lowing inhalation.¹⁰ It has consequently been implicated as a cause of occupational asthma, but it has not been shown to cause late asthmatic reactions nor to cause increases in airway responsiveness, and it is unclear whether the operator we describe could have been exposed to airborne metabisulphite during the three days of the workplace challenge study. We would consequently regard this as a much less plausible cause for his occupational asthma.

Isothiazolinone biocides are being used increasingly in a number of industries – for example, the manufacture of cosmetics, shampoos, synthetic fibre, and paper products. Their sterilising properties are used also in humidifier and air conditioning systems. Dermatitis has been reported in consumers as a result of topical contact in both occupational and domestic settings,^{4,5,9} so the potential risk for the development of asthma may be widespread. The opportunity for respirable exposure may, however, be limited. Furthermore, isothiazolinones are generally encountered in

much greater dilution by consumers than by workers engaged in their manufacture.

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Regression of polyvinylchloride polymer pneumoconiosis

Neil W White, Rodney I Ehrlich

Abstract

A 35 year old man heavily exposed to polyvinylchloride (PVC) polymer dust developed dyspnoea and a mild restrictive lung disorder consistent with PVC pneumoconiosis. Clinical and radiological abnormalities cleared on removal from exposure, suggesting that in its early stages PVC pneumoconiosis is reversible.

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Keywords: polyvinylchloride, pneumoconiosis, lung disease.

Case report

In November 1991 a 35 year old plastics factory employee presented with sore throat, blocked nose, chest tightness, and pleuritic chest pains. Physical examination and chest radiography were normal. Pulmonary function test values were at the lower limit of normal (table 1).¹ Serial peak flows recorded over two weeks showed no variability, and no diagnosis was made.

In July 1993 he returned with persistent sore throat, nasal and eye irritation, occasional dyspnoea, and a non-productive cough which had been present for several months. His effort tolerance had declined and he was no longer

able to play soccer. A detailed occupational history established that he had worked since 1985 primarily with a fine polyvinylchloride (PVC) powder, manually loading hoppers under very dusty conditions using only a simple mask as respiratory protection. A visit to the factory confirmed his description; there was no local exhaust ventilation and general ventilation was inadequate. Various pigments and other chemicals were added to the PVC, but he was not involved in this later part of the process.

He had never smoked, had no domestic bird exposure, no tuberculosis contact, nor any history suggestive of allergy or atopy. Examination showed sparse fine crackles over the left lower zone. Full blood count, differential count, international normalised ratio, and serum levels of angiotensin converting enzyme (ACE) were normal.

Pulmonary function testing showed a reduction of more than 600 ml in volumes and a slight decline in gas transfer, indicating mild restriction (table 1). The chest radiograph was unchanged. A high resolution computed tomographic (HRCT) scan of the chest showed a fine nodular pattern in both lower lobes, more extensive on the left, with no pleural or other pathology seen (fig 1). There was no lymphadenopathy nor other signs to suggest sarcoidosis such as nodularity along the bronchovascular markings.

Transbronchial biopsy specimens obtained by fiberoptic bronchoscopy from the left lower lobe showed focal areas of interstitial fibrosis. Conspicuous fibrin deposition was noted within the interstitium. There was a general paucity of inflammatory cells, but the bronchial wall showed a moderate infiltrate of chronic inflammatory cells. The basement membrane was mildly thickened. No granulomatous changes were seen. No exogenous material was

Respiratory Clinic,
Department of
Medicine,
University of Cape
Town, Groote Schuur
Hospital,
South Africa 7925
N W White

Department of
Community Health,
University of Cape
Town,
South Africa
R I Ehrlich

Correspondence to:
Dr N W White.

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Table 1 Serial changes in pulmonary function

	November 1991	June 1993	November 1993	June 1994	July 1995	Mean (SD) predicted
FEV ₁ (ml)	2440	1800	2120	2360	2400	3270 (840)
FVC (ml)	3050	2370	2750	2990	3170	3850 (1000)
FEV ₁ /FVC (%)	80	76	77	79	76	85
TLCO (mmol CO/kPa/min)	78.6	68.7		84.3	80.4	82.8 (20.7)
Kco (mmol CO/kPa/min/l)	20.7	19.8		21.3	21.3	18.3 (3.9)

FEV₁=forced expiratory volume in one second; FVC=forced vital capacity; TLCO=single breath carbon monoxide diffusing capacity; Kco=TLCO/alveolar volume.

identified in the macrophages by scanning electron microscopy.

In November 1993, three months after removal from exposure to PVC powder, improvement in the pulmonary function tests was noted (table 1). By June 1994 he was asymptomatic, with lung function values close to those at his first visit in 1991. On repeat HRCT scanning the nodularity present a year before was much less evident.

Discussion

Pulmonary reaction to polyvinylchloride polymer dust has been documented in epidemiological²⁻⁵ and histological studies.⁶⁻⁸ Clinically, PVC pneumoconiosis is a diagnosis of exclusion in the context of appropriate exposure. Sarcoidosis was the main alternative diagnosis here, but the absence of extrapulmonary clinical features, normal serum levels of ACE, the absence of non-caseating granulomas on transbronchial biopsy specimens, and no characteristic features on the HRCT scan effectively excluded this possibility. Furthermore, the natural history of his complaint and the absence of corresponding positive features were thought to exclude tuberculosis and connective tissue disorders. No risk factors for an extrinsic alveolitis were identified. The only environmental modification that occurred prior to his improvement was his removal from exposure to PVC dust.

Histological examination showed focal areas of fibrosis and conspicuous fibrin in the interstitium, but not some of the features documented in heavily dusted experimental animals⁶ and in case reports of workers with respiratory disease following heavy exposure to PVC who

died⁷ or from whom lung biopsy samples were taken.⁶ Intracellular foreign particles have been seen in macrophages by scanning electron microscopy of specimens from heavily dusted animals.⁶ The other case report in English which described a lung biopsy specimen found that the nodules represented a histiocytic infiltrate in a collagen matrix.⁶ These histiocytes/macrophages have clear vacuoles with a few multinuclear giant cells also present.

The non-specific abnormalities in the airways and parenchyma seen in our case could reflect limited biopsy tissue. Alternatively, we may have documented an earlier stage or milder form of PVC pneumoconiosis, where macrophage mediated fibrosis is limited and largely reversible.

Our patient developed mild ventilatory restriction and slightly reduced gas transfer. Respiratory symptoms, pulmonary function changes, and abnormalities on the chest radiograph have been documented in cross sectional studies of workers employed in PVC production and fabrication. In two studies^{3,4} exposure to PVC dust was associated with mild functional restriction and small rounded or irregular opacities of low profusion radiologically. A third study⁷ found PVC exposure to be associated with airflow limitation, while a fourth documented both patterns of spirometric abnormality.²

The epidemiologic studies did not describe the natural history of the cases of PVC pneumoconiosis identified. We could find no published description suggesting that PVC pneumoconiosis might regress. This case suggests that in its early stages or after a relatively short exposure, PVC pneumoconiosis is largely reversible. Further longitudinal studies are needed before we fully understand the natural history of PVC pneumoconiosis.

Figure 1 HRCT scan of chest showing small regular nodules in both lower lobes.

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