Medical emergencies

Inhalation of chlorine gas

John G Williams

Summary

The clinical features of acute chlorine gas inhalation, and its management are reviewed. Current medical views on the chronic effects of an acute overwhelming exposure on lung function (reactive airways dysfunction syndrome), and the more controversial field of lung disease secondary to repeated inhalations of lower concentrations of chlorine gas are discussed.

Keywords: chlorine gas, respiratory disease, reactive airways dysfunction syndrome

Singer's moving painting (figure) illustrates the horrors of chemical warfare. Many gases, including chlorine, were released during the First World War. The horrendous morbidity of those affected led to the wholesale distribution of gas masks to the population during the Second World War. Chlorine is still being manufactured and used in industrial processes such as making paper. The acute effects of a significant inhalation are well recognised, namely pulmonary oedema and tracheo-bronchitis. There is increasing evidence that there may be long-term consequences on the respiratory system following excessive exposure. The time is opportune for a review of these persistent abnormalities.

Acute high-dose inhalation

CASE REPORT 1

A 41-year-old man, a maintenance worker at an industrial plant, was asked to replace a relief valve through which chlorine gas was passing. Unfortunately the valve was not functioning properly and he was knocked over by a sudden blast of chlorine gas emitted at an extremely high pressure. He was enveloped in the fumes for a few minutes while trying to make his escape and subsequently developed productive cough with sputum and marked wheeze which has never left him in the nine years since the incident. He is now a steroid-dependent asthmatic, using 6-hourly nebuliser therapy (he was previously a twice-a-week squash player with normal spirometry on medical examinations). He was made redundant on medical grounds and received compensation for his industrial injury five years after the accident.

CASE REPORT 2

In the early 1980s, a 53-year-old man took his employers to court claiming that he had suffered permanent lung damage as a result of an acute inhalational accident in an industrial setting in 1969. He was suffering from an upper respiratory tract infection when he inhaled an intense dose of chlorine gas. Since that time he had been prone to cough, sputum and breathlessness, punctuated by episodes of acute infection. Bronchograms performed seven years later showed bilateral bronchiectasis. A non-respiratory physician acting for the patient could not support the view that the gassing had caused the lung disease and the case was not pursued.

COMMENT

These cases outline some of the hazards of acute inhalation of irritant gases in an industrial setting. A resumé of the current literature on specific aspects of acute effects of chlorine gas inhalation is given below.

Pathophysiology

Irritant gases such as sulphur dioxide and ammonia are highly water-soluble and tend to dissolve in the upper airways. Ozone and phosgene are much less soluble and tend to cause damage to the more peripheral airways and alveoli. The whole of the respiratory tract is affected by chlorine inhalation as it is of intermediate solubility.

The *post-mortem* appearance of the lungs of persons who have died following acute chlorine exposure show pulmonary oedema, denudation of alveolar and bronchial epithelium and intravascular thrombi within the pulmonary vasculature.¹

Signs and symptoms

The initial reports on the respiratory effects of high-dose inhalation of irritant gases such as chlorine appeared shortly after the cessation of the First World War. The acute effects, such as tracheitis, cough and transient dyspnoea, were appreciated quickly. It gradually became apparent, however, that the lungs could be permanently affected, manifesting clinically as a chronic suppurative cough complicated by wheezes and breathlessness of variable intensity.^{2,3}

Halton Hospital, Runcorn,
Cheshire WA7 2DA, UK
JG Williams

Accepted 29 January 1997

Figure

Acute inhalation of high-dose irritant gases followed by an asthmatic-type illness has been called reactive airways dysfunction syndrome.⁴ This syndrome has been described after an acute inhalation of chlorine gas and patients may have symptoms for several years after the incident.^{5,6} Bronchial biopsy of affected subjects shows changes compatible with 'ordinary' asthma. However, immunohistological studies showed fewer T-lymphocytes and excess activated eosinophils, suggesting that cell-mediated mechanisms were not involved.⁷

Patients with pre-existing pulmonary disease or who are current smokers may have more protracted symptoms than healthy non-smokers.⁸⁻¹⁰ The role of atopy in the natural history of reactive airways dysfunction syndrome secondary to chlorine inhalation is unknown.

Lung function abnormalities

A number of studies have measured patients' pulmonary function for varying intervals of time following an acute inhalation accident. Temporary physiological abnormalities have been well-documented in the literature. Some authors have described a temporary obstructive pattern affecting both large and small airways,^{11,12} while others detected a mixture of both restrictive and obstructive spirometric patterns associated with abnormalities of gas exchange.^{13,14} These abnormalities resolved with time, usually within three months. These findings led to the belief that chlorine inhalation was not associated with long-term effects.

