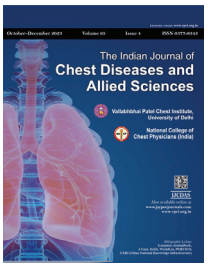
CASE REPORT

A Rare Case of Chlorine Inhalation-induced Severe Acute Respiratory Distress Syndrome: A Case Report and Review of Literature



This article is available on www.vpci.org.in

Arjun Kumar[1], Ashok K Janmeja[2], Rohit Manchanda[3], Balbir Singh[4]

Received on: 03 January 2024; *Accepted on:* 08 February 2024; *Published on:* 26 March 2024

Abstract

Chlorine (Cl_2) exposure-induced acute respiratory distress syndrome (ARDS) is rarely reported in clinical practice. Here, we report a case of a 45-year-old male patient presented with complaints of sudden onset of cough and breathing difficulties, which started after accidental inhalation of fumes coming from bleaching powder. The patient developed chlorine inhalation-induced ARDS and severe hypoxemia. The patient was well managed with early systemic corticosteroids along with bronchodilator and other supportive care. Hence, supportive therapy along with early systemic corticosteroid can be helpful in moderate-to-severe chlorine inhalation-induced ARDS patients requiring mechanical ventilation support, if carefully selected.

Keywords: Acute respiratory distress syndrome, Case report, Chlorine inhalation, Corticosteroid, Ventilation support.

The Indian Journal of Chest Diseases and Allied Sciences (2023): 10.5005/jp-journals-11007-0096

Abbreviations used in this Article

ABG = Arterial blood gas; ARDS = Acute respiratory distress syndrome; BiPAP = Bilevel positive airway pressure; CXR = Chest X-ray; HRCT = High resolution computed tomography; ICU = Intensive care unit; PCT = Procalcitonin; RADS = Reactive airways dysfunction syndrome; SpO_2 = Peripheral artery oxygen saturation; VALI = Ventilator-associated lung injury.

Introduction

Chlorine (Cl_2) is a dense, highly reactive, irritating, pungent smelling gas, which is non-combustible and moderately soluble at room temperature.[1] It is responsible for upper and lower airway injuries when inhaled in mild concentration.[2] Chlorine can be dangerous for even shorter period exposure at concentrations of 40–60 ppm, leading to irritation of eyes, nose, increased mucus production, airway resistance, and breathing difficulties in humans.[2–4] At concentration above 1000 ppm, it can penetrate into distal lung structures and damage alveolar epithelium and surfactant producing cells, resulting in severe hypoxemia, pulmonary edema, and even rapid death due to fatal asphyxia.[2–4]

Chlorine was used as a chemical warfare agent during World War I, nowadays, it is frequently used in various industrial products including paper, pulp industry, as bleaching agents and household chemicals, as also used in water purification in city water sources and swimming pools.[1,5] Chlorine inhalation exposures can be due to Cl_2 leaks while handling or transportation of chlorine, chlorine disinfection systems, and improper mixing of ammonia and hypochlorite bleach (forming chloramines gas), accidental explosions, leaks, or malfunction of chlorine containing chemicals and school experiments.[2,6,7]

Symptoms due to chlorine inhalation can vary depending upon concentration and duration of inhaled gas, including eye, throat, and skin irritation, cough, breathing difficulties and strider, chest pain and headache.[5–8] Chlorine exposure-induced acute respiratory distress syndrome (ARDS) is rarely reported in the literature and clinical practice due to its irritating nature.[8,9] However, intense exposure of Cl_2 gas can lead to severe hypoxemia and death if aggressive treatment strategies are not applied. Here we present a case of ARDS caused by chlorine gas exposure and a timely aggressive treatment strategy for better clinical outcome.

Case Description

A 45-year-old never smoker, who works in a water treatment facility for the last 2 years, and having no history of illicit drugs or alcohol intake presented to the emergency department of tertiary care hospital with shortness of breath for 30 minutes, which was acute in onset and progressive in nature. Patient also complains of throat irritation and burning sensation in both the eyes which started within 5 minutes of exposure to fumes coming from bleaching powder [$Ca(ClO)_2$] which he was mixing for water

[1–4]Department of Respiratory Medicine, Maharishi Markandeshwar Medical College and Hospital, Kumarhatti, Solan, Himachal Pradesh, India

