Type of Article: Case Report

Title Combined Respiratory and Heart Failure After Chlorine Exposure: A Rare Case Report

Authors: Shawn Cho[1], Harrison Chu[1], Alexis Leo[1], Stephanie Yeh[1]

[1] [1]California Northstate University, College of Medicine

Correspondence: Shawn Cho

Address: 9700 West Taron Drive, Elk Grove, CA 95757, USA

College of Medicine, California Northstate University

Email: shawncho32@gmail.com

Tel: 916-686-7300

Clinical Question Box

Can acute exposure to chlorine gas lead to cardiac dysfunction in addition to respiratory injury?

While chlorine gas primarily causes respiratory tract irritation and pulmonary injury, it can also lead to cardiac complications such as myocarditis or toxic cardiomyopathy, resulting in reduced left ventricular function. Clinicians should consider cardiac evaluation in patients presenting with chest symptoms or hemodynamic instability following chlorine exposure.

Abstract

Background: Chlorine gas is a common industrial chemical with known toxic effects on the respiratory system. However, cardiac complications are rarely reported. Case Presentation: We report the case of a 23-year-old previously healthy male who developed acute respiratory distress and reduced left ventricular systolic function following accidental exposure to chlorine gas. The patient presented with dyspnea, hypoxia, and chest discomfort. Imaging revealed bilateral infiltrates, and echocardiography showed a reduced left ventricular ejection fraction (LVEF, 40–45%). He was diagnosed with heart failure with reduced ejection fraction (HFrEF) likely due to toxic cardiomyopathy or myocarditis. Supportive oxygen therapy and guideline-directed medical therapy for heart failure led to clinical improvement. Conclusion: This case highlights the potential for chlorine gas to cause not only pulmonary injury but also cardiac dysfunction. Clinicians should be aware of this rare but serious complication to enable timely diagnosis and management.

Keywords: Chlorine Poisoning, Heart failure, Respiratory Failure, Case report

Introduction

Chlorine gas (Cl_2) is widely used in various industries, including plastics manufacturing, waste management, water treatment, and the pharmaceutical industry.[1] Historically, it was employed as a chemical weapon during World War I.[2] In modern times, most human exposures to chlorine are accidental, typically occurring in occupational settings, transportation-related incidents, or household accidents.[3] At low concentrations (1–3 ppm), chlorine primarily irritates the eyes and oral mucous membranes, while concentrations around 15 ppm can also impact the pulmonary system. Prolonged exposure (30 minutes) to high concentrations, such as 430 ppm, can be fatal.[4] Despite the potential for severe toxicity, most chlorine exposures tend to be nonfatal due to their accidental nature. Chlorine was identified as the most common inhalation irritant in the United States in 2016.[5] The National Poison Data System reported 13,503 exposures to chlorine gas in 2022 alone.[6]

Research into chlorine gas toxicity remains limited, primarily due to the scarcity of controlled trials that quantify exposure levels and assess treatment options. Existing literature predominantly addresses pulmonary toxicity, while the cardiotoxic effects of chlorine gas are not yet fully understood. [7,8] Nonetheless, several reports suggest that chlorine gas exposure may trigger serious cardiovascular events, such as atrial fibrillation, myocardial infarction, and acute ischemic stroke.[9-11]Most studies have concentrated on the acute phase of these complications.[12] Here, we present a case of a patient with no prior medical history who developed both respiratory and cardiac failure following chlorine gas exposure.

Case Presentation

A 23-year-old man with a history of allergic rhinitis and obesity (Body Mass Index: 44.1) presented to the emergency department (ED) with a 24-hour history of nonproductive cough, shortness of breath, and burning substernal chest pain, worsened by inspiration and physical exertion. The symptoms began after he was exposed for approximately five minutes to chlorine fumes while sleeping on a couch near a bucket of concentrated chlorine mixed with hot water, which his father had prepared to clean a pool. He reported eye and nasal irritation upon awakening. He was not on any medications, had no recent travel to tuberculosis-endemic areas, no known TB contact, and no history of smoking or baseline oxygen use. He denied fever, chills, nausea, vomiting, abdominal pain, or diarrhea.

