

Short reports

Acute and long term respiratory damage following inhalation of ammonia

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Abstract

A lifelong non-smoker who was the victim of a massive accidental exposure to anhydrous ammonia gas was followed up for 10 years. In the acute phase the patient presented with severe tracheobronchitis and respiratory failure, caused by very severe burns of the respiratory mucosa. After some improvement he was left with severe and fixed airways obstruction. Isotope studies of mucociliary clearance, computed tomography, and bronchography showed mild bronchiectasis. It is concluded that acute exposure to high concentrations of ammonia may lead to acute respiratory injury but also to long term impairment of respiratory function.

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Ammonia (NH₃) is a colourless gas with a very pungent odour and is highly water soluble and irritant. Ammonia intoxication occurs most usually after accidental exposure during the manufacture of fertilisers, textiles, leather, plastics, pesticides, or explosives. It is also used in some refrigerant installations and, in lower concentrations, in household cleaners.

Although the acute pattern of ammonia intoxication is well known,¹⁻⁵ long term anatomical and functional lesions are less clearly documented. We report the case of a patient who has been followed for 10 years after severe inhalation injury caused by ammonia.

Case report

A 28 year old man was admitted to hospital in November 1979. He was a lifelong non-smoker and had no past history of respiratory disease. Although he was a solderer, he had no respiratory symptoms immediately before the accident and had a normal exercise tolerance. He had no personal or family history of atopy, his IgE blood levels were not raised (100 U/ml; normal less than 120 U/ml), and skin tests for common allergens gave negative results. He was working as solderer in the maintenance team of an industrial butter plant when he was



Figure 1 The tracheobronchial matrix that the patient ejected, testifying to the importance of the respiratory lesions.

exposed accidentally to a high concentration of anhydrous ammonia gas after the explosion of a tank.

On admission he presented with bilateral corneal scarring, first and second degree burns of the chest and both hands, and injury to the upper airways consisting of pharyngeal and laryngeal oedema with exudative lesions. The patient complained of dyspnoea and chest tightness and produced copious bronchial secretions. At one point he expectorated a tracheobronchial matrix formed by burned respiratory mucosa (fig 1). Physical examination indicated crackles and wheezes, mainly at the lung bases. The chest radiograph was normal. Arterial blood gas analysis while he was breathing room air showed: pH 7.40, arterial carbon dioxide (Paco₂) 4.4 kPa, and arterial oxygen tension (Pao₂) 8.8 kPa. The erythrocyte sedimentation rate was 64 mm in one hour and the white blood cell count 30 (normal 5.0-10.0) × 10⁹/l. The blood concentrations of ammonia and α₁ antitrypsin were normal.

Fibreoptic bronchoscopy, performed one week after admission, showed severe tracheobronchial damage with diffuse erythema, inflammation of the airway walls, haemorrhagic areas, and abundant purulent secretions. Lung function tests, performed for the first time one week after admission, showed severe airflow obstruction (table). The patient recovered progres-

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Results of lung function tests, 1979-91

	1979		1981		1988	1991	
	Predicted	1 week after admission	Before fenoterol	After fenoterol		Before fenoterol	After fenoterol
VC (l)	4.53	2.15	2.75	2.6	3.31	3.21	3.39
TLC (l)	5.78	5.26	5.54		6.04	7.00	
FEV ₁ (l)	3.65	1.05	1.39	1.74	1.63	1.37	1.71
FEV ₁ /VC (%)	81	49	50.6		49.4	42.62	
TLCO (ml/min/kPa)	219.5	210.5	222.5		204.5		

VC—vital capacity; TLC—total lung capacity; FEV₁—forced expiratory volume in one second; TLCO—transfer factor for carbon monoxide.