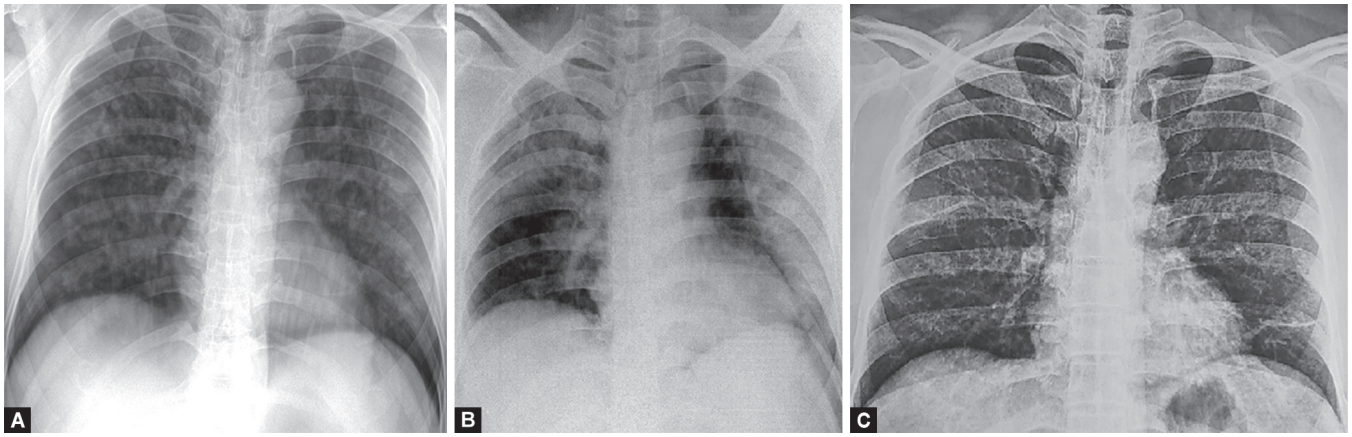
Corresponding Author: Arjun Kumar, Department of Respiratory Medicine, Maharishi Markandeshwar Medical College and Hospital, Kumarhatti, Solan, Himachal Pradesh, India, Phone: +91 8054809188, e-mail: aknlnegi@gmail.com

How to cite this article: Kumar A, Janmeja AK, Manchanda R, *et al*. A Rare Case of Chlorine Inhalation-induced Severe Acute Respiratory Distress Syndrome: A Case Report and Review of Literature. Indian J Chest Dis Allied Sci 2023;65(4):193–196.

Source of support: Nil

Conflict of interest: None

Patient consent statement: The author(s) have obtained written informed consent from the patient for publication of the case report details and related images.



Figs 1A to C: Chest X-ray (CXR) apico-posterior view showing: (A) Day 1—CXR showing diffuse area of patchy, nodular opacification in bilateral lung fields; (B) Day 3—CXR showing increased bilateral diffuse opacities more predominant in peripheral regions; (C) Day 12—CXR showed remarkable improvement compared with previous CXR

purification in the city water source. He mixed around 500 gm of bleaching powder in 6–7 liters of water on that day without wearing any protective mask/cloth as he used to wear normally. Patient also developed a headache and cough associated with expectoration which was minimal in quantity, whitish in color, non-foul smelling and not associated with blood. Patient also complained of six episodes of vomiting before reaching the hospital. He also had similar episodes of irritation in nose and throat in the past also which were minimal and self-limiting. On admission, his peripheral artery oxygen saturation (SpO_2) by pulse oximetry was 77% on room air, pulse rate of 120 bpm, respiratory rate of 24 breath per minute and blood pressure of 116/76 mm Hg. Physical examination revealed bilateral lung crackles in the infra axillary area and infra scapular area. A chest X-ray (CXR) was ordered which was suggestive of a diffuse area of patchy, nodular opacification in bilateral lung fields (Fig. 1). Routine blood investigations including complete blood count and electrolytes, renal function test, biochemistry profiles including liver function test, and coagulation profile were unremarkable. Arterial blood gas (ABG) revealed a pH of 7.40, pCO_2 of 24.2 mm Hg and pO_2 of 48.4 mm Hg, under high flow oxygen via non-rebreathing mask at the rate of 14 liters per minute. The patient was shifted to respiratory intensive care unit (ICU) started on supportive oxygen therapy via non-rebreather mask along with prophylactic broad spectrum antibiotics. For further evaluation, high-resolution computed tomography of chest (HRCT) was performed on the second day of admission and revealed multiple discrete and confluent centrilobular nodules with secondary ground glassing haze diffusely scattered in bilateral lung fields (Fig. 2).

