Original Article

Acute Accidental Exposure to Chlorine Gas: Clinical Presentation, Pulmonary Functions and Outcomes

Alladi Mohan,[1] S. Naveen Kumar,[1] M.H. Rao,[2] S. Bollineni[1] and I. Chiranjeevi Manohar[1]

Division of Pulmonary, Critical Care and Sleep Medicine, Department of Medicine,[1] and Department of Anesthesiology and Critical Care Medicine,[2] Sri Venkateswara Institute of Medical Sciences, Tirupati (Andhra Pradesh), India

ABSTRACT

Background. To study the clinical presentation, pulmonary functions and outcomes in subjects who were accidentally exposed to chlorine gas.

Methods. Prospective observational study of 64 patients who sustained acute accidental exposure to chlorine gas during a leak in the chlorination system of the public bathing pool of a temple.

Results. The major presenting symptoms and signs included acute dyspnoea (100%), chest discomfort (100%), cough (97%), eye irritation (88%), giddiness (72%), vomiting (46%), and heaviness in the head (44%); tachycardia (100%), tachypnoea (96%) and polyphonic wheezing (28%). All patients were managed in the emergency room with humidified oxygen inhalation and beta-2 agonist nebulisation and 52 were discharged within six hours. Twelve patients were severely affected and required hospitalisation; three of them were admitted into the intensive care unit. Three patients developed pulmonary oedema six to eight hours following admission. Pulmonary function testing (n=12) at presentation revealed obstructive defect in eight and mixed obstructive-cum-restrictive defect in four patients. The mean duration of hospital stay was 5.1±2.1 days. None of the patients died. Reactive airway dysfunction syndrome (RADS) was observed in three of the 12 hospitalised patients, who complained of manifested persistent cough that lasted for three months period following discharge. Serial pulmonary functions recovered to normal range by the end of the six months in all patients and remained so at one-year follow-up.

Conclusion. Acute exposure to chlorine gas is an uncommon, but important public health hazard and can cause RADS, acute lung injury and pulmonary function abnormalities , which are reversible on prompt and appropriate management.
[Indian J Chest Dis Allied Sci 2010;52:149-152]

Key words: Chlorine, Pulmonary toxicity, Reactive airways dysfuncion, Acute lung injury.

INTRODUCTION

In several parts of the world, including India, chlorination is the most common procedure employed for disinfection of public water supplies and swimming pools.[1] Consequent to its widespread use, acute exposure to chlorine gas due to malfunctioning, leaking or explosion in chlorine-disinfection installations is a well recognised public health hazard.[2] There have been occasional reports of respiratory hazards of acute chlorine gas inhalation from developed countries[3-5] and India.[6-8]

In India, a public bathing pool (called *Pushkarini*) is commonly present in several temples and is used by visitors for religious beliefs. Chlorination, is the procedure most frequently employed for disinfection and ensuring microbiological safety of these public bathing pools. In this observational study, we describe the clinical presentation and pulmonary function abnormalities following acute accidental exposure to chlorine gas due to malfunctioning of chlorination system of a public bathing pool in a temple. We have also documented the long-term consequences of acute chlorine gas exposure in the hospitalised patients who were followed-up for a period of one year from the time of the incident.

MATERIAL AND METHODS

Accidental emission of chlorine gas occurred in the public bathing pool in a temple as a result of

[Received: July 8, 2009; accepted after revision: April 26, 2010]

A part of this work (Mohan A, Naveen Kumar S, Rao MH. Acute accidental exposure to chlorine gas: clinical presentation, pulmonary functions and outcomes. Proc Am Thorac Soc 2006;3:A202) was presented in the ATS2006 International Conference of the American Thoracic Society, May 19-24, 2006, at San Diego, CA, USA.

Correspondence and reprint requests: Dr Alladi Mohan, Chief, Division of Pulmonary and Critical Care Medicine, and Professor and Head, Department of Medicine, Sri Venkateswara Institute of Medical Sciences, Tirupati - 517 507 (Andhra Pradesh), India, Phone: 91-877-2287777, Extn 2256; Fax: 91-877-2286803; E-mail: alladimohan@rediffmail.com

malfunction of chlorination equipment. The chlorine vapours spread through the environment exposing the pilgrims and devotees who were taking bath as well as their relatives and onlookers who were present in the vicinity of the public bathing pool at that time. While the exact number of persons who were affected is not clearly known, 64 patients who sustained acute accidental exposure to chlorine gas presented to the emergency room seeking medical care. The Institutional Ethical Committee clearance was obtained to carry out this observational study.

