



IARS' International Research Journal
ISSN: 2202-2821
ISSN: 1839-6518
iars.research@gmail.com
International Association of Research Scholars
Organismo Internacional

Delayed Inhalational Injury due to Accidental Muriatic Acid Poisoning

Naga Siri, Kavuru; Kanna, Suresh; Shanmuganadan, K.

Delayed Inhalational Injury due to Accidental Muriatic Acid Poisoning

IARS' International Research Journal, vol. 11, núm. 2, 2021

International Association of Research Scholars, Organismo Internacional

Disponible en: <https://www.redalyc.org/articulo.oa?id=663872670003>



Esta obra está bajo una Licencia Creative Commons Atribución 4.0 Internacional.

Delayed Inhalational Injury due to Accidental Muriatic Acid Poisoning

Kavuru Naga Siri

Sree Balaji Medical College and Hospital, India

Suresh Kanna

Sree Balaji Medical College and Hospital, India

K. Shanmuganadan

Sree Balaji Medical College and Hospital, India

IARS' International Research Journal,
vol. 11, núm. 2, 2021

International Association of Research
Scholars, Organismo Internacional

Revisado: 28 Julio 2021
Aprobación: 15 Agosto 2021
Publicación: 29 Agosto 2021

Redalyc: [https://www.redalyc.org/
articulo.oa?id=663872670003](https://www.redalyc.org/articulo.oa?id=663872670003)

Abstract: Muriatic acid is the commonly used toilet bowl cleaner in India. It is delivered industrially and is utilized for cleaning, pickling, electroplating metals, in refining mineral metals, in petrol well extraction, in cowhide tanning, in the refining of fats, cleansers, and consumable oils. Inhalation is the most common exposure of muriatic acid contamination. In this article, medical studies about a case of delayed inhalational injury due to muriatic acid poisoning has been reported.

Keywords: Safety, Inhalation Injury, Acid Gas, Acid poisoning.

I. INTRODUCTION

Muriatic acid is the commonly used toilet bowl cleaner in India. It is delivered industrially and is utilized for cleaning, pickling, electroplating metals, in refining mineral metals, in petrol well extraction, in cowhide tanning, in the refining of fats, cleansers, and consumable oils. Inhalation is the most common exposure of muriatic acid contamination.

Inward breath is a significant course of exposure to any gas poisoning and same may also cause Acid Poisoning by exposure to Acid Gas.

II. The Case History

A 37-year-old male patient sanitary worker by profession, came to the OPD with complaints of difficulty in breathing and chest discomfort of 2 hours duration. On direct inquiry he gave history of accidental inhalation of toilet cleaner while working in his office 2 days prior to onset of symptoms followed by exposure to fumes of bathroom cleaner. He also complained of palpitations, headache, nausea. He had no co morbid illnesses. His blood pressure is 130/90 mm of Hg, pulse rate of 96/ min and respiratory rate of 26/ min spO2 was 90% on room air. Examination of respiratory system revealed bilateral crackles at both lung bases. Other systemic examination was unremarkable.

Investigations showed complete blood picture as normal and chest x ray had bilateral non homogenous lung fields. ABG respiratory

failure type 1. Patient was managed with antibiotics and treated symptomatically.

III. Acid Gas and Fumes

Hydrogen chloride is a dreary, destructive, nonflammable gas that exhaust in air with sharp scent. Hydrogen chloride isn't ingested through the skin, yet when hydrogen chloride gas interacts with dampness, it structures hydrochloric corrosive, which is destructive and can cause disturbance and consumes.

Inward breath is a significant course of exposure. Its fume is heavier than air and may cause suffocation in encased, ineffectively ventilated, or low-lying zones. Youngsters presented to similar levels as grown-ups may get bigger portion since they have more prominent lung surface area. Standards and Guidelines: promptly hazardous to life or wellbeing 50 ppm. Practically everything people could be presented for as long as 1 hour without encountering or creating irreversible or other genuine wellbeing impacts or manifestations which could hinder a person's capacity to make a defensive move 20 ppm. Brief openness to 35 ppm causes throat bothering, and levels of 50 to 100 ppm are scarcely okay for 60 minutes. The best effect is on the upper respiratory tract; openness to high fixations can quickly prompt expanding and fit of the throat and suffocation. Most genuinely uncovered people have quick beginning of fast breathing, blue shading of the skin, and narrowing of the bronchioles. Patients who have monstrous openings may build up a gathering of liquid in the lungs. Openness to hydrogen chloride can prompt Reactive Airway Dysfunction Syndrome (RADS), an artificially or aggravation-initiated sort of asthma. After intense exposure, respiratory work by and large revisitations of benchmark in 7 to 14 days. Albeit complete recuperation is normal, manifestations and delayed pneumonic shortages can persevere. Patients may create Reactive Airways Dysfunction Syndrome (RADS).

