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CASE REPORT

The Toxic Fumes: A Case Report of an Accidental Inhalation of HCL 33%

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Abstract

Introduction: Most household cleaners contain chlorine, an irritating chemical commonly used to purify water. The inhalation of chlorine causes chemical pneumonitis and the treatment of inhalation injuries from this chemical is difficult due to the lack of evidence and human studies. We will discuss a case of a man, who developed chemical pneumonitis after being exposed to chlorine fumes while cleaning swimming pool.

Case Presentation: A 30-year-old man showed up after being exposed to 33% hydrochloric fumes at work and developed coughing, sore throat, and dyspnea immediately. He received initial treatment at nearby clinic and referred to the emergency department. In emergency department, he was in respiratory distress. His respiratory rate was 28 breaths per minute and oxygen saturation was 81% with a 15 L/min non-rebreathing mask. Lung auscultation showed crepitation in both lower zones with expiratory rhonchi. Noninvasive ventilation support was initiated with continuous nebulization of salbutamol, ipratropium bromide, and sodium bicarbonate. His initial blood gases showed type 1 respiratory failure. He was then intubated for severe acute respiratory distress syndrome. The patient was then admitted to the intensive care unit for further treatment and discharged after 18 days of hospitalization.

Conclusion: Acute exposure to chemical irritants can cause asthma exacerbation, chronic bronchitis, bronchial hypersensitivity, and ARDS. There are no biomarkers for the inhalation of HCL gas. Medical history, clinical symptoms, and radiographic findings are decisive factors in the diagnosis. Treatment usually focuses on supportive care such as oxygen therapy, broncholytic therapy, and sodium bicarbonate, inhaled or systemic corticosteroids.

Keywords: Chlorine, Hydrochloric Acid, Respiratory Distress Syndrome, Sodium Bicarbonate

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INTRODUCTION

Hydrochloric acid (HCL), is an aqueous solution of hydrogen chloride, frequently utilized for water treatment [1]. On exposure to air, the gas forms corrosive vapors, which has highly irritating properties. Due to a lack of studies, evidence-based guidelines and recommendations, neither a particular antidote nor a tested therapy exists [2]. Therefore, treating inhalation chemical pneumonitis remains challenging. We shared our successful experience in managing a patient, who was diagnosed with chemical pneumonitis and complicated by severe ARDS following exposure to HCL fumes using the "kitchen sink" approach.

CASE REPORT

A 30-year-old pool cleaner presented at the hospital after inhaling 33% HCL, which is used to disinfect the water in the pool. He claimed that he was not wearing any protective gear, and was exposed to the chemical's vapors for approximately 30 minutes in a poorly ventilated room. He started experiencing coughing, sore throat, and dyspnea right away. Nebulized salbutamol 5 mg, an intramuscular injection of adrenaline 0.3 mg, and IV hydrocortisone 200 mg were administered at a local clinic before transported to hospital for further management.

Upon arrival at ED, the patient was tachypnoeic, sweating profusely, in tripod posture, with oxygen saturation of 81% on a 15 L/min non-rebreathing mask. Further physical examination revealed crepitation in both lower zones with expiratory rhonchi, which is suggestive of chemical pneumonitis. No central cyanosis, clubbing, or limb edema observed. He was afebrile with blood pressure of 125/71 mmHg, heart rate of 122 per minute and respiratory rate of 28 per minute. Non-invasive ventilation (NIV) support was initiated promptly with continuous nebulization of salbutamol, adrenalin, ipratropium bromide, and sodium bicarbonate (NaHCO3). Due to its bronchodilator and anti-inflammatory qualities, magnesium sulphate infusions were given concurrently. His laboratory investigation and blood gaseous showed type 1 respiratory failure with a P/F ratio of 60 (Table 1).

The chest x-ray (CXR) shows homogenous opacities at bilateral lower zones of the lungs, with a normal cardiothoracic ratio (Figure 1). The patient was then intubated for severe ARDS following a failed NIV and pharmacological therapy. Despite high doses of sedatives (propofol and midazolam), analgesics (fentanyl), and boluses of non-

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depolarizing agent, the patient was started on atracurium infusion in view of persistent ventilatory dyssynchrony with a Richmond Agitation Sedation Scale of +1 to +2. A repeated CXR after intubation (12 hours of post-exposure) reveals scattered, poorly defined coalescent opacities in both lungs (Figure 2). The patient was then admitted into the ICU, ventilated for 14 days, and discharged well after 18 days of hospitalisation without any oxygen support.