sively from the injuries to his skin, eyes, and upper airways without specific treatment. Initial treatment consisted of oxygen, bronchodilators, and antibiotics. Corticosteroids were administered for the first time one week after admission at a dose of 30 mg prednisolone a day followed by gradually decreasing doses, while the airflow obstruction and symptoms were monitored. There was a decrease of respiratory symptoms during the first year and improvement in the bronchoscopic appearance but the patient still had a chronic productive cough, frequent bronchial infections (four times a year), and exertional dyspnoea preventing his return to work. Lung function measurements during over 10 years of follow up are summarised in the table. On presentation the FEV₁ was less than 30% of the predicted value (1.05 l). Subsequent measurements, from 1979 until 1991, gave values varying from 1.37 (41%) to 1.63 (48%) l. Bronchodilator challenge tests on several occasions showed that the obstructive pattern was only partially reversible (table). The mucociliary clearance test (technetium-99m labelled diethylenetriaminepenta-acetic acid aerosol) showed a slowing of elimination of inhaled particles ($T_{1/2}$ = 105 min; normal: 45 (SD 10) min). In 1988 computed tomography of the lung (fig 2) and bronchography showed moderate tubular bronchiectasis in both lungs. Fibreoptic bronchoscopy showed moderate scarring of the bronchial tree and bronchial biopsy suggested residual non-specific inflam-

mation of the bronchial mucosa. In 1991 the patient is still treated with oral and inhaled bronchodilators and intermittent corticosteroids. He continues to have cough, exertional dyspnoea, and recurrent bronchial infections.

Discussion

Ammonia is an irritant gas. Although exposure may lead to skin and eye injuries, lung damage is often the most dangerous feature. The extent of the respiratory injury after inhalation of ammonia depends on the duration of exposure, the concentration of the gas, and the depth of inhalation. Because of its high water solubility, ammonia is quickly absorbed on to mucosal membranes and skin and reacts with water to form ammonium hydroxides, which are highly irritant and caustic. The formation of ammonium hydroxide is an exothermic reaction and this may also cause thermal injury.⁶ Burns and desquamation of the epithelial layer of the upper bronchial tree (tracheobronchitis) are therefore common. Absorption of ammonia in the upper airways provides some protection for the lower bronchial tree and alveoli from ammonia toxicity.

Our patient developed acute tracheobronchitis with severe airflow obstruction after inhaling ammonia, followed by chronic airflow obstruction with bronchiectasis. The acute airflow obstruction that occurs after exposure to such an irritant gas is initially caused by stimulation of the irritant receptors leading to bronchoconstriction and narrowing of the bronchial lumen by oedema and inflammation of the airway walls, with accumulation of desquamated epithelial cells and bronchial secretions in the airways.⁷

The course of the ventilatory impairment during 12 years of follow up showed that, after a small improvement, severe fixed airflow obstruction remained. In this young man, who had never smoked, bronchial injury caused by ammonia exposure was almost certainly responsible for this. The fact that the obstructive pattern was only slightly reversible after bronchodilators suggests the presence of pathological lesions in the airways. Our patient had coexisting bronchiectasis. This has been reported as a sequel of ammonia inhalation^{3,8,9} but the frequency of this complication is not known. It is not clear whether the bronchiectasis resulted from the chemical burns or from subsequent bacterial infections. The obstructive respiratory defect might also be associated with

