ISSN: 2515-8260, Volume 09, Issue 07, 2022

AN INTRESTING CASE OF CHEMICAL PNEUMONITIS DUE TO ACCIDENTAL INHALATION OF HF ACID FUMES

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ABSTRACT

Hydrofluoric (HF) acid is an extremely powerful and corrosive inorganic acid. It is used in many industrial branches. Ingestion of solution or inhalation of fumes and vapors with unintentional and intentional exposures of hydrofluoric acid presents with dermal burns, eye injury, acute respiratory symptoms. A 27year old male, polishing factory worker by occupation,with no significant past medical history presented with complaints of accidental inhalation of acid fumes at work place followed by breathlessness, chest tightness and cough. Laboratory investigations showed significant hypocalcemia. HRCT thorax showed diffuse extensive patchy areas of ground glass opacities and consolidations involving bilateral lung fields. Patient was treated with oxygen therapy, antibiotics, steroids, intravenous calcium supplementation and nebulizations. HF acid provokes potential life-threatening systemic toxicity and organs failure. This imposes an extraordinary caution and great awareness of health consequences when using, and requires implementation of all personal and general protective measures.

INTRODUCTION

Hydrofluoric (HF) acid is an extremely powerful and corrosive inorganic acid and a vigorous dehydrating agent.It is used in many industrial branches like in the cleaning of bricks, the etching of semiconductor microchips, the tanning of leather, the fabrication of aluminium, and the manufacture of fire extinguishers[1]. Domestic concentrations of hydrofluoric acid are typically around 0.5 percent with industrial concentrations approaching 100 percent. It penetrates into deep tissue causing liquefactive necrosis and release of cellular products. Ingestion of solutions or inhalation of fumes and vapors with unintentional and intentional

ISSN: 2515-8260, Volume 09, Issue 07, 2022

exposures of hydrofluoric acid presents with dermal burns, eye injury, acute respiratory symptoms due to pneumonitis. It causes systemic fluoride toxicity including cardiovascular, pulmonary, renal and neuromuscular symptoms, electrolyte imbalance and enzyme inhibition which can lead to cardiac arrhythmias and death [2] Hydrofluoric acid is highly toxic and damaging to humans due to the "double danger" properties of the corrosive nature of the hydrogen ions and toxic effect due to the ability of fluoride ions to penetrate into deep tissues. Chronic symptoms may occur or persist for months after HF ingestion or respiratory exposure [2]. Unintentional exposure of hydrofluoric acid in and out of the workplace include inappropriate operations, mechanical failure of equipment, explosions of containers and tanks containing HF, during traffic incidents with leakage of HF, inadequate protective equipment and children accidentally exposed through ingestion of domestic cleaners[3-5]

CASE REPORT

A 27year old male, polishing factory worker by occupation, with no significant past medical history was brought to emergency with complaints of accidental inhalation of acid fumes at work place followed by sudden onset breathlessness MMRC grade 3 associated with chest tightness, cough with expectoration (whitish) and difficulty in swallowing. On examination Tachycardia(122bpm), Tachypnoea(32cpm), Blood pressure (130/90mmhg) and oxygen saturation (85% on Room air). On auscultation bilateral coarse crepitations and rhonchi heard.

Laboratory investigations revealed mild leucocytosis, and significant hypocalcemia, hypomagnesemia rest with in normal limits. Electrocardiogram showed prolonged QTc interval. Arterial blood gas analysis showed significant hypoxia suggestive of type 1 respiratory failure. Chest X-ray PA view showed multiple inhomogenous opacities in bilateral lung fields. HRCT thorax showed Diffuse extensive patchy areas of ground glass opacities and consolidations involving bilateral lung fields likely suggestive of acute hypersensitivity pneumonitis.

Patient was started on intermittent NIV, oxygen therapy at 8 litres, antibiotics and steroids (Inj Hydrocort 100mg IV TDS and gradually tapered in 5 days), IV calcium supplementation (Inj Calcium gluconate 6^{th} hrly for 3 days), Nebulizations with Budecort, N-acetyl cysteine, intravenous fluids and later oral calcium supplementation was started after 3 days. Treatment was continued and patient gradually improved over a week and oxygen requirement has reduced and patient was shifted to ward and discharged after 3 weeks.