DISCUSSION

Inhalational injury caused by HCL fumes is not uncommon. Swimming pools continue to be the site where

Table 1. Blood Investigations of the patient in Emergency Department and Blood gaseous throughout admission.

FBC		LFT	
TWC	10 x10^9/L	Total Protein	67 g/dL
Hb	13.7 g/dL	Albumin	39 g/dL
Hct	42	Bilirubin	8 micromol/L
Plt	319 x10^9/L	ALT	22 U/L
		ALP	88 U/L
RP			
Urea	5.5 mmol/L	Lactate	1.7 mmol/L
Sodium	138 mmol/L		
Pottasium	3.5 mmol/L	Blood Glucose	11.6 mmol/L
Creatinine	83 micromol/L		

ABG

	on HFM (15L/min)	1Hour on NIV	Post Intubation	In ICU Day1	In ICU Day7	Upon discharge
pH	7.34	7.26	7.25	7.32	7.41	7.42
pO2	48.7	79.8	87.4	79.4	95.8	133
pCo2	38.9	42.3	48.6	42.2	39.8	34.9
HCO3	20.6	18.7	19.7	21.2	25.4	23.6
sO2 (%)	78.7	92.3	95.2	94.8	96.8	98.9
FiO2	1.0	1.0	1.0	0.8	0.5	0.21

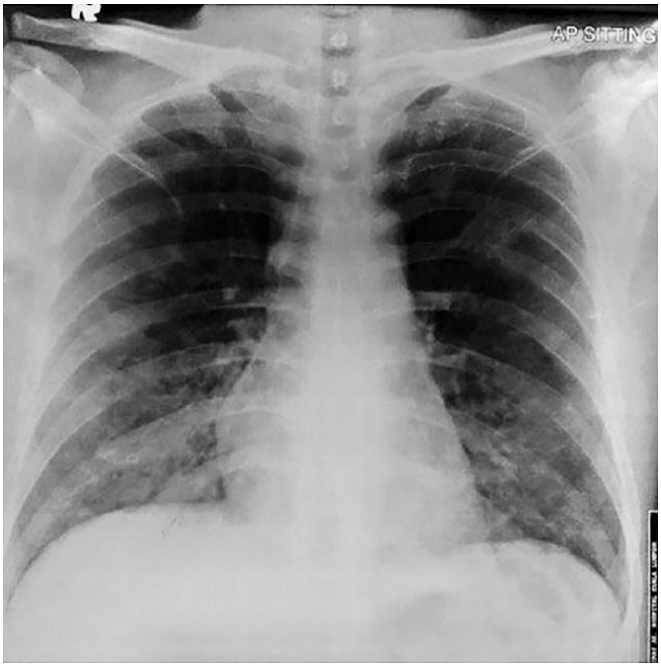

Figure 1. The chest x-ray after 2 hours of exposure

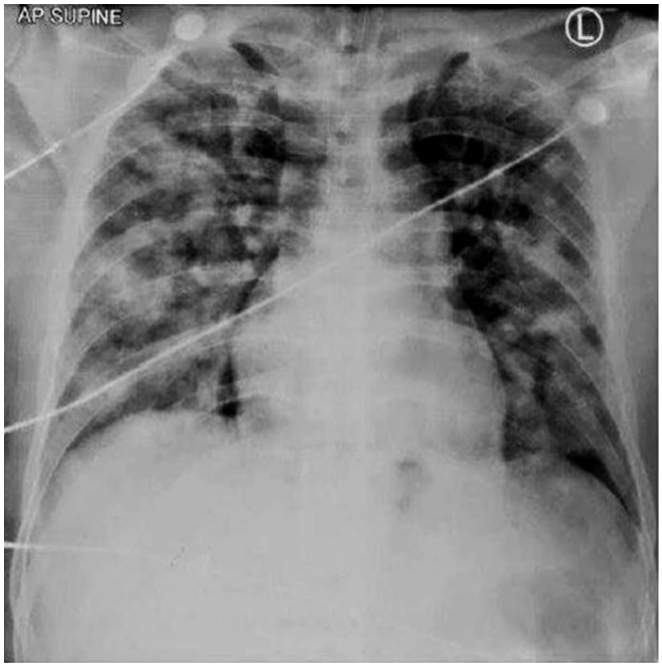

Figure 2. The chest x-ray after 12 hours of exposure to the chemical

acute chemical incidents involving chlorine and its derivatives are most frequently reported [3]. Malaysia's data on poisoning patterns is scarce and incomplete. A cross-sectional study from 1999 to 2001 in Malaysia found that there were 369 admissions (1.7%) and 3 fatalities caused by gaseous, toxic fumes and vapors [4].