Studies of pulmonary function over a period of several years following acute high-dose inhalation have also been performed. People involved in two separate accidents in which chlorine gas had been released accidentally in a civilian setting have been studied by the same unit. No evidence was found for an increased annual decline in lung function in patients who should have been the most severely at risk and who were investigated for up to seven years.^{15,16} However, other investigators studying patients following a similar accident detected persistent abnormalities of pulmonary function, namely, a significant decrease in the total lung capacity, functional residual capacity, vital capacity and transfer factor accompanied by a significant increase in residual volume and airway resistance in affected individuals over a two-year period.¹⁷ The decreased residual volume has been reported in other studies.^{6,16} A long-term (12 year) study of 13 construction workers working in a pulp mill factory who were exposed to a high concentration of chlorine gas in 1975 demonstrated that, after the initial increase immediately post-exposure, there was a progressive decline in residual lung volume, in contrast to the expected increase with increasing age. The authors postulated that the decreased residual volume was due to either peribronchial fibrosis or diffuse interstitial fibrosis, undetectable on plain chest X-ray.¹⁸ Animal studies seem to favour the former argument.¹⁹ Furthermore, five out of the 13 patients had increased bronchial hyperreactivity. The degree of air trapping within 24 hours of exposure was directly associated with airway hyperreactivity 12 years later. Without pre-exposure spirometric measurements, however, it was impossible to determine whether the chlorine exposure led to the development of bronchial hyperreactivity or the initial air trapping was due to the presence of hyperreactive airways prior to the accident.

Exposure levels

- chlorine gas can be detected at 0.5 ppm
- levels above 20 ppm cause respiratory damage
- levels above 40 ppm may cause pulmonary oedema
- gas is visible at 20 000–30 000 ppm
- recommended exposure limits: 1 ppm per 15-min sampling period, 0.5 ppm per 8-h sampling period

The different conclusions drawn from these studies are difficult to explain. The circumstances surrounding each incident were different. It has been suggested that the major difference between industrial populations who work with chlorine and community residents who have a single exposure is that the industrial workers are at risk of repeated exposure incidents against a background of very low exposure. It is possible that an inflammatory response persists after an acute gassing in the worker, having had no chance to resolve because of the continuous low level exposure.

Effect of treatment

There is no agreed specific antidote for the effect of chlorine inhalation. Supportive treatment seems to be the consensus view. The effect of the immediate treatment of the inhalation injury on the long-term sequelae of acute chlorine poisoning is unknown. The case report of two sisters who were treated in differing ways following the same inhalation accident makes interesting reading.²⁰ The woman treated with corticosteroids fared much better than her sister who was not. There is no other such evidence for the benefits of steroids or any other drug in preventing long-term pulmonary damage.

Repeated inhalations of chlorine gas

CASE REPORT 3

A 71-year-old man was seen at the Chest clinic. He had suffered from a cough and wheeze without sputum for many years, despite being a life-long non-smoker. The family was adamant that his asthma started when he worked in the chemical industry. He was subjected to frequent chlorine gassings at work which was accepted as part of the job when he was a young man. These were treated at the works medical centre with linctus and a cup of tea. He had never required hospitalisation. His spirometric measurements performed at the end of his working career were of an obstructive pattern (FEV₁/FVC 58%) with 15% reversibility. Were his chest symptoms due to the gassings and noxious fumes that he had inhaled during his working life?

COMMENT

Many industrial process workers complain that their chronic chest disease is due to repeated gassings at work. The vast majority of these people smoke heavily and are unlikely to be labelled as having industrial lung disease. Recently, however, there have been several studies conducted to determine whether repeated small gassings can cause long-term pulmonary problems.

Cross-sectional studies

The majority of these studies have been performed on pulp mill workers. Chlorine and chlorine dioxide are used in the bleaching process to create pulp suitable to produce paper. It is accepted that accidental leakage does occur from time to time despite the best efforts. In one study 60% of pulp mill workers reported one or more episodes of accidental chlorine exposure even though the average chlorine level was within recommended levels. This study detected an increased prevalence of airflow obstruction and respiratory symptoms in those subjects who had been repeatedly gassed.²¹ An earlier study had detected a decreased maximal mid-expiratory flow rate in chlorine workers which could not be attributed to smoking habits, despite exposure limits of chlorine of less than 1 ppm.²²

Longitudinal studies

A longitudinal study of workers exposed over an eight-year period²³ indicated that those subjects who had attended the first-aid units following gassing episode(s) had a greater prevalence of respiratory symptoms and a greater annual decline in the FEV₁ and FEV₁/FVC ratio than their non-gassed controls. The annual decline in FEV₁ of subjects repeatedly gassed was greater than anticipated in two different studies, although the rate of decline varied from 29 ml/year²⁴ to 290 ml/year.²⁵

Prognosis

The prognosis of subjects removed from a high-risk gassing environment can best be described as guarded. Construction workers who were exposed intermittently to unacceptably high concentrations of chlorine gas over a three to six month period were studied in detail 18 to 24 months after exposure ended.^{26,27} A total of 257 workers reported an average of 24 exposure episodes during the period of accidental leakage; 60% of the workers described a flu-like

Respiratory sequelae of chlorine inhalation

Acute

- pulmonary oedema
- tracheobronchitis
- temporary airflow obstruction

Chronic

- reactive airways dysfunction syndrome
- bronchiectasis
- accelerated decline in lung volumes
- increased airways resistance

syndrome lasting for 11 days after an acute exposure in addition to the well-recognised irritant effects on the mucosa of the eyes and upper respiratory tract. Within a group of 71 subjects who had "dyspnoea one month or less after the exposure period had ended and those with persistent dyspnoea and/or abnormal lung sounds" 82% had persistent respiratory symptoms, 23% FEV₁ levels less than 80% of predicted and 41% had increased bronchial hyperresponsiveness.

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