On the 3rd day of admission, the patient developed increased difficulty in breathing and hypoxia even on high flow oxygen via non-rebreathing mask at the rate of 15 liters per minute, having oxygen saturation (SpO_2) of 85%. Furthermore, his ABG showed respiratory acidosis with severe hypoxemia. Repeat CXR was ordered which showed increased bilateral diffuse opacities more predominant in peripheral regions compared with the first day (Fig. 1). Bed side 2D echocardiography was normal. Repeat routine blood investigations including complete blood count, troponin T, D-dimer (342 ng/mL), serum procalcitonin (PCT) level (0.27 ng/mL) blood, urine, and sputum culture were all unremarkable except mild increased peripheral white blood cell count (11,700/µL). The patient was started on a bilevel positive airway pressure (BiPAP) respiratory support for 16–18 hours per day and intravenous hydrocortisone 100 mg 8 hourly under the cover of upgraded antibiotic, that is Piperacillin (4000 mg) + Tazobactam (500 mg) along with nebulization with bronchodilators and corticosteroid.

Patient started showing clinical improvement after 48 hours of corticosteroid initiation and BiPAP support was given intermittently and oxygen requirement also decreased to 8 liter per minute.

On 8th day of admission, he was completely weaned off from BiPAP support, and his ABG showed improvement and maintained oxygen saturation of 94% on 2 liter/minute oxygen via nasal prongs. By the end of 12th day his oxygen saturation was 93% on room air and CXR showed remarkable improvement compared to previous CXR (Fig. 1). Patient was shifted to oral prednisone 20 mg per day which was tapered off in the next 5 days and discharged to home under stable condition. He is currently doing well without any clinical and radiological sequelae.

Discussion

Chlorine is a greenish-yellow, dense, toxic and highly reactive, irritating, pungent smelling gas, which was discovered first time in discovered in year 1772.[1] Chlorine is used in both households and industrial fields, and it has also been used as a chemical warfare weapon agent during World War I.[1,5]

Humans can be exposed to chlorine inhalation by different types of settings including swimming pool chlorine gas inhalation, industrial mishaps, domestic exposure, water purification or leaks while transportation, chemical warfare agent, etc.[3,6–8] Chlorine is an extremely water soluble gas therefore accidental exposure primarily affects the exposed body parts including skin, eyes, upper and larger airways. Initial symptoms especially on exposure to low concentrations of chlorine (1–10 ppm) lead to burning sensation over exposed skin, eye and nasal irritation, headache, sore throat, and coughing. While exposure to higher concentrations of chlorine gas (>15 ppm) can lead to life-threatening complications including ARDS, acute lung injuries, reactive airways dysfunction syndrome (RADS), lung collapse, airway pulmonary edema, hemoptysis, and even death.[6,10,11]

Our patient also complains of throat irritation and burning sensation in both the eyes which started within a few minutes