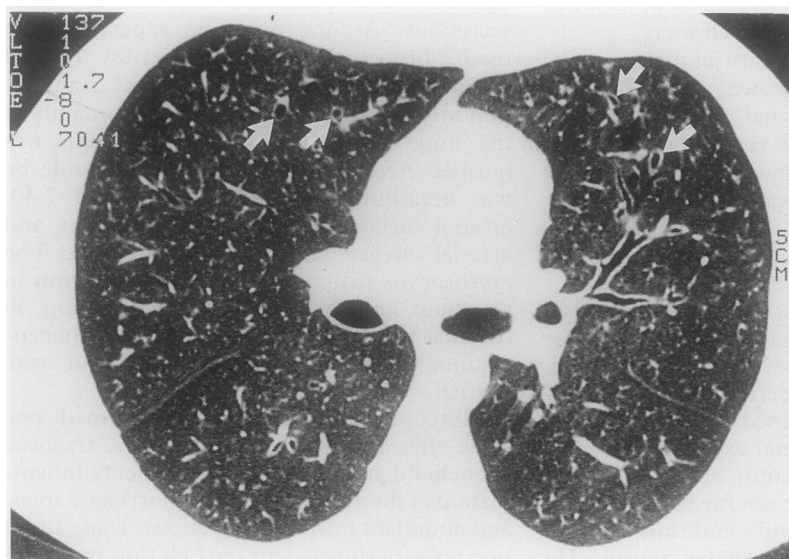


Figure 2 Computed tomogram of the lung showing tubular dilatations of the bronchial structures in comparison with the accompanying vessels.

small airways injury (bronchiolitis). The presence of bronchiolitis could not, however, be confirmed as no lung biopsy was performed.

We conclude that acute exposure to high concentrations of anhydrous ammonia gas leads not only to acute respiratory injury but also to long term impairment of respiratory function.

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Expandable metal stents for tracheal obstruction: permanent or temporary? A cautionary tale

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Abstract

An expandable metal stent inserted via a long term tracheostomy successfully relieved life threatening respiratory obstruction due to benign tracheal stenosis. Later the patient's tracheostomy suction catheter became stuck on the stent and dislodged it. The stent was removed electively, without damaging the trachea, with a rigid biopsy forceps.

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Several reports have highlighted the value of expandable metal stents in the treatment of respiratory obstruction due to benign or malignant conditions affecting the trachea or main bronchi.¹⁻³ Such stents are usually considered permanent as the fixation of the stent hooks to the mucosal wall is thought to make removal difficult.² We describe a patient whose stent became dislodged two weeks after insertion and was therefore removed, without difficulty, via a rigid bronchoscope.

Case report

A 51 year old housewife presented as an emergency with a three year history of increasing dyspnoea and stridor. Ten years previously she had received external radiotherapy to the neck for a postcricoid carcinoma. Three years later she developed laryngeal stenosis secondary to the radiotherapy,

and for the last seven years she had therefore required a tracheostomy tube. On admission she was cyanosed and breathless at rest.

On the day of her admission a flexible endoscope inserted via her tracheostomy site showed a benign looking stricture 3 cm below the tracheostomy site, which had reduced her tracheal lumen to about 2 mm in diameter. An attempt to dilate the tracheal stenosis with a 24 French gauge bougie caused bleeding at the stenosed site, and the patient became increasingly distressed. She was considered too ill to be transferred to the regional cardiothoracic centre and, in the absence of local facilities for rigid bronchoscopy, we decided to introduce a stent.

Two 2.5 × 2 cm self expandable metal stents (Cook Inc, Bloomington, Indiana) were

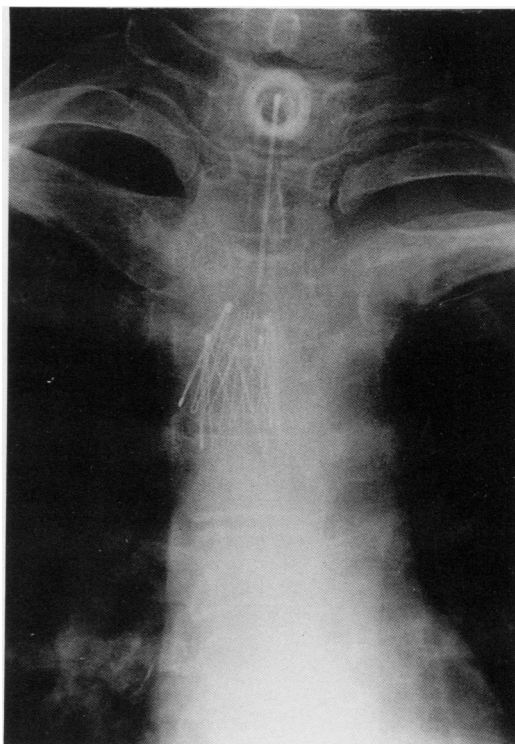


Figure 1 Detail of chest radiograph after disruption of the stent.

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