Initial vital signs in the emergency department showed a blood pressure of 129/104 mmHg, heart rate of 116 bpm, temperature of 36.8°C, respiratory rate of 34 breaths per minute, and oxygen saturation of 84% on room air, which improved to 93% with 3 L/min of oxygen via nasal cannula (NC). The physical examination was notable for tachypnea and decreased breath sounds on bilateral lung auscultation. The complete blood count and complete metabolic panel were within normal limits. Viral swabs for COVID-19, Influenza A/B, and RSV were negative. Laboratory findings included elevated serial troponin levels of 190 pg/mL (< 26.2 pg/mL) and a brain natriuretic peptide level of 173 pg/mL (<18.4 pg/mL). Electrocardiogram (EKG) showed sinus tachycardia at 117 bpm without ST-T elevation. Chest X-ray revealed bilateral hazy opacities suggestive of pulmonary edema (Figure 1). A Computed Tomography pulmonary angiogram ruled out pulmonary embolism but revealed multifocal patchy opacities, centrilobular nodules, and tree-in-bud opacities in a miliary pattern, raising suspicion for atypical infection, hypersensitivity pneumonitis, pneumoconiosis, tuberculosis, or other atypical pneumonias (Figure 2).

Toxicology was consulted because of the chlorine exposure. The toxicologist noted high suspicion for acute hypoxemic respiratory failure secondary to chlorine poisoning due to timing of onset of symptoms but mentioned that most patients experience complete resolution of pulmonary systems within 72 hours of exposure. Chest pain and substernal soreness was attributed to the possible presence of tracheobronchitis. Recommendation was made for supportive treatment involving bronchodilators, humidified oxygen, and a trial of steroids and possible 5% nebulized bicarbonate. Cardiology was consulted because of the pleuritic chest pain as well as elevated troponins. The cardiologist recommended an echocardiogram for evaluation of left ventricular function with concern for etiologies such as myocarditis.

On hospital day one, the patient's respiratory status showed slight improvement, and he was weaned to 1–2 L/min of oxygen via NC. A transthoracic echocardiogram revealed a left ventricle of normal size and wall thickness but with mildly reduced systolic function. The left ventricular ejection fraction (LVEF) was estimated at 40–45%, and right ventricular systolic function appeared grossly normal (Figure 3). The working diagnosis was heart failure with reduced ejection fraction, possibly due to toxic cardiomyopathy or myocarditis. Cardiology recommended initiating metoprolol XR 25 mg and losartan 12.5 mg, both administered orally once daily. A consultation with an imaging cardiologist was also recommended to assess the need for a cardiac MRI. C-reactive protein (CRP) and erythrocyte sedimentation rate (ESR) were recommended for evaluation.

On hospital day two, the patient remained stable on 1–2 L/min oxygen via NC. CRP and ESR levels were within normal limits. Cardiac MRI showed mildly to moderately reduced LVEF at 36% (≥47%) and reduced right ventricular ejection fraction (RVEF) at 35% (≥45%), with no

evidence of myocarditis (Table 3). Cardiology advised continuing metoprolol and losartan, and adding spironolactone and empagliflozin.

By hospital day three, the patient had been weaned off supplemental oxygen and was able to ambulate around the unit while maintaining an oxygen saturation level in the mid-90s. He and his parents were counseled on the importance of weight loss for improving long-term respiratory function. A referral for nocturnal polysomnography was placed to evaluate for obstructive sleep apnea. The patient was discharged home the same day, with plans for outpatient follow-up with cardiology.

Discussion

This case highlights a rare instance of combined respiratory and cardiac dysfunction following acute exposure to chlorine gas in a previously healthy young adult. While chlorine inhalation injury is well-documented for its pulmonary toxicity,[13] the concomitant development of cardiac impairment remains a less recognized but clinically significant consequence. Chlorine gas, a potent oxidizing agent, primarily affects mucosal surfaces and the respiratory tract, resulting in symptoms ranging from mild irritation to acute lung injury and acute respiratory distress syndrome.[14] Most clinical manifestations occur within minutes of exposure and resolve within a few days with supportive therapy.[3] Our patient exhibited typical respiratory symptoms, including cough, dyspnea, and hypoxemia, as well as radiographic findings consistent with chemical pneumonitis. His respiratory symptoms responded well to bronchodilators, corticosteroids, and supportive oxygen therapy, consistent with prior reports.