IV. The Exposure

Exposure of the eyes to concentrated hydrogen chloride fume or hydrochloric corrosive can cause corneal cell passing, waterfalls, and glaucoma. Openness to weaken arrangements can cause stinging agony and wounds, for example, ulcers of the eye surface.

Exposure or delayed openness to hydrogen chloride has been related with changes in pneumonic capacity, persistent aggravation of the bronchi, nasal ulceration, and manifestations looking like intense viral disease of the upper respiratory tract just as irritation of the skin, staining and disintegration of dental polish, and aggravation of the eye film. Chlorosis may happen with delayed openness.

Muriatic corrosive isn't ingested through the skin. Direct contact with fluid arrangements of hydrogen chloride or with concentrated fume can cause serious substance consumes. Ingestion of concentrated

hydrochloric corrosive can make extreme destructive injury the lips, mouth, throat, throat, and stomach, dying, hole, scarring, or injury arrangement, torment, trouble gulping, queasiness, and vomiting. Liver harm and ischemia might be observed. Renal disappointment and nephritis may happen. Ingestion of concentrated hydrochloric corrosive or huge skin openness to either hydrochloric corrosive or hydrogen chloride gas may cause low circulatory strain because of gastrointestinal draining or liquid relocation.

V. Discussion

The determination of intense hydrogen chloride harmfulness is basically clinical, considering side effects of the destructive activity of the gas or corrosive. All normal examinations to be done. If respiratory-parcel disturbance is available, screen with chest radiography and heartbeat oximetry (or ABG estimations).

There is no cure for hydrogen chloride harming. Treatment comprises of help of respiratory and cardiovascular capacities. Positive-pressure, independent breathing mechanical assembly is suggested accordingly circumstances that include openness to conceivably dangerous degrees of hydrogen chloride. If the patient is seriously uncovered Quickly access for a patent aviation route, guarantee satisfactory breath and heartbeat rate. Control supplemental oxygen as required. Flushing of uncovered eyes with lukewarm plain water or saline for 15 minutes.

Administration of supplemental oxygen by veil to patients who have respiratory side effects treating patients who have bronchospasm with aerosolized bronchodilators ought to be finished. Consider the soundness of the myocardium prior to picking the type of bronchodilator ought to be regulated. Cardiovascular sensitizing can be possible.

VI. CONCLUSION

As prevention is better than cure, it is suggested that use of safety precautions and switching over to non-irritant disinfectant is preferable in any case of exposure to Acid Gas and Acid Poisoning and in any such case an immediate healthcare support is advised.

VII. REFERENCES

- (NIOSH) National Institute for Occupational Safety and Health. Criteria for a recommended standard: Occupational exposure to nitric acid. Springfield (VA): National Technical Information Service; 1976. DHEW (NIOSH) Pub No. 76-141; NTIS Pub No. PB 81-227-217. [Google Scholar]
- Lee-Chiong TL, Jr Smoke inhalation injury. Postgrad Med J. 1999; 105:55–62. [PubMed] [Google Scholar]
- Hajela R, Janigan DT, Landrigan PL, Boudreau SF, Sebastian S. Fatal pulmonary edema due to nitric acid fume inhalation in three pulp-mill workers. Chest. 1990; 97:487–9. [PubMed] [Google Scholar]

- Meduri GU, Golden E, Freire AX, Taylor E, Zaman M, Carson SJ, et al. Methylprednisolone infusion in early severe ARDS: Results of a randomized controlled trial. *Chest*. 2007; 131:954– 63. [PubMed] [Google Scholar]
- Sadegh Soltan-Sharifi M, Mojtahedzadeh M, Najafi A, Reza Khajavi M, Reza Rouini M, Moradi M, et al. Improvement by N-acetylcysteine of acute respiratory distress syndrome through increasing intracellular glutathione, and extracellular thiol molecules and anti-oxidant power: Evidence for underlying toxicological mechanisms. *Hum Exp Toxicol*. 2007; 26:697– 703. [PubMed] [Google Scholar]