The primary uses of HCL and its derivatives are as disinfectants, laboratory reagents, and in the manufacturing of metals, rubber, food, and pharmaceuticals [5,6]. It is a non-flammable corrosive gas that emits pungent odors into the air. For acute, high-level exposures, its odor and highly irritating characteristics typically serve as an effective warning. However, at permitted exposure limit of 5 mg/L, only 50% of exposed individuals can detect the smell. As the vapor is heavier than air, it can asphyxiate people in small, poorly ventilated space that are low to the ground.

The severity of the poisoning is determined by the amount of exposure, concentration, and duration of the exposure. To date, there are neither specific clinical findings nor specific investigations to identify HCL inhalation victims or estimate the inhaled dose of the toxic fumes. In a study on the victims of HCL inhalation following a South Carolina train derailment accident in 2005, they found that the presentation was different: cough (37%), wheeze (84%), low PaO2/FiO2 ratio (58%), and abnormal chest radiograph (57%) with 75% showing abnormalities within the first day after exposure. These findings are not specific and could be found after the inhalation of any types of chemical irritants (White et al 2010) [7]. In this case, the amount of fumes inhaled was unknown. However, based on the history which included the type of chemical involved, time and length of exposure, location of the incident, and the symptoms that appeared after contact, diagnosis of chemical pneumonitis from inhalation of HCL acid fumes can be ascertained.

The clinical approach to patients exposed to HCL fumes is similar to smoke inhalation injury [8,9]. The care is supportive with a focus on maintaining the airway patency, optimizing the oxygenation with oxygen therapy and administration of bronchodilators (beta-agonists and anti-cholinergic drugs). However, a few small studies showed that corticosteroids might be helpful in treating acute exposure to HCL fumes. In an animal study by Gunnarson et al. (2000), it was discovered that immediate treatment with aerosolised beclomethasone-dipropionate after HCL gas injury enhanced the lungs compliance and oxygen delivery (p 0.01) in comparison to the control group that received no steroid treatment [9]. Another study by Wang et al. came to the conclusion that early treatment with either systemic or inhaled corticosteroids significantly improved cardiovascular and pulmonary function. They also found that the combination of aerosolised terbutaline and budesonide improved the oxygen partial pressure in the blood (PaO2) and lung compliance compared to therapy with either drug alone. Nevertheless, the mortality rate was the same for both groups [10, 11].

Several published case reports have shown the safety and effectiveness of nebulized sodium bicarbonate (NSB) when given for HCL fumes inhalation, despite the lack of controlled clinical trials. Theoretically, it can neutralize HCL cum decreasing the severity of lung injury [12]. In a two-year retrospective review by Bosse et al (1994) that involved 86 patients exposed to the HCL fumes, NSB (3 mL of 8.4% NaHCO3 with 2 mL of normal saline to prepare 5 mL of a 5% NaHCO3 solution) was given to 86 patients after exposure to the chlorine fumes. All patients improved and had a mean hospital stay of 1.4 days (ranging from 1 to 3 days) [13]. Aslan et al. (2006) further conducted a prospective randomized trial to evaluate the efficacy of NSB in the treatment and quality of life (QoL) of HCL fumes exposure patients [14]. All subjects, who developed wheezing after exposure to HCL gas received neb salbutamol, prednisolone, and either 4 mL of 4.2% NaHCO3 solution or a placebo (normal saline). None of the participants had a past history of pulmonary illness. The QoL questionnaire scores considerably increased (p.001) and the FEV1 values at 120 and 240 min were significantly higher in the NSB group (p 0.05). In our case, systemic steroid therapy, continuous broncholytics therapy, and NSB did result in some improvement in the patient. Nevertheless, the role of NSB in this context has several limitations and decision to use it should be based on a thorough assessment of the patient's clinical condition, the severity of the injury, and the availability of other essential treatments. There is no universally accepted dosing regimen for NSB. Hence, certain physician might not be familiar with the dosing. In our case, we gave 5 mL of 4.2% NSB by mixing 8.4% NaHCO3 with water for injection at a 1:1 ratio every 6 hours.

