RESEARCH ARTICLE

Chlorine gas hazardous material incident in Karachi, Pakistan: A clinical experience from an emergency department of a tertiary care hospital

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Abstract

Objective: To determine the clinical characteristics, management and outcomes of patients presenting with chlorine gas exposure in an emergency setting.

Method: The single-centre, retrospective cross-sectional study was conducted at the emergency department of Aga Khan University Hospital, Karachi, and comprised data of all patients who presented on March 06, 2020, due to acute chlorine gas exposure after a specific industrial accident. Demographic and clinical data was recorded from the medical record files. The association between risk factors and complications was explored. Data was analysed using SPSS 20.

Results: There were 51 male patients with a mean age of 33.10±8.37 years. The most commonly affected organ system was respiratory 49(96%), with 43(84.3) having shortness of breath. Eye irritation was found in 44(86.3%) cases and the central nervous system was involved in 14(27.4%). Most of the patients were admitted from the emergency department 36(70%). Regarding treatment, 1(1.9%) patient each required invasive and non-invasive mechanical ventilation. Complications included toxic pneumonitis 3(5.9%) and pneumomediastinum 1(1.7%). No correlation was found between smoking and complications (p>0.05).

Conclusion: Most patients showed complete resolution of symptoms after receiving supportive treatment, while complications were rare and there was no mortality.

Keywords: Chlorine gas, HAZMAT incident, Emergency department. (JPMA 73: 834; 2023)

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Introduction

Hazardous materials (HAZMAT) releases are of growing concern due to increase in population density and accelerating industrial development. Accidental or disaster-related toxic gas releases may result in significant public health consequences.[1] Chlorine gas is one of the most important industrial chemicals used in the production of thousands of products. The effects on the health of chlorine inhalation depend on the chlorine concentration and duration of exposure.[2] At low concentration (<30ppm), chlorine gas is known to cause mild irritation of mucous membranes, coughing, choking and shortness of breath. Exposure to high concentrations (>30ppm) may damage the lung parenchyma, causing complications, such as rapid development of interstitial pneumonia, pulmonary oedema, and death due to progressive respiratory failure.[3]

Humans can encounter chlorine gas during short-term, high-level exposures due to traffic or rail accidents, spills, or other disasters. By contrast, some work and public places, like swimming pools, are known for long-term, low-level exposures with higher frequency.[4]

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Incidents of large-scale chlorine gas exposure due to industrial accidents have been reported from both the developed and the developing countries.[5,6] On Friday, March 6, 2020, at 10:10am, there was an isolated incident of chlorine gas release through the vents of a chemical plant at Port Qasim in Pakistan industrial hub, Karachi. Due to swift identification of the incident and a rapid response by the relevant staff, the leak was quickly contained, and the affected people were immediately taken to nearby medical facilities for necessary assistance.[7]

Currently, to the best of our knowledge, there is no clinical study available on HAZMAT incidents involving chlorine gas in Pakistan. The current study was planned to fill the gap by determining the clinical characteristics, management and outcomes of patients presenting with chlorine gas exposure in an emergency setting.

Materials and Methods

This single-centre, retrospective, cross-sectional study was conducted in May 2020 at the emergency department (ED) of Aga Khan University Hospital (AKUH), Karachi, and comprised data of all patients who were brought there on March 6, 2020, with acute chlorine gas exposure. After exemption from the institutional ethics review committee, data was collected from the medical record files and electronic health records without identifying the patients.

The medical record numbers were obtained from triage data. Files were reviewed by two senior emergency medicine residents. Demographic and clinical data, including age, gender, signs and symptoms, triage category, co-morbidity, allergy, smoking status, treatment provided, complications, and disposition was recorded using a standardised form. Triage had been done as per the Emergency Severity Index criteria (ESI), which is a five-level tool that stratifies the patients into five groups from 1 - most urgent to 5=least urgent based on acuity and resource needs.[8] Outcomes had been reported as hospitalisation or discharge from the ED.

Data was analysed using SPSS 20. Data was expressed as mean and standard deviation Or frequencies and percentages, as appropriate. Chi-square test was applied to examine the association between the risk factors and complications P<0.05 was considered statistically significant.