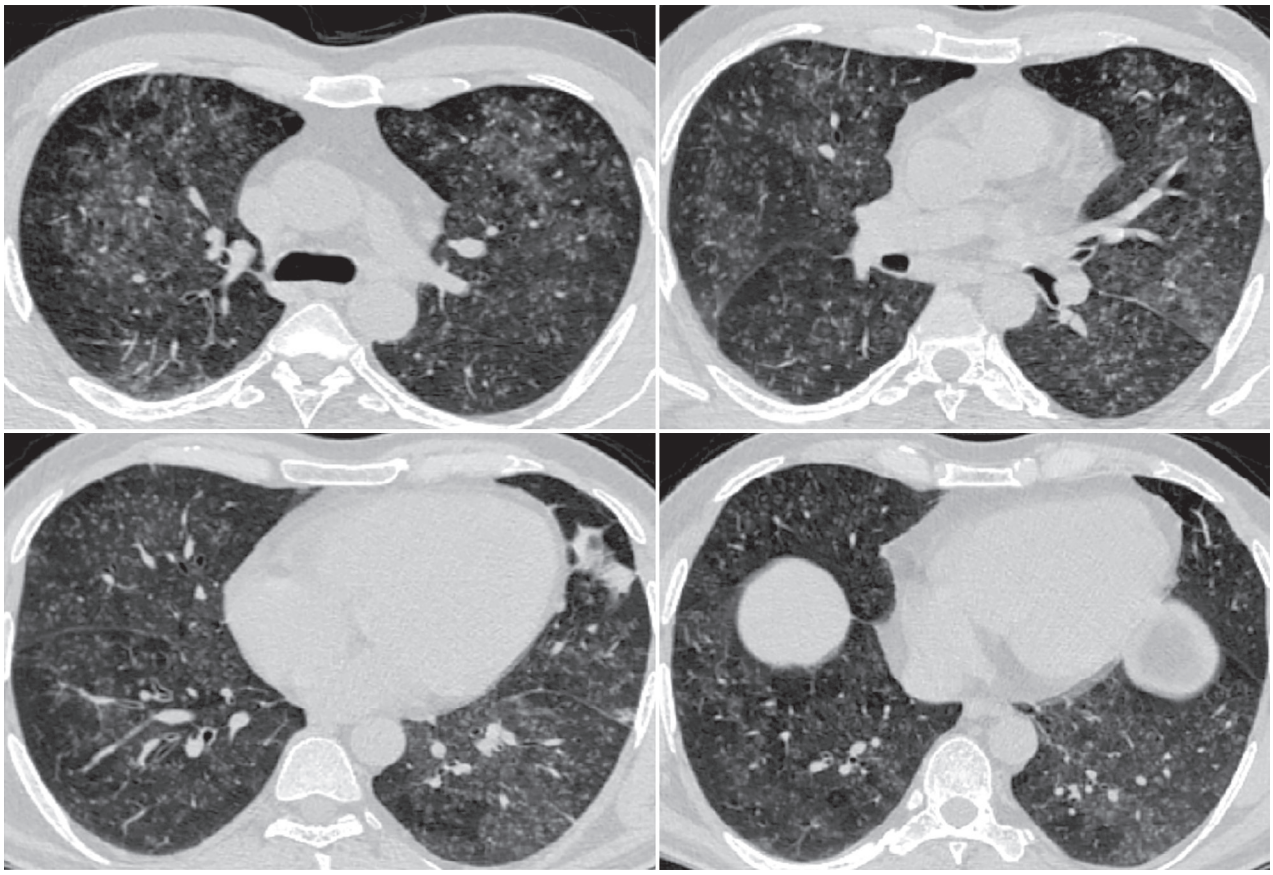


Fig. 2: High-resolution computed tomography of chest (HRCT): showing multiple discrete and confluent centrilobular nodules with secondary ground glassing haze diffusely scattered in bilateral lung fields

of inhalation of fumes coming from bleaching powder [$Ca(ClO)_2$] which he was mixing for water purification in the city water source. There are few case reports on CT findings of acute inhalation injury due to chlorine gas in the literature.[12,13] It has been found that the site of inhalation injury due to chlorine fumes depends on the solubility gas, and less soluble gases tend to form centrilobular lesions while more soluble gases tend to form extensive GGO on chest CT. Interestingly, both of these (GGO, and centrilobular nodules) radiological findings were found in the HRCT scan of our case. The toxicity of chlorine gas depends on the concentration and total duration of chlorine gas exposure. High concentrations of chlorine gas exposure can be fatal, which can cause severe ARDS and toxic pneumonitis.[9]

Our patient also developed ARDS on the 3rd day of chlorine gas inhalation, which suggests higher chlorine concentration exposure leading to severe hypoxia and respiratory failure. The possible causes of hypoxia in chlorine gas inhalation-induced hypoxia may be ventilation perfusion (V/Q) mismatch and shunt formation. There is no specific treatment for acute inhalation injuries caused by chlorine gas, management is supportive and patients who have bronchospasm or wheezing bronchodilators might help. However, most of the time mild amounts of chlorine inhalation are self-limiting and do not warrant any specific treatment.[14] After thorough literature review regarding chlorine inhalation-induced acute lung injury, ARDS is a rare manifestation in clinical practice. The outcomes of mild chlorine gas inhalation have been reported to be generally good, having a low mortality rate (0.58%).[15] On the other hand, patients who develop ALI or ARDS, after chlorine exposure had a poor prognosis, having a mortality rate of 38.5%, the majority of deaths occurred within the first 3 days of hospitalization.[10] Early (within the first 3 days of exposure) corticosteroid therapy may have some advantages in chlorine-induced ARDS patients; however, using systemic or inhaled corticosteroids is still debatable. The possible therapeutic role of early systemic corticosteroids administration in chlorine-induced ARDS patients might be due to its anti-inflammatory effect on the acute inflammatory phase ARDS.[9,10]

