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Prolonged hypoxaemia following inhalation of hydrogen chloride vapour

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ABSTRACT A patient is described who developed prolonged hypoxaemia and subsequent asthma after a single exposure to a high concentration of hydrogen chloride vapour (and a small amount of phosphorus trichloride).

Case report

A 34 year old woman was working on her boat in an open marina when she was surrounded by a cloud of vapour, released from a chemical factory some 100 metres away. She inhaled the vapour, which consisted of hydrogen chloride, water vapour, and a small amount of phosphorus trichloride for 15 minutes. No one else was affected. The vapour caused the paint on the boat to blister. She was admitted to the local hospital with irritation of exposed skin, eyes, and respiratory tract. She was treated symptomatically and discharged on the third day, but became dyspnoeic at home and was admitted later on the same day to the Brompton Hospital.

At the time of admission she was dyspnoeic while talking. Her past history included a cardiac arrest when she had a general anaesthetic. Examination showed tachypnoea, facial erythema, and a hoarse voice. Chest examination showed nothing abnormal and her chest radiograph, peak flow, forced expiratory volume in one second (FEV_1) and forced vital capacity (FVC) were normal initially and throughout her stay in hospital. Initially the arterial oxygen tension (Pao_2) was 7·1 (normal 11·0–12·5) kPa, oxygen saturation 86%, carbon dioxide tension ($Paco_2$) 4·9 (normal 4·7–6·0) kPa, and pH 7·34. With 24–28% inspired oxygen she was able to achieve a Pao_2 of over 10 kPa throughout her hospital stay. $Paco_2$ was normal throughout.

Routine respiratory function tests were performed six times between 12 days and eight months after the incident. Transfer factor for carbon monoxide was at or just below the lower limit of normal on each occasion; transfer coefficient was normal. Total lung capacity (body plethysmograph) was normal, though the residual volume was low throughout. Anatomical shunt[1] was 3·2% at a time when her Pao_2 was 9·0 kPa. A ventilation-perfusion scan was normal (when Pao_2 was 8·5 kPa). During an exercise test arterial oxygen desaturation fell from 94% to 82% (Ohmeda Biox 3700 pulse oximeter). Lung permeability was measured by diethylene triamine penta-acetic acid (DTPA) clearance 46 days after the event and was normal. A lung compliance curve was normal, the quasi static method being used to measure oesophageal pressure during a slow (125 ml/s) expiration from TLC into a rolling seal dry spirometer. The timing of these investigations and Pao_2 measurements is shown in figure 1. A haemoglobin oxygen dissociation curve was normal, P_{50} being 3·45 kPa. Computed tomography of the thorax in expiration showed multiple, mainly peripheral areas of transradiancy scattered throughout the lung fields (fig 2), which were not visible on the inspiratory scans. These abnormalities were still substantial one month after the accident, were less obvious at five weeks, but had not fully cleared at six months.

She was treated with 40 mg of prednisolone daily with inhaled salbutamol and beclomethasone. These did not appear to be effective, and the hypoxaemia persisted. After four weeks the resting Pao_2 rose (fig 1), although she had demonstrable arterial desaturation on exercise. After six weeks she improved symptomatically and was discharged.

Subsequently she has had two admissions with dyspnoea after viral infections and reversible bronchoconstriction (peak flow on admission 300 l/min increasing to 500 l/min with bronchodilators). On each occasion she was hypoxaemic, Pao_2 being 7·4 and 8·7 kPa respectively. During one episode bubble contrast echocardiography showed no evidence of cardiac shunt. She has subsequently developed chronic, reversible airflow obstruction and has been treated with inhaled corticosteroids.

Discussion

Phosphorus trichloride reacts with water to form hydrogen chloride and phosphorus acid and then phosphoric acid. This patient was therefore predominantly exposed to hydrogen chloride vapour, which is irritant to mucous membranes.[2] She experienced prolonged hypoxaemia after a 15 minute exposure. At Guy's Hospital poison centre, 47 cases of hydrogen chloride fume inhalation have been recorded. Most had nausea and vomiting. Those with a past history of asthma tended to develop bronchoconstriction and one developed laryngospasm. Most were rapidly discharged from hospital, but none of those who were admitted developed hypoxaemia and all recovered symptomatically in one week (National Poisons Information Service, personal communication).