In all the patients, a detailed history was taken and a thorough physical examination was carried out. History of previous chronic respiratory disease including asthma, chronic obstructive pulmonary disease, pulmonary tuberculosis, emphysema was also recorded. Smoking history was recorded and the patients were categorised as current smokers, past-smokers or never smokers. All patients were managed in the emergency room with humidified oxygen inhalation and β-2 agonist nebulisation and 52 were discharged within six hours.

Twelve patients were severely affected and required hospitalisation; three of them were admitted into the intensive care unit (ICU). In patients who were admitted to the hospital, haemogram, serum biochemistry, chest radiograph, electrocardiogram (ECG) and arterial oxygen saturation measurement by pulse oximetry (SpO_2) were done at the time of admission. In patients with a SpO_2 value of less than 90%, arterial blood gas (ABG) analysis was done at the time of admission. Thereafter, the SpO_2 was monitored 12-hourly during the period of hospitalisation. Hospitalised patients required intravenous hydrocortisone and aminophylline administration in addition to the above measures. Within 24 hours of admission, pulmonary function testing including testing for reversibility with bronchodilators was carried out in those hospitalised patients using Morgan Transfer Test Benchmark PFT System (Morgan Scientific, Inc., USA) as per the standards suggested by the American Thoracic Society[9] as described previously.[10] In three patients admitted to the ICU, the heart rate, ECG, non-invasive blood pressure measurement and SpO_2 by pulse oximetry were monitored around the clock. Following discharge, the hospitalised patients were evaluated at three monthly intervals for one year.

RESULTS

The mean age was 44.0±6.4 years and there were 26 females among the patients. The major presenting symptoms and signs are presented in table 1. Almost all the patients complained of acute dyspnoea (100%), chest discomfort (100%), and cough (97%). Physical examination revealed polyphonic wheezing in nearly one-third of the patients (28%). Sixteen of the 38 males were current smokers; 11 of them smoked *bidis* and the remaining five were cigarette smokers. All were asymptomatic till the time they were exposed to chlorine gas. All the women and the remaining men were 'never-smokers'.

Table 1. Clinical manifestations in 64 patients with acute accidental exposure to chlorine gas

Variable	% Present
Symptoms	
Acute dyspnoea	100
Chest discomfort	100
Cough	97
Eye irritation	88
Rhinorrhoea	78
Giddiness	72
Vomiting	46
Heaviness in the head	44
Sore throat	36
Stridor	06
Signs	
Tachycardia	100
Tachypnoea	100
Use of accessory muscles of respiration	96
Polyphonic wheezing	28
Irregular pulse	01*

More than one symptom or sign were present in several patients.
** Electrocardiogram revealed ventricular ectopics*

The 12 severely affected patients requiring hospitalisation complained of chest pain and severe dyspnoea; three of them who manifested severe bronchospasm were admitted into the ICU. Their mean age was 32.8±12.1 years; there were four females. All of them were 'never smokers'. The haematological and biochemical parameters were with-in normal limits in all the patients at baseline and remained so during the follow-up period. The base-line haematological parameters (mean± standard deviation [SD]) were as follows: haemoglobin (g/dL) 12.9±1.4; total leucocyte count (/mm^3) 11733±4408; neutrophils (%) 64.5±4.3; lymphocytes (%) 26.8±2.9; eosinophils (%) 1.2±1.1; platelet count (/mm^3) 208583±43271. The biochemical parameters on admission (mean ± SD) were as follows: random blood sugar (mg/dL) 101.7±12.0; blood urea (mg/dL) 23.9±5.8; serum creatinine (mg/dL) 1.0±0.2; serum bilirubin (mg/dL) 0.9±0.2; serum alanine trasnaminase (IU/L) 31.8±8.4; aspartate aminotransferase (IU/L) 34.8±7.3. The ECG was normal in all except one patient who had recurrent ventricular ectopics during the first 24 hours.