In contrast, cardiac involvement in cases like this is relatively uncommon in the literature. Studies have demonstrated that the adverse effects of inhaled toxic gases extend beyond the respiratory tract, potentially causing systemic endothelial dysfunction. This dysfunction may contribute to the development of cardiovascular conditions, including atherosclerosis and myocardial injury.[15, 16] Elevated troponin levels and reduced biventricular ejection fractions raised concern for chlorine-induced cardiotoxicity. While myocarditis was initially suspected, cardiac MRI showed no signs of inflammation. Takotsubo cardiomyopathy and viral myocarditis were considered but deemed unlikely based on imaging findings, absence of typical triggers, and lack of viral symptoms. These findings support a diagnosis of toxic cardiomyopathy.[17] This case underscores the importance of early cardiology input and advanced imaging in assessing atypical cardiac symptoms after chemical exposure.

Management with standard heart failure medications, including beta-blockers, angiotensin receptor blockers, mineralocorticoid receptor antagonists, and SGLT2 inhibitors, was initiated promptly and contributed to the patient's clinical improvement.[18] A limitation of this case is the absence of long-term follow-up data, which restricts our ability to assess the persistence or resolution of cardiac dysfunction over time.

Conclusion

This case highlights the potential for both respiratory and cardiac complications following acute exposure to chlorine gas exposure. Prompt recognition and early supportive care, including heart failure management, were key to recovery. Clinicians should remain vigilant for cardiac involvement in similar toxic exposures.

Acknowledgments: We appreciate the patient's cooperation in this study.

Funding Source: None

Author Contributions: S.C. and H.C. contributed to drafting the manuscript. A.L. conducted the literature search, quality assessment, and data extraction. S.Y. was responsible for data revision. All authors have read and approved the final manuscript and agree with its content and data.

Data Availability: The corresponding author shall make the datasets available upon reasonable request.

Ethical Statement: The patient gave written informed consent to the publication of this report and the accompanying images.

Conflict of Interest: The authors report no conflicts of interest in this work.