Among 11 workers accidentally exposed to a gaseous mixture of hydrogen chloride, phosphorus oxychloride, phosphorus pentachloride, oxalyl chloride, and oxalic acid, one subject remained persistently hypoxaemic for months in association with a reduction in the transfer factor for carbon monoxide, airways obstruction, and lung crackles.[3]

The cause of our patient's hypoxaemia was not determined despite detailed investigations. In particular, there was no evidence of airways obstruction initially, no reduction in transfer coefficient, and no evidence of an anatomical shunt

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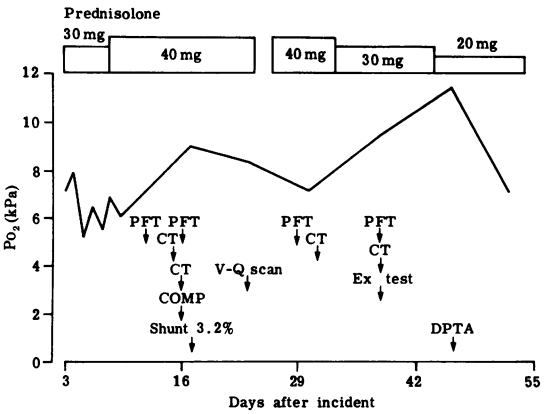

Fig 1 *Partial pressures of arterial oxygen in relation to the timing of investigations after exposure to hydrogen chloride vapour. CT—computed tomography; PFT—pulmonary function tests; DTPA—diethylenetriamine penta-acetic acid clearance; Ex test—exercise test; V-Q—ventilation-perfusion scan; COMP—pulmonary compliance; shunt—anatomical shunt.*

and her haemoglobin oxygen dissociation curve was normal. Possibly she had a large shunt due to pulmonary vasoconstriction when breathing air, which was abolished by the effect of oxygen on the pulmonary vasculature and consequently not detected.

The cause of the scattered areas of low attenuation in the lung fields on the computed tomography scan is not clear; they may represent areas of air trapping or ischaemia. As her total lung capacity and residual volume were less than predicted, local ischaemia due to vasoconstriction, which was reversed when oxygen was administered, is the more likely explanation.

Single exposures of normal subjects to high concentrations of irritant gases may cause persistent asthma, which has been termed reactive airways dysfunction syndrome.[4] This patient would appear to fit into this category, although her bronchial responsiveness has not been measured. Bronchiolitis obliterans has been recorded after exposure to irritant gases,[5] but this patient had no evidence of airflow obstruction during her initial admission and her subsequent airflow obstruction was reversible. Lung function tests between attacks of asthma showed no airways obstruction.

We have described for the first time a patient with prolonged hypoxaemia following hydrogen chloride inhalation. The physiological cause of our patient's hypoxaemia was not determined despite detailed investigation.

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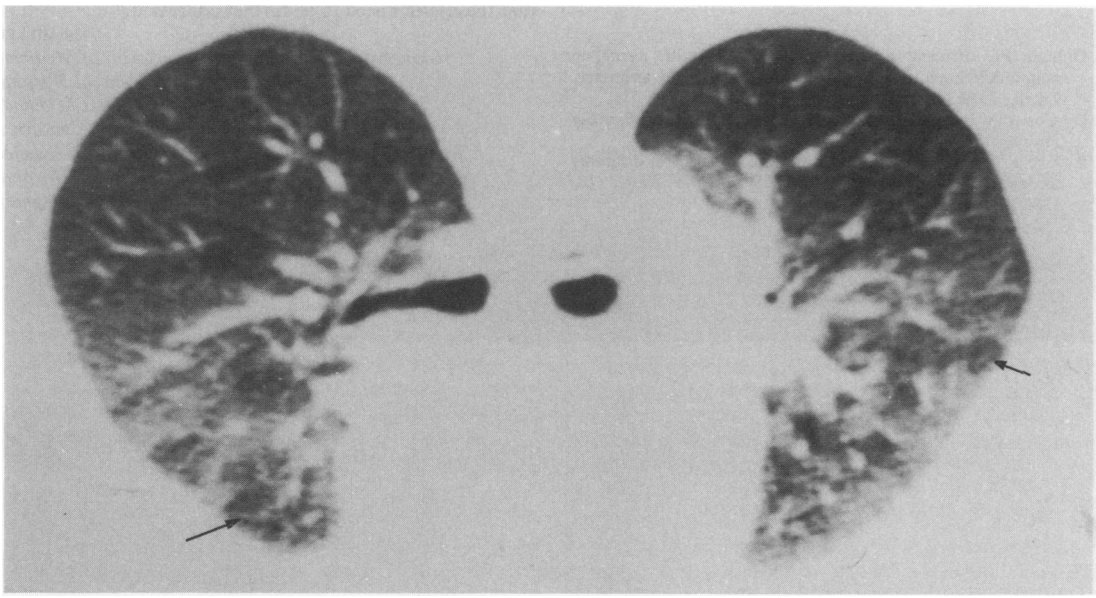

Fig 2 *Representative prone expiration computed tomography scan of the thorax showing low attenuation areas (arrowed).*