Pulmonary function testing (n=12) at presentation (Table 2) revealed obstructive defect in eight and mixed obstructive-cum-restrictive defect in four. In patients with mixed obstructive-cum-restrictive defect

at admission, the forced vital capacity (FVC) (% predicted) (mean±SD=62.8±6.2), and total lung capacity (TLC) (% predicted) at admission (mean±SD =71.8±1.7) were low. In patients with obstructive defect, the FVC and the TLC were within normal limits. Reversibility testing revealed reversible airflow obstruction in three patients.

Table 2. Pulse oximetry and spirometry findings in 12 hospitalised patients with acute chlorine gas exposure at admission, three months and six months follow-up

Variable*	At Admission	At 3 Months Follow-up	At 6 Months Follow-up
Pulse oximetry†			
SpO_2 (%)	94.2±4.9 (86-99)	97.3±1.4 (95-99)	98.2±0.75 (97-99)
Pulmonary functions			
FEV_1‡	49.0±8.8 (35-59)	79.6±12.9 (59-95)	86.2±4.3 (80-92)
FVC‡	77.3±11.5 (58-89)	87.6±4.7 (82-99)	88.3±3.6 (84-96)
FEV_1/FVC (%)	54.4±5.0 (47-64)	77.3±11.1 (57-90)	83.1±2.5 (81-89)
PEFR‡	30.8±5.8 (22-42)	60.8±11.4 (38-77)	81.5±1.9 (79-85)
FEF_{25-75}‡	29.9±6.3 (22-43)	62.5±15.1 (31-80)	85.6±3.5 (81-90)

***** All data expressed as mean±standard deviation (range)

† At admission, pulse oximetry revealed arterial hypoxaemia in three patients.

‡ Expressed as % predicted

SpO_2=Arterial oxygen saturation measured by pulse oximetry; FEV_1=Forced expiratory volume in the first second; FVC=Forced vital capacity; FEF_{25-75}=Forced expiratory flow at 25%–75%; PEFR=Peak expiratory flow rate; PaO_2=Partial pressure of arterial oxygen; $PaCO_2$=Partial pressure of arterial carbon dioxide; HCO_3^-=Arterial bicarbonate level.

In the three patients who were admitted to the ICU, SpO_2 measurement by pulse oximetry revealed arterial hypoxaemia. The baseline SpO_2 findings and arterial blood gas analysis results in these three patients are as follows: SpO_2(%)=88, pH=7.39, PaO_2=54 mmHg, $PaCO_2$=42 mmHg, HCO_3^-=24 meq/L; SpO_2(%)=86, pH=7.42, PaO_2=51 mmHg, $PaCO_2$=38 mmHg, HCO_3^-=22 meq/L; SpO_2 (%)=86, pH=7.39, PaO_2=51 mmHg, $PaCO_2$=40 mmHg, HCO_3^-=25 meq/L. The SpO_2 values became normal by the second day with supportive management. Three of the admitted patients developed pulmonary oedema six to eight hours following admission and required intravenous frusemide administration. Mean duration of hospital stay was 5.1±2.1 days. None of the patients died.

Serially repeated pulmonary functions (Table 2) returned to normal range by three months in nine patients. Reactive airway dysfunction syndrome (RADS)[11] was observed in three of the 12 hospitalised patients, (all 'never-smokers') who presented with reversible obstructive defect and these patients complained of manifested persistent cough that lasted for three months period following discharge. These patients were treated with inhaled salbutamol and budesonide. In these three patients, repeat pulmonary functions became normal by the end of six months of follow-up (Table 2). At one-year of follow-up, all the patients were asymptomatic and continued to have normal pulmonary functions.

DISCUSSION

Chlorine is a yellow-green gas with a pungent, irritating odour. It is slightly water soluble, is about two times as heavy as air and is a strong oxidising agent. Sources of exposure to chlorine gas include accidental releases of chlorine vapour at swimming pools, improper mixing of hypochlorite bleach with acidic cleaning agents, mixing of ammonia and hypochlorite bleach (forming chloramine gas), school chemistry experiments, and industrial or chemical transportation accidents.[1,2] Chlorine gas is a toxic inhalant and is classified as a pulmonary irritant. As it has high reactivity with water, acute exposure to chlorine gas may initially cause eye and throat irritation. Bronchoconstriction or tracheobronchitis that may manifest as cough, wheezing and breathlessness of variable intensity may then develop. These manifestations may be transient. Subsequently, delayed pneumonia, acute lung injury (ALI), acute respiratory distress syndrome (ARDS)[12] and death may ensue.