Inhalation injuries commonly produce copious secretions, hemorrhage, and mucosal slough, which impairs gas exchange and necessitates aggressive pulmonary toileting. N-acetylcysteine (NAC) has a mucolytic and antioxidant properties that can destabilise the mucus, while heparin attenuates pulmonary coagulopathy and reduce pulmonary inflammation in critically ill patients [15,16]. In a few animal experiments and human trials, both have been demonstrated to be a successful treatment for inhalation injuries (greater lung compliance, shorter ventilator-free days, early extubation, reduced morbidity and mortality rate) [17,18,19]. Given these, it might be beneficial to administer aerosolized NAC 20% in 3 mL together with aerosolized heparin 5,000–10,000 units in 3 mL of normal saline every 4–6 hours as a result [20,21]. Fortunately, our patient did produce copious secretions necessitating their use throughout the ED stay

One of the complications of toxic fume inhalation is ARDS - an acute and diffuse inflammatory lung injury that leads to increased pulmonary vascular permeability, loss of lung aeration, increased physiological dead space, and decreased pulmonary compliance [22,23]. According to the New Berlin criteria for diagnosing ARDS, the symptoms of respiratory distress must appear within a week of the clinical insult, which is demonstrated by the appearance of scattered opacities in both lungs on a CXR without a sign of heart failure or volume overload. It is further classified based on the value of the PaO2/FiO2 ratio at a minimum positive end-expiratory pressure (PEEP) of 5 cmH2O [24,25]. In settings with limited resources (no PEEP value nor PaO2 level), the Kigali Modification of the Berlin Criteria can be used to characterise ARDS; a hypoxic cut-off value of SpO2/FiO2

less than or equal to 315, and positive findings of bilateral capacities either using lung ultrasonography or CXR [26]. Recent studies have shown that NIV, CPAP, and HFNC that are frequently used in patients with mild ARDS could successfully improve oxygenation. However, the applicability of these oxygen delivery therapies in chemical induced ARDS, has not been proven with a very limited literature available yet to support its use [25,27]. A retrospective observational study revealed that patients, who fail NIV have outcomes similar to those who are intubated early, while those who successfully avoid intubation with NIV have a better prognosis. The FLORALI trials revealed that intubation rates were the same for both NIV and high-flow oxygen therapy for acute hypoxic respiratory failure, but high-flow was better in terms of mortality at 90 days. With regard to our case, despite the patient's early use of CPAP, the one-hour HACOR score remained high, indicating a higher propensity for NIV failure. The patient was then intubated for hypoxic respiratory failure and ventilated using the lung-protective strategies for the next 48 hours. A continuous infusion of neuromuscular blocking (NMB) drug was started, as it is associated with a lower risk of barotrauma and improved oxygenation after 48 hours with no worsening of ICU-acquired weakness, and was beneficial in a subset of patients who required more sedation, according to a meta-analysis of five major randomized controlled trials. However, regular use of continuous NMB in an unselected population of ARDS patients is not recommended [27,28]. This is corroborated by the ACURASYS trial, which found that the early administration of a NMB agent to ARDS patients receiving lung protective ventilation increased ventilator-free days and decreased 90-day mortality. In addition, the use of diuretics and personalized fluid therapy has been proven to reduce the need for mechanical ventilation in ARDS patients.

Through the course of the hospital stay, our patient made good improvement without contracting a nosocomial infection. The earlier initiated empirical antibiotic and systemic steroid therapy was stopped on day 7 of admission. Although our patient was extubated, he occasionally still required bronchodilators, which is warranted for treatment of reactive airway dysfunction syndrome (RADS); a type of asthma, which affects patients, who have significant exposures to irritants or chemicals (absence of history of underlying respiratory diseases, onset of symptoms within 24 hours of exposure, positive test for bronchial hyper-reactivity, and airflow obstruction) [29,30].

CONCLUSION

Based on this study, it can be concluded that the inhalation of corrosive HCL toxic fume can cause chemical pneumonitis leading to severe ARDS. To date, there are no biomarkers to suggest HCL fume exposure. The history of presentation and clinical symptoms, as well as radiographic findings, are critical in making a diagnosis. Treatment remains supportive using oxygen therapy, broncholytic therapy, and sodium bicarbonate, inhaled or systemic corticosteroids.