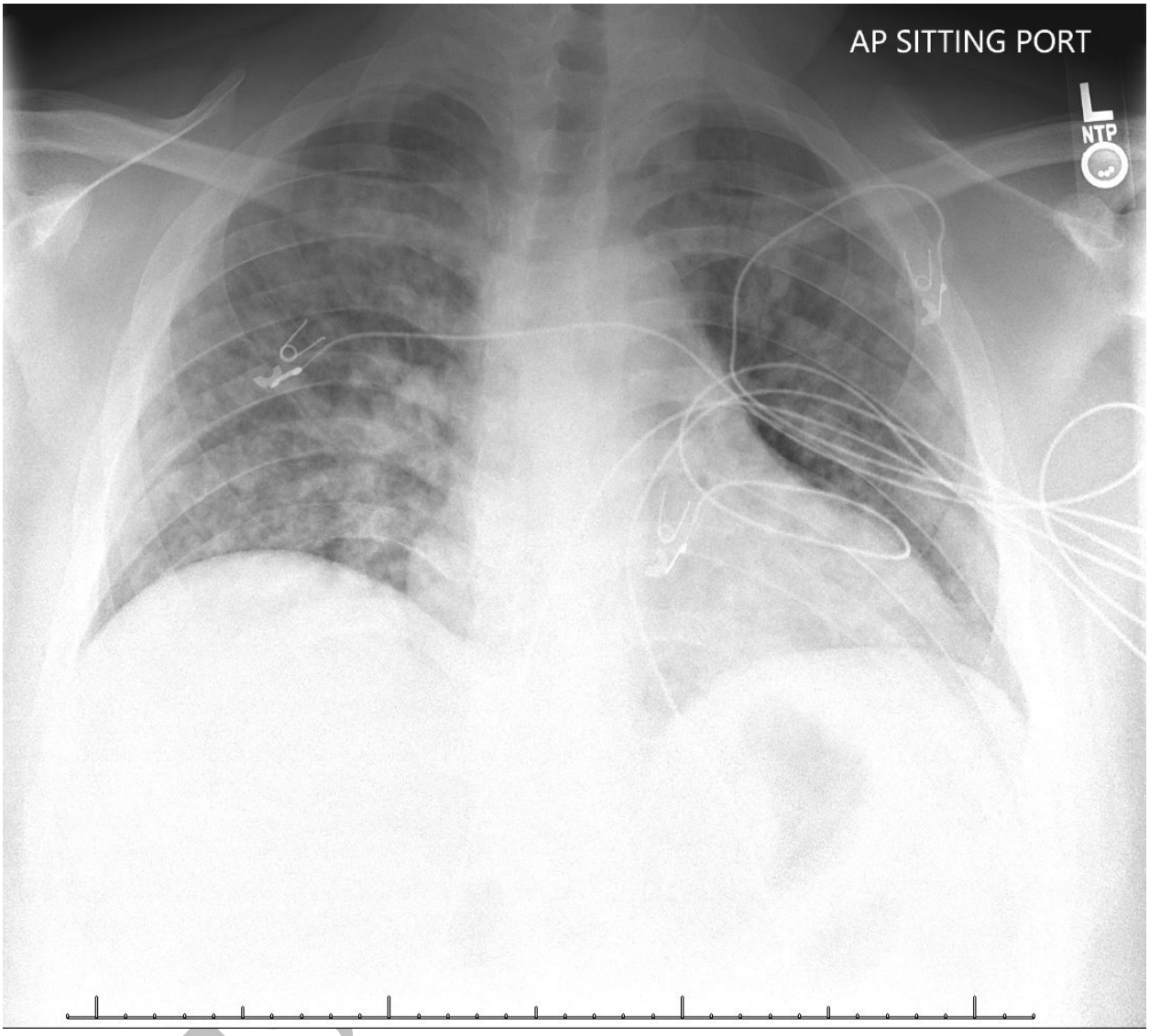
Reference

1. Nambiema A, Coyo G, Barbe-Richaud JB, et al. Human chlorine gas exposition and its management - an umbrella review on human data. *Crit Rev Toxicol*. Jan 2022;52(1):32-50. doi:10.1080/10408444.2022.2035317
2. de Bruin-Hoegée M, van der Schans MJ, Langenberg JP, van Asten AC. Biomarker profiling in plants to distinguish between exposure to chlorine gas and bleach using LC-HRMS/MS and chemometrics. *Forensic Sci Int*. May 2024;358:112022. doi:10.1016/j.forsciint.2024.112022
3. Achanta S, Jordt SE. Toxic effects of chlorine gas and potential treatments: a literature review. *Toxicol Mech Methods*. May 2021;31(4):244-256. doi:10.1080/15376516.2019.1669244
4. Morim A, Guldner GT. Chlorine Gas Toxicity. *StatPearls*. StatPearls Publishing