In our case also, we have introduced systemic steroid on 3rd day of chlorine gas exposure and patient recovered remarkably, suggesting benefits of early introduction of steroid therapy. Special attention should be given while treating the ARDS patients to prevent possible complications, including hypotension, secondary infections, coagulopathy, hemoptysis, thrombus formation in arteries and/or veins, ventilator-associated lung injury (VALI) and barotrauma.[16]

Conclusion

Chlorine inhalation-induced acute lung injury or ARDS is a rare entity in clinical practice. A fatal outcome leading to refractory hypoxemia necessitating ventilator support is rarely seen. Supportive therapy along with early systemic corticosteroid can be helpful in moderate-to-severe ARDS patients requiring mechanical ventilation support, if carefully selected.

Orcid

Arjun Kumar https://orcid.org/0000-0002-5581-6176

References

1. Sexton JD, Pronchik DJ. Chlorine inhalation: The big picture. J Toxicol Clin Toxicol 1998;36:87–93. DOI: 10.3109/15563659809162593.
2. Winternitz MC. Collected studies on pathology of war gas poisoning. New Haven, CT: Yale University Press; 1920. p. 3.
3. Adelson L, Kaufman J. Fatal chlorine poisoning: Report of two cases with clinicopathologic correlation. Am J Clin Pathol 1971;56:430–442. DOI: 10.1093/AJCP/56.4.430.
4. Nodelman V, Ultman JS. Longitudinal distribution of chlorine absorption in human airways: Comparison of nasal and oral quiet breathing. J Appl Physiol 1999;86:1984–1993. DOI: 10.1152/jappl.1999.87.6.2073.
5. Kennedy SM, Enarson DA, Janssen RG, et al. Lung health consequences of accidental chlorine gas exposures among pulp mill workers. Am Rev Respir Dis 1991;143(1):74–79. DOI: 10.1164/ajrccm/143.1.74.
6. Deschamps D, Soler P, Rosenberg N, et al. Persistent asthma after inhalation of a mixture of sodium hypochlorite and hydrochloric acid. Chest 1994;105:1895–1896. DOI: 10.1378/chest.105.6.1895.
7. Guloglu C, Kara IH, Erten PG. Acute accidental exposure to chlorine gas in the Southeast of Turkey: A study of 106 cases. Environ Res 2002;88:89–93. DOI: 10.1006/enrs.2001.4324.
8. Traub SJ, Hoffman RS, Nelson LS. Case report and literature review of chlorine gas toxicity. Vet Hum Toxicol 2002;44:235–239. PMID: 12136975.
9. Babu RV, Cardenas V, Sharma G. Acute respiratory distress syndrome from chlorine inhalation during a swimming pool accident: A case report and review of the literature. J Intensive Care Med 2008;23:275–280. DOI: 10.1177/0885066608318471.
10. Chen TF, Wang CH, Hermes GL, et al. Chlorine inhalation injury with acute respiratory distress syndrome treated by extra-corporeal membrane oxygenation system. Glob J Medical Clin Case Rep 2020;7:005-009. DOI: 10.17352/2455-5282.000079.
11. Yadav AK, Bracher A, Doran SF, et al. Mechanisms and modification of chlorine-induced lung injury in animals. Proc Am Thorac Soc 2010;7:278-283. DOI: 10.1513/pats.201001-009SM.
12. Akira M, Suganuma N. Acute and subacute chemical-induced lung injuries: HRCT findings. Eur J Radiol 2014;83:1461–1469. DOI: 10.1016/j.ejrad.2014.04.024.
13. Katano T, Murao H, Kato T, et al. A case of acute inhalation injury caused by premeditated chlorine gas exposure. Respirol Case Rep 2021;9(5):e00743. DOI: 10.1002/rcr2.743.
14. Swinarew AS, Stanula AJ, Gabor J, et al. The influence of chlorine in indoor swimming pools on the composition of breathing phase of professional swimmers. Respir 2020;21:88. DOI: 10.1186/s12931-020-01350-y.
15. Evans RB. Chlorine: State of the art. Lung 2005;183:151–167. DOI: 10.1007/s00408-004-2530-3.
16. Moloney ED, Griffiths MJ. Protective ventilation of patients with acute respiratory distress syndrome. Br J Anaesth 2004;92:261–270. DOI: 10.1093/bja/aeh031.