INVESTIGATION	VALUE
Hemoglobin	11.1gm%
Total leucocyte count	13,200 (70% neutrophils)
Platelets	1,40,000/mm^3
Total Bilirubin	1.1mg/dl
Direct Bilirubin	0.7mg/dl
Aspartate transaminase	19U/L
Alanine transaminase	20U/L
Alkaline phosphatase	57U/L

ISSN: 2515-8260, Volume 09, Issue 07, 2022

Urea	20mg/dl
Creatinine	0.9mg/dl
Serum Sodium	140 mEq/L
Serum Potassium	3.5 mEq/L
Serum Calcium	6.5 mg/dl
Serum Magnesium	1.6 mg/dl
Serum Phosphorous	3.4 mg/dl
Arterial blood gas (ABG) on room air	
pH	7.32
PO2	56
PCO2	38
HCO3	18.6
Urine routine microscopy	Normal
Proteins	Nil
RBCs, Pus cells	Nil

Table 1: Laboratory investigations at presentation

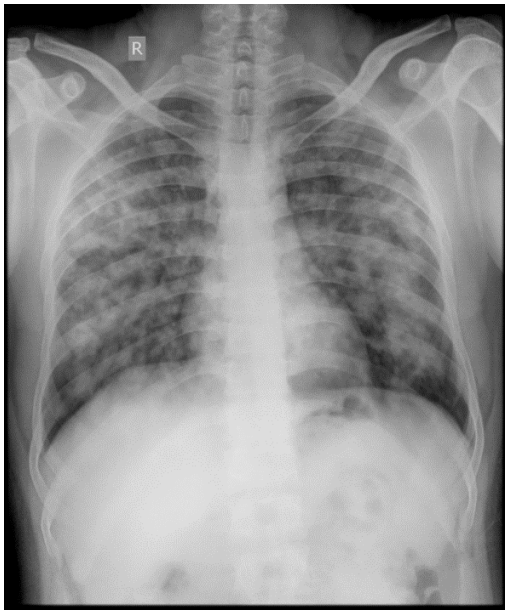
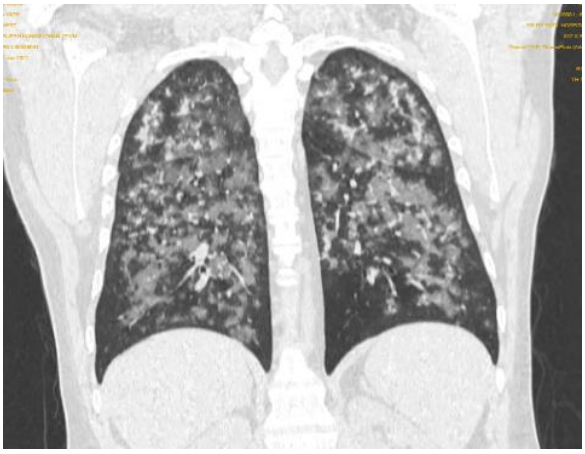


Figure 1- Chest Xray PA showing multiple inhomogenous opacities in bilateral lung fields



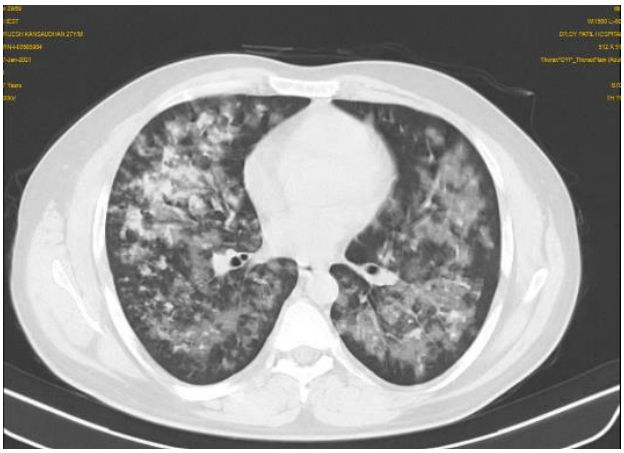


Figure:2 **Figure :3**

Figure 2,3: High resonance computed tomography (HRCT) showing diffuse extensive patchy areas of ground glass opacities and consolidations involving bilateral lung fields likely S/0 Acute hypersensitivity pneumonitis.

DISCUSSION

Hydrofluoric acid is characterised by its corrosiveness and high local and systemic toxicity. At room temperature (20°C), it has a strong acidic pH-value of 2.0 [6]. The devastating effects are not due to the low pH value, but on the toxicity of this acid [7].