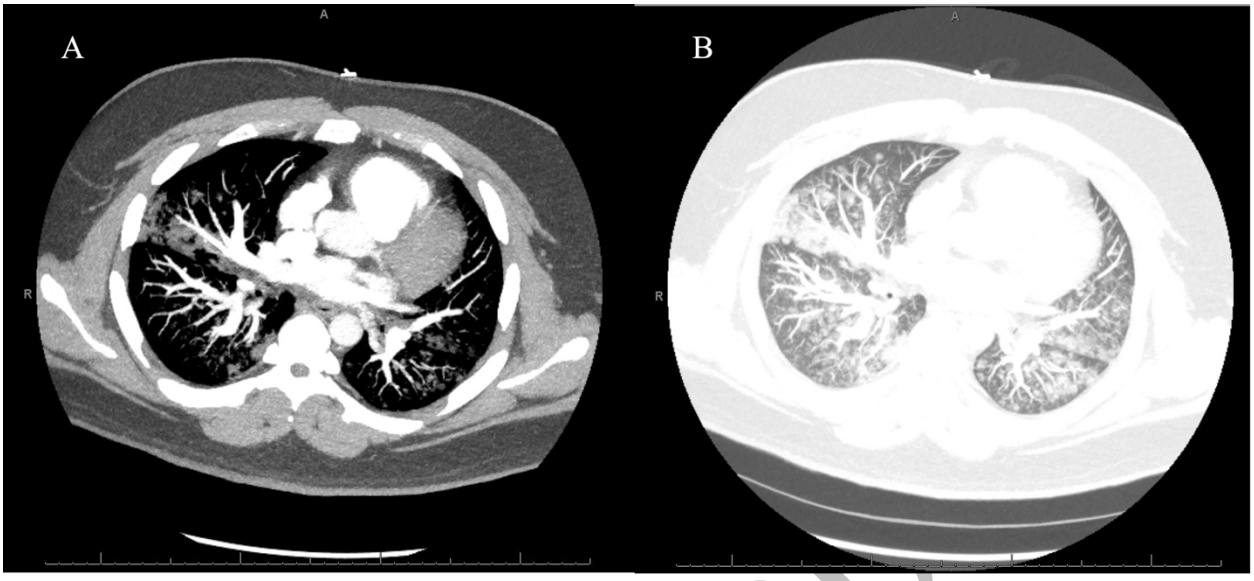
Copyright © 2025, StatPearls Publishing LLC.; 2025.
5. White CW, Martin JG. Chlorine gas inhalation: human clinical evidence of toxicity and experience in animal models. *Proc Am Thorac Soc*. Jul 2010;7(4):257-63. doi:10.1513/pats.201001-008SM
6. Atalla A, Shulman J, Rose J, Lynch M. Trends in chlorine and chloramine gas exposures reported to United States poison centers. *Clin Toxicol (Phila)*. Sep 2024;62(9):589-595. doi:10.1080/15563650.2024.2390139
7. Mashak KA, Agin K, Emaratkar E, Gholami Fesharaki M, Namdar H. The effect of wet cupping on reactive airway dysfunction syndrome (RADS) caused by early exposure to chlorine gas: A randomized clinical trial. *Avicenna J Phytomed*. Jan-Feb 2025;15(2):997-1015. doi:10.22038/ajp.2024.25107
8. Bleifuss W, Pepin L, Lock S, Kaluzniak H, Kempainen R. Chlorine gas induced acute respiratory distress syndrome due to pool shock. *Am J Emerg Med*. Apr 2 2025;doi:10.1016/j.ajem.2025.04.001
9. Gülaşti F, Gülaşti S. Acute Atrial Fibrilation After Inhalation of Chlorine Gas. *GKD Anest Yoğ Bak Dern Derg*. 2020;26(1):45-49. doi:10.5222/gkdad.2020.08941
10. Kose A, Kose B, Açikalin A, Gunay N, Yildirim C. Myocardial infarction, acute ischemic stroke, and hyperglycemia triggered by acute chlorine gas inhalation. *Am J Emerg Med*. Oct 2009;27(8):1022.e1-4. doi:10.1016/j.ajem.2008.12.029
11. Polat E, Demir MC. Myocardial Injury Due to Inhalation of A Mixture of Sodium Hypochlorite and Hydrochloric Acid. Sodyum Hipoklorit ve Hidroklorik Asit Karışımının İnhalasyonuna Bağlı Gelişen Miyokard Hasarı. *Anatolian Journal of Emergency Medicine*. December 2019;2(4):25-27.
12. Rasouli HR, Hasani Nourian Y, Ghanei M, Salesi M, Nobakht Motlagh Ghoochani BF. A Systematic Review of the Effect of Acute, Chronic, Low, and High Chlorine Gas on People's Early and Delayed Complications. *Iranian Red Crescent Medical Journal (IRCMJ)*. 2024;26(1):-. doi:10.22034/ircmj.2024.199016
13. Pham DT, Dao BN, Nguyen DT, et al. A rare case of acute respiratory distress syndrome due to chlorine gas inhalation: Rapid progression with favorable ICU outcome. *Respir Med Case Rep*. 2025;53:102148. doi:10.1016/j.rmcr.2024.102148
14. Chen T-F, Wang C-H, Lain Hermes G, Ko W-J. Chlorine inhalation injury with acute respiratory distress syndrome treated by extra-corporeal membrane oxygenation system. *Global Journal of Medical and Clinical Case Reports*. 04/11 2020;7(1):005-009. doi:10.17352/2455-5282.000079