Results

Of the 137 chemical plant workers affected during the incident, 51(37.22%) were brought to the AKUH; all (100%) were males. The mean age was 33.10±8.37 years (range: 21-55 years), 23(45.1%) patients were smokers, 26(51%) were in triage category P3, and 36(70.6%) were admitted, and the mean length of hospital stay was 1.47±1.56 days (range: 01-11 days) (Table 1). The most commonly affected organ system was respiratory 49(96%), with 43(84.3) having shortness of breath. Eye irritation was found in 44(86.3%) cases and the central nervous system (CNS) was involved in 14(27.4%) (Table 2).

On physical examination, 29(56%) patients were tachypnoeic (respiratory rate >20/min), 12(23.5%) were tachycardic (heart rate >100/min), and 20(39%) had elevated blood pressure (BP) (systolic blood pressure [SBP] >140mmHg or diastolic blood pressure [DBP] >90mmHg). Electrocardiogram was done in 43(84.3%) cases, and 3(5.9%) had sinus tachycardia. Regarding laboratory investigations, arterial blood gas (ABG) was tested in 33(64%) patients. Respiratory acidosis with potential of hydrogen (pH) <7.35 and partial pressure of carbon dioxide (pCO_2) >45mmHg was found in 5(10%) patients, while 9(17.6%) showed hypoxaemia with partial pressure of oxygen (pO_2) <65mmHg. Chest X-ray was done in 41(80%) cases.

Complications included toxic pneumonitis 3(5.9%) and pneumomediastinum 1(1.7%), with 1(1.9%) toxic pneumonitis patient requiring non-invasive mechanical ventilation. The patient with pneumomediastinum required invasive mechanical ventilation and bilateral chest tube insertion. No correlation was found between smoking and complications (p>0.05).

Regarding treatment, 40(78.4%) patients received humidified oxygen, 45(88.2%) received nebulisation with salbutamol and ipratropium, 25(49%) received intravenous (IV) steroids, and 4(7.84%) received nebulisation with sodium bicarbonate (Table 3).

Table-1: Demographics, co-morbidity, and outcome of patients with chlorine gas intoxication (n=51).

Characteristics	Results
Mean Age (years)	33.10±8.37 (21-55)
Gender [n (%)]	
Male	51 (100)
Female	00 (00)
Co-morbidity [n (%)]	
Diabetes Mellitus	01 (2)
Hypertension	04 (7.8)
Chronic Obstructive Pulmonary Disease	01 (2)
Smoking history	23 (45.1)
Triage Category	
P1	02
P2	23
P3	26
P4	00
P5	00
Disposition of patients from ED [n (%)]	
Admitted to general care	29 (56.9)
Admitted to monitored bed	06 (11.8)
Admitted to ICU	01 (02)
Discharged	11 (21.6)
Left against medical advice	04 (7.8)
Mean Hospital stay (days)	1.47±1.56 (01-11)

Table-2: Common presenting symptoms.

Organ system	n (%)
Respiratory system	49 (96)
Shortness of breath	43 (84.3)
Cough	36 (70.5)
Chest heaviness	22 (43.1)
Sore throat	09 (17.6)
Eye irritation	44 (86.3)
Neurological system	14 (27.4)
Drowsiness	09 (17.6)
Headache	05 (9.8)
Gastrointestinal system	07 (13.7)
Nausea and Vomiting	06 (11.7)
Diarrhoea	02 (3.9)

Table-3: Treatment of the victims (n=51).

Characteristics	n(%)
Humidified oxygen	40 (78.4)
Nebulisation	
-Salbutamol and ipratropium	45 (88.2)
-Sodium bicarbonate	04 (7.8)
Intravenous steroids	25 (49)
Non-invasive mechanical ventilation	01 (1.9)
Invasive mechanical ventilation	01 (1.9)

Discussion

All the patients in the current study had unintentional chlorine gas exposure at workplace. The respiratory tract was the most affected organ system. The majority of the patients showed complete resolution of symptoms after receiving symptomatic treatment with humidified oxygen, bronchodilators, and IV steroids. Four patients developed complications and only 2 required mechanical ventilation. There was no mortality.