There are three different ways through which HF acid can be absorbed into the human body - skin/eye contact, inhalation and ingestion. The most frequent exposure is by cutaneous contact [8][9]. It could also be absorbed through eyes[10]. Inhalation intoxication occurs not only from exposure to hydrogen fluoride gas[11],but also from vapors arising from concentrated hydrogen fluoride liquid [11], while ingestion of a small amount of this acid is likely to produce systemic effects and may be fatal. Serious systemic toxicity may arise from burns bigger than 25 square inches (160 square cm). The activity of corrosive hydrogen ion when using a high concentration of this acid (>50%) and is associated with cutaneous and ocular lesions, as well as digestive and respiratory mucous membrane damage.

The cytotoxic fluoride anion responsible for local and systemic toxicity when HF acid products with high, as well as with low concentrations have been used [12]. The fluoride ion is very small and absorbed into the bloodstream, it is carried to all body organs in proportion to their vascularity and fluoride concentration in the blood [13]. When reacting with cellular calcium and magnesium, forms insoluble chelates, CaF_2 and MgF_2, thus provoking local calcium depletion and inhibition of Na^+K^+ ATP-ase pump. This causes hypocalcemia, hypomagnesemia, hyperkalemia or hypokalemia, fluorosis and metabolic acidosis [14].

Respiratory exposure of HF acid fumes-

Symptoms from respiratory system may occur when exposed to HF gas, fumes or vapours. The toxicological effect of HF gases or vapours on the airway epithelia depends on the

ISSN: 2515-8260, Volume 09, Issue 07, 2022

inhaled doses: when exposed up to 1.5 mM HF, no toxic effect has been observed; repairable damage to the epithelial cells has been detected when inhaled 7.5 mM HF, while severe, irreversible damage has been caused by 75 mM inhaled HF gas [15].

Inhalation of toxic gases or vapours provokes nasal irritation and inflammation, dryness and mucosal bleeding with subsequent ulceration and/or perforation of nasal septum, erythema and odema of the oral, nasal end laryngeal mucous membrane. Continued exposure can results in coughing, dyspnea, laryngitis, laryngospasm and retrosternal pain, followed by chills, fever, and cyanosis and can cause tracheobronchitis, bronchiolar obstruction and bleeding accompanied by stridor and wheeze. Gaseous HF when reaches the pulmonary tissue provokes pulmonary oedema and congestion, pleural effusion, pneumonia. [13].Chest radiograph revealed pulmonary oedema or diffuse infiltrative shadows over the lungs' parenchyma.[16]

Management

Correct diagnosis and timely treatment are of great importance when one is exposed to HF acid. There are several methods that can be used for decontamination and neutralisation of the exposed skin and hair like rinsing with water, saline or solution of soap and water, and neutralisation performed with calcium gluconate, benzalkonium chloride, polyethylene glycol, magnesium oxide. Topically applied, calcium gluconate has limited ability in term of chelating the F^- ions that have been already penetrated deeply into the skin tissues. The recommended dose is limited to 0.5 mL/cm^2 affected skin surface area of a 5% or 10% calcium gluconate solution (with a maximum of 0.5 mL per digit for finger burns) [17].

Treatment after HF inhalation

After inhalation of HF gases, fumes or vapours, the affected person should be immediately given 100% oxygen (10 to 12 L/min flow rate) and a bag-valve-mask for assistant ventilation. Calcium gluconate solution, 2.5% - 5%, given by intermittent positive-pressure ventilation using a nebuliser is the therapy of choice when starting the hospital treatment. Aerosolised bronchodilator should be administered in the patients with bronchospasm considering the myocardial condition; the risk of cardiac arrhythmias (especially in the elderly) should be estimated. Aspiration and lavage of the affected bronchi or lungs may also be performed [19] the respiration is compromised (in case of oedema, laryngospasm and hypoxemia), an airway may be secured via endotracheal intubation [20]. Continuous renal replacement therapy, hemofiltration or hemodialysis, should be conducted as an effective and potentially lifesaving treatment in patients with severe systemic toxicity. Additionally, glucocorticoid (methylprednisolone 40mg/8h) and antibiotic to prevent bacterial infection should be administered.

CONCLUSION

The person who uses HF acid should be aware of the toxicity of this agent and be familiar with all information and procedures regarding the safety when using, way of transporting and storing the acid, managing with HF containing waste, decontamination procedures, antidote and medications that should be used in case of contact and intoxication. Electrolyte replacement therapy, fluid resuscitation, bronchodilators, glucocorticoids and antibiotics, vasopressors is the treatment of choice. Extracorporeal membrane oxygenation to improve oxygenation and to support hemodynamic profile in case of acute respiratory distress

syndrome or cardiac arrest, and renal replacement therapy, to remove serum fluorides, are sometimes necessary procedures to sustain life in severe fluoride intoxication.