Author's Contributions

Agree to be accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved

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REFERENCES

1. Environmental Health Expert Advisory Committee (A Committee under National Environmental Health Action Plan – NEHAP Malaysia). Quick Reference Database of Toxic Chemicals; 2020.
2. Medical Management Guidelines for Hydrogen Chloride [Internet]. 2014. Available from: https://www.atsdr.cdc.gov/MHMI/mmg173.pdf [Assessed Nov15, 2022]
3. H. Lucy Thomas, Virginia Murray. Review of acute chemical incidents involving exposure to chlorine associated with swimming pools in England and Wales, June– October 2007. Journal of Public Health; 2007;30(4):391–397 doi:10.1093/pubmed/ fdn073.
4. R Rajasuriar. Profile of poisoning admissions in Malaysia. Human & Experimental Toxicology 2007; 26:73-81. http:het.sagepub.com
5. Min-Po Ho. Chlorine Gas Exposure Manifesting Acute Lung Injury. J Intern Med Taiwan 2010; 21:210-215.
6. Health Protection Agency Compendium of Chemical Hazards. Chlorine. [Internet] 2008. Available from: http:// www.hpa.org.uk/web/HPAwebFile/HPAweb_C/ 1194947365071 (7 September 2008, date last accessed).
7. White CW, Martin JG. Chlorine gas inhalation: human clinical evidence of toxicity and experience in animal models. Proc Am Thorac Soc 2010;7(4):257-63.
8. Gunnarsson M, Walther SM, Seidal T, Lennquist S. Effects of inhalation of corticosteroids immediately after experimental chlorine gas lung injury. J Trauma 2000;(48):101-7.
9. Omar Saeed, MC, USN, Nathan L Boyer, MC, USA, Jeremy C Pamplin, MC, USA, Ian R Driscoll, MC, USA, Jeff DellaVolpe, USAF, MC, Jeremy Cannon, USAFR, MC, Leopoldo C Cancio, MC, USA, Inhalation Injury and Toxic Industrial Chemical Exposure, *Military Medicine*, 2018;September-October 183(2):130–132, https://doi.org/10.1093/milmed/usy073
10. Wang J, Zhang L, Walther SM. Administration of aerosolized Terbutaline and Budesonide reduces chlorine gas–induced acute lung injury. The Journal of Trauma: Injury, Infection, and Critical Care. 2004;56(4):850–62.
11. Wang J, Zhang L, Walther SM. Administration of aerosolized Terbutaline and Budesonide reduces chlorine gas–induced acute lung injury. The Journal of Trauma: Injury, Infection, and Critical Care. 2004;56(4):850–62.
12. John E. Vajner III & Derrick Lung. Case Files of the University of California San Francisco Medical Toxicology Fellowship: Acute Chlorine Gas Inhalation and the Utility of Nebulized Sodium Bicarbonate. J. Med. Toxicol 2013;(9):259–265 DOI 10.1007/s13181-013-0309-8.
13. Bosse GM. Nebulized sodium bicarbonate in the treatment of

chlorine gas inhalation. J Toxicol Clin Toxicol 1994;32(3):233-41.

