British Journal of Industrial Medicine 1991;48:286–287

Inhalation of hydrogen sulphide: a case of subacute manifestations and long term sequelae

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Accidents to agricultural workers using storage facilities for liquid manure, mainly in swine confinement buildings, have been described.[1 2] Fermentation of organic matter releases toxic gases such as hydrogen sulphide, ammonia, methane, and carbon monoxide. Usually the concentrations of these gases exceed their threshold limit values in the confinement buildings. A liquid manure pit may be another source of serious threat when the pit is entered without preventive measures.[3] We report a related accident in non-rural circumstances and describe a peculiar clinical manifestation.

A 30 year old man was admitted to the hospital because of dyspnoea, chest tightness, and haemoptysis. He had smoked a pack of cigarettes a day since the age of 18. He had been working as a keeper in a steel foundry during the past two years and had no history of chronic cough, sputum production, or respiratory disease.

Three weeks before admission he noticed eye, nose, and throat irritation when entering the toilet facilities of the building where he worked. These symptoms disappeared after an hour. On the same day three other workers were admitted to hospital because of nausea, vomiting, dizziness, and dyspnoea; one of them died a few hours later. Only one of the workers noticed on odour of rotten eggs when entering the toilet facilities. Ten more workers reported nausea, vomiting, itchy eyes, and nose irritation and recovered without complications after a few hours. Our patient was free of symptoms until three weeks later when be began to notice dyspnoea, chest tightness, and haemoptysis. His temperature was 36·5°C, pulse rate 80 beats a minute, and blood pressure 120/60 mmHg. Physical examination and routine laboratory studies were normal. Chest *x* ray film showed a mild bilateral interstitial pattern (International Labour Office Classification 1/1). Bronchoscopy showed a reddish mucosa all over the bronchial tree. A cell count in bronchoalveolar lavage showed 98% macrophages, 1% neutrophils, and 1% lymphocytes. Bronchoalveolar lavage, bronchial aspiration, and sputum smears were negative for mycobacteria and neoplastic cells. Pulmonary function tests showed a mild restrictive disease (forced vital capacity 4·48 l (81% of predicted value)), forced expiratory volume in one second (FEV_1) 3·56 l (79%), FEV_1/FVC 77%, functional residual capacity 2·87 l (64%), residual volume 1·33 l (61%), total lung capacity 5·81 l (72%), carbon monoxide diffusing capacity (D_LCO) 7·80 mmol/min^{-1}, KPa 63%)). Arterial blood gases with fractional inspiratory oxygen 0·21 showed Pao_2 84 mmHg. $Paco_2$ 35 mmHg, pH 7·45, So_2 96%. The patient remained asymptomatic except for a residual exertion dyspnoea after five months. Blood parameters had returned to normal values but lung volumes and D_LCO were still low five months after the admittance.

Our diagnosis from this background was pneumonitis produced by inhalation of toxic gas. After the reported accident the toilet facilities were closed and the Generalitat de Catalunya Occupational Health Department carried out an investigation two days later. The autopsy of the dead worker showed haemorrhagic bronchitis and identified asphyxia as the cause of death probably secondary to inhalation of a toxic gas. Concentrations of toxic materials in blood were not measured. Concentrations of toxic gases above the threshold limit values were not found in the toilets two days after the accident. The lavatories were connected, however, without a siphon, to a manure pit. The facts that could explain a leak through this circuit were the lack of syphon and the fact that the toilets and the manure pit where they drained had been out of order for several months, being used again only just before the accident. That would have allowed hydrogen sulphide and other gases from the organic decomposition in the pit to accumulate. Neither the absence of hydrogen sulphide in the toilet air nor the absence of the characteristic smell in most cases rule out the diagnostic hypothesis of hydrogen sulphide toxicity as the investigation was carried out two days after the accident and the toilet had been aired before the samples were taken. Furthermore it is well known

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that high concentrations of hydrogen sulphide induce anosmia. Ammonia, methane, and carbon monoxide, which can also be released from liquid manure, cause different clinical syndromes and cannot be responsible for the manifestations seen.[4]

Several similar reports have been published[1-3 5] but the occurrence of our particular patient makes two interesting points not previously noted. The first is the lateness in appearance of the dyspnoea and haemoptysis, consistent with a subacute lung disease. As far as we know only acute illness has been previously reported, the severity of which depends on the gas concentration. The second is the evidence of sequelae manifested by dyspnea on exertion previously observed by Ip *et al*[6] and a mild decrease in lung volumes and D_LCO five months after the exposure. The histological studies made on lungs of dead victims show the presence of an unspecific injury oedema with necrobiosis of the alveolar membrane and of bronchial epithelium without any sign of fibrosis. One of the characteristics of pulmonary injury oedema however, is its evolution to interstitial fibrosis when it does not cause death by acute respiratory failure, as massive inhalation of hydrogen sulphide does in most cases.[5] We think that the development of a mild fibrosis as a sequel to the toxic exposure could explain the maintenance of exertion dyspnoea and the decrease in lung volumes and D_LCO in our patient after five months. This case illustrates a new type of pulmonary injury by inhalation of hydrogen sulphide that appears as subacute illness and develops into chronic functional disability.

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Accepted 1 October 1990