REFERENCES

1. Bajraktarova-Valjakova E, Korunoska-Stevkovska V, Georgieva S, Ivanovski K, Bajraktarova-Misevska C, Mijoska A, Grozdanov A. Hydrofluoric acid: burns and systemic toxicity, protective measures, immediate and hospital medical treatment. Open access Macedonian journal of medical sciences. 2018 Nov 11;6(11):2257.
2. McKee D, Thoma A, Bailey K, Fish J. A review of hydrofluoric acid burn management. Plast Surg (Oakv). 2014 Summer;22(2):95-8.
3. Ozsoy G, Kendirli T, Ates U, Perk O, Azapagasi E, Ozcan S, Baran C, Goktug A, Dindar H. Fatal Refractory Ventricular Fibrillation Due to Ingestion of Hydrofluoric Acid. Pediatr Emerg Care. 2019 Nov;35(11):e201-e202.
4. Gradinger R, Jung C, Reinhardt D, Mall G, Figulla HR. Toxic myocarditis due to oral ingestion of hydrofluoric acid. Heart Lung Circ. 2008 Jun;17(3):248-50.
5. Vohra R, Velez LI, Rivera W, Benitez FL, Delaney KA. Recurrent life-threatening ventricular dysrhythmias associated with acute hydrofluoric acid ingestion: observations in one case and implications for mechanism of toxicity. Clin Toxicol (Phila). 2008 Jan;46(1):79-84.
6. http://www.ivoclarvivadent.us/zooluwebsite/media/document/4176/IPS+Ceramic+Etching+gel+
7. Makarovsky I, Markel G, Dushnitsky T, Eisenkraft A. Hydrogen fluoride - the protoplasmic poison. Isr Med Assoc J. 2008;10(5):381–5.
8. Bartlett D. Dermal exposure to hydrofluoric acid causing significant systemic toxicity. J Emerg Nurs. 2004;30(4):37
9. Hatzifotis M, Williams A, Muller M, Pegg S. Hydrofluoric acid burns. Burns. 2004;30(2):156–9.
10. Atley K, Ridyard E. Treatment of hydrofluoric acid exposure to the eye. Int J Ophthalmol. 2015;8(1):157–61.
11. Zierold D, Chauviere M. Hydrogen fluoride inhalation injury because of a fire suppression system. Mil Med. 2012;177(1):108–12.
12. Upfal M, Doyle C. Medical management of hydrofluoric acid exposure. J Occup Med. 1990;32(8):726–31.
13. Martínez MA, Ballesteros S, Piga FJ, Sánchez de la Torre C, Cubero CA. The tissue distribution of fluoride in a fatal case of self-poisoning. J Anal Toxicol. 2007;31(8):526–33.
14. Onohara T, Komine M, Yoshidomi Y, Amari K, Fujita R, Matsumoto Y, Koyama T, Koga M, Mitsumizo S, Satake Y, Masumoto K, Matsunaga T, Mae T, Fujita N. Chemical burn caused by high-concentration hydrofluoric acid: a case that followed a lethal course.
15. Mathieu L, Burgher F, Constant S, Huang S, Hall AH, Blomet J, Fortin J-L, Schrage NF. Hydrofluoric acid: update about knowledge on eye/skin and

respiratory exposure damages and decontamination. Occup Environ Med. 2018;75(Suppl 2):A19

16. Kono K, Watanabe T, Dote T, Usuda K, Nishiura H, Tagawa T, Tominaga M, Higuchi Y, Onnda M. Successful treatments of lung injury and skin burn due to hydrofluoric acid exposure. Int Arch Occup Environ Health. 2000
17. Yoshimura CA, Mathieu L, Hall AH, Monteiro MG, de Almeida DM. Seventy per cent hydrofluoric acid burns: delayed decontamination with hexafluorine®and treatment with calcium gluconate. J Burn Care Res. 2011;32(4):e149–54.
18. Pu Q, Qian J, Tao W, Yang A, Wu J, Wang Y. Extracorporeal membrane oxygenation combined with continuous renal replacement therapy in cutaneous burn and inhalation injury caused by hydrofluoric acid and nitric acid. Medicine. 2017;96(48):e8972.
19. Zierold D, Chauviere M. Hydrogen fluoride inhalation injury because of a fire suppression system. Mil Med. 2012;177(1):108–12.
20. Whiteley PM, Aks SE. Case files of the Toxikon Consortium in Chicago: survival after intentional ingestion of hydrofluoric acid. J Med Toxicol. 2010;6(3):349