestation of a specific hypersensitivity response to styrene. Exposure to styrene in an atmospheric concentration of 12 ppm (TLV 100 ppm), insufficient for mandelic acid to be detectable in blood or urine, reproducibly provoked both an immediate and a late asthmatic response and an associated reduction in histamine PC₂₀. Styrene seems able to initiate asthma by inducing a specific hypersensitivity response.

- Guillemin M, Berode M. Biological monitoring of styrene: a review. Am Ind Hyg Ass J 1988;49:497-505.
 Moscato G, Biscaldi G, Cottica D, et al. Occupational asthma due to styrene. J Occup Med 1987;29:957-60.

Thorax 1991;46:397-398

Long survival after excision of a primary malignant melanoma of the oesophagus

F C Hamdy, J H F Smith, A Kennedy, J A C Thorpe

Abstract

A woman who had a large primary malignant melanoma of the oesophagus, with evidence of submucosal invasion and several local metastases, underwent resection two years after the onset of retrosternal pain and has survived for 12 years with no recurrence.

Primary malignant melanoma of the oesophagus is an extremely rare tumour associated with poor survival. Of the 115 cases reported worldwide, the five year survival is 4%.¹ The treatment of choice is surgical resection. We present a patient with primary malignant melanoma of the oesophagus who survived 12 years after surgery.

Case report

A 40 year old woman presented with a two year history of retrosternal pain associated

with progressive dysphagia and weight loss (6 kg in four months). She had no important past medical history and physical examination showed nothing remarkable. Haematological and biochemical investigations gave results within normal limits. Barium swallow examination showed a large, irregular soft

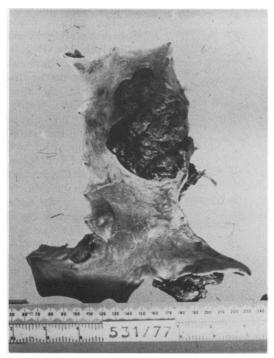


Figure 1 Resected specimen showing an ulcerated black fungating tumour of the lower oesophagus. At its widest the specimen is 17 cm.

Department of Cardiothoracic Surgery F C Hamdy J A C Thorpe **Department** of Histopathology J H F Smith A Kennedy **Northern General** Hospital

Sheffield S5 7AU Reprint requests to: Dr Smith

Accepted 15 February 1991