15. Zaky A, Ahmad A, Dell'Italia LJ, et al. Inhaled matters of the heart. *Cardiovasc Regen Med*. 2015;2doi:10.14800/crm.997
16. Zaky A, Bradley WE, Lazrak A, et al. Chlorine inhalation-induced myocardial depression and failure. *Physiol Rep*. Jun 2015;3(6)doi:10.14814/phy2.12439
17. Amin HZ, Amin LZ, Pradipta A. Takotsubo Cardiomyopathy: A Brief Review. *J Med Life*. Jan-Mar 2020;13(1):3-7. doi:10.25122/jml-2018-0067
18. Maddox TM, Januzzi JL, Jr., Allen LA, et al. 2024 ACC Expert Consensus Decision Pathway for Treatment of Heart Failure With Reduced Ejection Fraction: A Report of the American College of Cardiology Solution Set Oversight Committee. *J Am Coll Cardiol*. Apr 16 2024;83(15):1444-1488. doi:10.1016/j.jacc.2023.12.024

Figure 1. Chest X-ray Image



220 Figure 2. Computed Tomography of the Chest with Contrast of Axial View

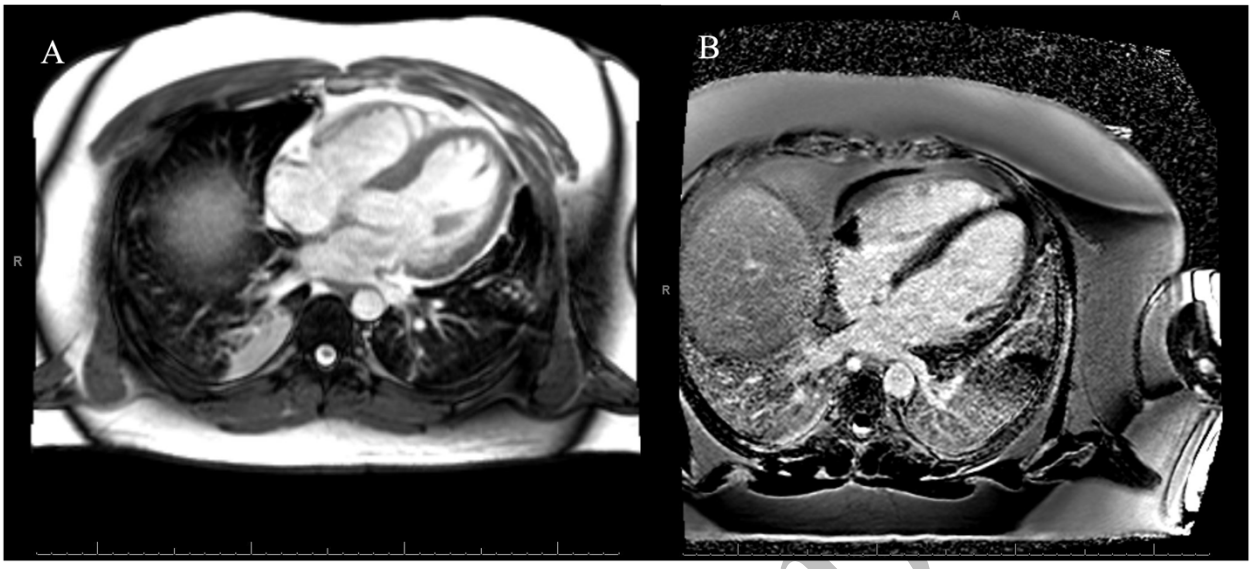


221

222 A: soft tissue window; B: lung window

223

224 Figure 3. Cardiac Magnetic Resonance Imaging Findings in the Myocardial Wall



225

226 A: T2-weighted; B: T1-weighted post-contrast

227