The most common presenting symptoms were shortness of breath, eye irritation, cough, and chest tightness. Similar results have been reported earlier.[9,10] Chlorine is a respiratory irritant and its effects depend on its concentration, as well as the duration of exposure and the water content of the tissue involved.[11] At low concentration (0.2-15ppm), this can cause mild to moderate mucosal irritation. Severe symptoms, such as cough, shortness of breath, chest tightness, and toxic pneumonitis, occur at concentrations >30 ppm.[12] In the current study, 3 patients developed toxic pneumonitis, and 1 required non-invasive mechanical ventilation.

Pneumomediastinum is a rare complication of acute chlorine gas toxicity.[13,14] It occurs due to an increase in pressure gradient between the alveoli and the interstitium, resulting from severe strain or cough. This pressure gradient leads to the rupture of marginal alveoli, resulting in the dissection of the air along the pulmonary vascular sheaths into the mediastinum.[15] Pneumomediastinum was found in only 1 patient in the current study. On arrival, the patient was in severe respiratory distress with 35 breaths per minute. His ABG showed severe respiratory acidosis (pH: 6.89; PCO_2: 139mmHg). The patient required invasive mechanical ventilation and bilateral chest tube insertion. Of all the 4 patients with complications, only 1(25%) with pneumomediastinum had a positive smoking history. In contrast, studies have shown that smokers with chlorine inhalation tend to have a greater risk for upper airway obstruction and are vulnerable to complications.[16-18]

Around 27% of the patients in the current study had neurological symptoms, including headache, light-headedness and drowsiness. These symptoms are commonly reported following acute high exposures to chlorine, and are thought to be due to asphyxia induced by chlorine.[19] Gastrointestinal symptoms, such as nausea, vomiting and diarrhoea, were reported in 13.7% patients. These symptoms are believed to be reflex reactions, and not a specific effect of chlorine.[20] The present study showed a higher incidence of neurological and gastrointestinal symptoms compared to other studies that showed symptoms in 2% and 6% of patients, respectively.[11]

In this study, most of the chest radiographs were normal, but 4 radiographs showed pathological findings; bilateral heterogeneous airspace shadowing in 3 and pneumomediastinum in 1. Similar results were seen in a study where six out of 76 patients had abnormal chest radiograph findings, including heterogeneous density increase in the inferior zones bilaterally).[21] A baseline chest radiograph should be obtained in patients with acute chlorine gas toxicity if the patient is tachypnoeic, and respiratory functions should be monitored by pulse oximeter and ABGs.[22] Chest radiographs can be normal, or they may show diffuse nodular infiltrates, bilateral airspace shadowing, and signs of vascular congestion.[23]

There is no specific antidote for the acute inhalation injury caused by chlorine gas. It is important to immediately stop gas exposure and remove the patient from the toxic environment, followed by decontamination. Humidified oxygen is used as a primary treatment as it is less irritating to the airways than non-humidified oxygen.[24] Nebulisation with bronchodilators should be given for bronchospasm to reduce airway resistance. Salbutamol alone or in combination with ipratropium can also be used.[25] In the current study 78.4% received humidified oxygen and 88.2% received nebulisation with salbutamol and ipratropium. Nebulised sodium bicarbonate is used after chlorine gas inhalation as it can counteract hydrochloric acid, but no clear evidence of clinical improvement has been shown.[26] Only 4 patients received nebulisation with sodium bicarbonate in the current study.

In the current study, 49% patients received IV steroids with humidified oxygen and bronchodilators. None of the patients received oral or inhalation steroids. The role of steroids in inhalational trauma due to chlorine gas exposure is controversial. Neither IV nor oral or inhaled steroids have shown any negative impact on morbidity and mortality.[12] There is no clear benefit of steroids alone as they are not given without oxygen or bronchodilators.[27]

The current study has a few limitations. It was not possible to identify the exact concentration of chlorine gas that caused complications in 4 patients. Data on other factors that influence the exposure level of individual victims, such as the location of the victims and the direction of the wind could not be collected. Besides, it was unable to perform pulmonary function tests on the victims at admission and discharge, and that is why the study lacks data on overall damage caused by chlorine gas poisoning.

Conclusion

Chlorine gas incidents are among the top HAZMAT incidents. It negatively affects airways and breathing. There is no known antidote. Most of the patients showed

complete resolution of symptoms after receiving supportive treatment. The development of complications was rare, and there was no mortality.

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