14. Aslan S, Kandiş H, Akgun M, Cakir Z, Inandi T, Gorquner M. The effect of nebulized NaHCO3 treatment on "RADS" due to chlorine gas inhalation. Inhal Toxicol. 2006; Oct 18(11):895-900.
15. Brodier EA, Raithatha M, Kannan S, Karunasekara N. Use of nebulised N- acetylcysteine as a life-saving mucolytic in intensive care: A case report. Journal of the Intensive Care Society. 2020;21(4):296-298. doi:10.1177/1751143719870089
16. Elsharnouby NM, Eid HE, Abou Elezz NF, Aboelatta YA. Heparin/Nacetylcysteine: an adjuvant in the management of burn inhalation injury: a study of different doses. J Crit Care. 2014 Feb;29(1):182.e1-4. doi: 10.1016/j.jcrc.2013.06.017. Epub 2013 Aug 8. PMID: 23932140.
17. Juschten, J., Tuinman, P. R., Juffermans, N. P., Dixon, B., Levi, M., & Schultz, M. J. (2017). Nebulized anticoagulants in lung injury in critically ill patients-an updated systematic review of preclinical and clinical studies. *Annals of translational medicine*, 2017;*5*(22):444. https://doi.org/10.21037/atm.2017.08.23
18. Ashraf, Umair MDa,*; Bajantri, Bharat MDa; Roa-Gomez, Gabriella MDb; Venkatram, Sindhaghatta MD, FCCPa; Cantin, Amanda PharmD, BCCCPc; DiazFuentes, Gilda MD, FCCPa. Nebulized heparin and N-acetylcysteine for smoke inhalational injury: A case report. Medicine: 2018;May(97):Issue 19 - p e0638 doi: 10.1097/MD.0000000000010638
19. McGinn KA, Weigartz K, Lintner A, Scalese MJ, Kahn SA. Nebulized Heparin With N-Acetylcysteine and Albuterol Reduces Duration of Mechanical Ventilation in Patients With Inhalation Injury. J Pharm Pract. 2019;Apr;32(2):163-166. doi: 10.1177/0897190017747143. Epub 2017 Dec 12. PMID: 29233052.
20. Van Meenen, David M.P. ; Van Der Hoeven, Sophia M. ; Binnekade, Jan M. et al. / Effect of on-demand vs routine nebulization of acetylcysteine with salbutamol on ventilator-free days in intensive care unit patients receiving invasive ventilation : A randomized clinical trial. In: JAMA: Journal of the American Medical Association. 2018 ; 319(10):993-1001.
21. Noha M. Elsharnouby, Hala E.A. Eid, Nahla F. Abou Elezz, Yasser A. Aboela9a, Heparin/Nacetylcysteine: An adjuvant in the management of burn inhalaTon injury: A study of different doses,Journal of CriTcal Care, 2014 29(1):182 ISSN 0883-9441, h9ps://doi.org/10.1016/j.jcrc.2013.06.017.
22. Ioannis Pneumatikos. The new Berlin definition: What is, finally, the ARDS? PNEUMON Number 4, Vol. 25, October - December 2012.
23. Acute Respiratory Distress Syndrome Network, Brower, R. G., Matthay, M. A., Morris, A., Schoenfeld, D., Thompson, B. T., & Wheeler, A. (2000). Ventilation with lower tidal volumes as compared with traditional tidal volumes for acute lung injury and the acute respiratory distress syndrome. *The New England journal of medicine*, *342*(18), 1301–1308. https://doi.org/10.1056/NEJM200005043421801
24. Ferrer M, Torres A. Noninvasive ventilation for acute respiratory failure. Curr Opin Crit Care. 2015;21(1):1-6.
25. Long B, Liang SY, Lentz S. High flow nasal cannula for adult acute hypoxemic respiratory failure in the ED setting. Am J Emerg Med. 2021 Nov;49:352-359. doi: 10.1016/j.ajem.2021.06.074. Epub 2021 Jul 3. PMID: 34246166; PMCID: PMC8555976.
26. Lazzeri C, Peris A. The Kigali modification of the berlin definition: a new epidemiological tool for ARDS? J Thorac Dis. 2016 Jun;8(6):E443-5. doi: 10.21037/jtd.2016.03.84. PMID: 27294247; PMCID: PMC4886026.
27. Matthay MA, Zemans RL, Zimmerman GA, Arabi YM, Beitler JR, Mercat A, Herridge M, Randolph AG, Calfee CS. Acute respiratory distress syndrome. Nat Rev Dis Primers. 2019 Mar 14;5(1):18. doi: 10.1038/s41572-019-0069-0. PMID: 30872586; PMCID: PMC6709677.
28. H Ho ATN, Patolia S, Guervilly C. Neuromuscular blockade in acute respiratory distress syndrome: a systematic review and meta-analysis of randomized controlled trials. J Intensive Care. 2020 Jan 28;8:12. doi: 10.1186/s40560-020-0431-z. PMID: 32015880; PMCID: PMC6986163.
29. Faria VS, da Silva SAEHC, Marchini JFM. Reactive airways dysfunction syndrome following inhalation of hydrogen chloride vapor. Autops Case Rep. 2021 Apr 20;11:e2021266. doi: 10.4322/acr.2021.266. PMID: 33968833; PMCID: PMC8087388.
30. Shakeri MS, Dick FD, Ayres JG. Which agents cause reactive airways dysfunction syndrome (RADS)? A systematic review. Occup Med (Lond). 2008 May;58(3):205-11. doi: 10.1093/occmed/kqn013. Epub 2008 Feb 27. PMID: 18308694.