Clinical manifestations of acute chlorine gas exposure have been documented in some studies published more than a decade ago from India.[6-8] In the report from New Delhi,[6] people were exposed to chlorine gas when a cylinder containing liquid chlorine under pressure started leaking while in transit. In the accidents that occurred at Mumbai (then called Bombay),[7] chlorine leaked from a storage tank at a factory manufacturing chemicals. In contrast, accidental exposure to chlorine occurred at a public bathing tank in a temple in the present study. Given the fact that several million pilgrims and devotees all over India take a holy dip in such public bathing tanks, acute accidental chlorine gas exposure can be considered as a potentially avoidable public health disaster that should be watched for.

Majority of the patients in the present study presented with irritating cough, acute onset dyspnoea, eye and throat irritation being the most common presenting symptoms. In other published studies[6-8] on acute exposure to chlorine gas from India, similar symptomatology has been documented. Three of the 64 patients evaluated (5%) in the present study developed pulmonary oedema six to eight hours following admission. In the report from Mumbai,[7] where patients were exposed to at least 66 ppm of chlorine, two of the 82 patients (2.4%) had developed pulmonary oedema in the first eight hours following exposure. In another report,[13] pulmonary oedema was reported in 85% of the patients when the exposure level was 400 ppm. Mortality in acute exposure to chlorine gas has been attributed to ALI, ARDS; other mechanisms such as diffuse bronchitis, arrhythmias, also contribute.[1,6,5,12] Prompt relief of hypoxaemia with oxygen administration, use of

nebulised β-2 agonists and assisted ventilation when required can be life saving in ALI due to chlorine gas inhalation. Beneficial effect with parenteral corticosteroid administration in patients with severe manifestations has been documented by some workers[5,7] but needs further study.

Obstructive, and mixed obstructive-cum-restrictive pulmonary function abnormalities have been described in patients acutely exposed to chlorine gas in previously published studies[6-8] from India. Similar observations were made in the present study. In majority of the studies,[6-8] these abnormalities have been observed to be short-lived. Several factors, such as duration of exposure, concentration of chlorine gas, tobacco smoking status, presence of co-existing co-morbid conditions such as cardiac and lung disease, history of previous occupational exposure, among others have been thought to influence the long-term pulmonary consequences. In the present study and the other published studies from India,[6-8] long-term pulmonary consequences were not observed. These issues also merit further study.

In the present study, in three patients without a preceding history of respiratory complaints, chest symptoms developed within 24 hours of a single specific acute exposure to chlorine gas. Other types of pulmonary disease were ruled out in these patients. Pulmonary function testing in these patients revealed reversible airflow obstruction; methacholine test, however, was not carried out. These patients manifested symptoms that simulated asthma, such as persistent cough, wheezing, chest tightness and dyspnoea that persisted for at least three months. These three patients satisfy all the criteria for reactive airway dysfunction syndrome (RADS) as defined by Brooks,[11] except a positive methacholine test. In a recent systematic review,[14] acute chlorine gas exposure has been found to be an important cause of RADS.Observations from the present study also indicate that acute chlorine gas exposure is an under-recognised cause of RADS in India. Acute exposure to inhalational toxins is emerging as an important public health hazard in India. Recent times have witnessed several such episodes of chlorine gas leaks not only in other parts of the world,[3-5] but also from several parts of India,[15] sometime with fatal outcomes. Clinical observations from these incidents have seldom been published in medical literature suggesting that acute toxic inhalational lung injury, particularly due to chlorine gas inhalation is an under-recognised problem in India. A systematic study of the consequences of acute chlorine gas inhalation is warranted.

CONCLUSIONS

Acute exposure to chlorine gas is an uncommon, but important public health hazard and can cause RADS, acute lung injury and pulmonary function abnormalities. The kind of exposure reported in the present study is unique and should caution physicians, chest physicians, critical care experts and intensivists regarding such a health hazard during fares and fetes that are commonly conducted all over India. These consequences may be reversible with timely and appropriate management.

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