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Case Report Open Access

Accidental Ammonia Gas Leakage in a Cold Storage : A Case Report

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Abstract

Introduction: Ammonia is corrosive and exposure will result in a chemical-type burn. It is highly hygroscopic and readily transforms the moist areas of the body such as eyes, nose, throat, and moist skin areas. It is a highly toxic irritant gas, and its toxicity usually occurs from occupational exposure, most are unintentional toxicity. It causes tissue damage via exothermic reaction with body tissues causing liquefactive necrosis. Release of ammonia has potentially for harmful effects on workers and the public. Although there have been incidents of exposure to harmful concentrations of ammonia in the world there have been few fatal accidents.Presentation depends on the level and duration of exposure. Management is supportive and its antidote is not available yet.

Presentation of Case: Here, we present a case of accidental workplace acute ammonia intoxication. We report a case of a 38-yearold male with accidental workplace exposure from coolant damage and leakage. He developed cough and sputum and blurring of vision. He was unable to speak. Had intermittent episodes of vomiting on the same day. The patient also complained of throat pain which is associated with difficulty in eating, drinking & speaking. The patient was presented to the emergency department on nasal prongs. A muti- disciplinary approach and prompt management with respiratory support was initiated. He had to be managed with a high-flow nasal cannula with subsequent tapering of the oxygen supplementation with titration.

Specific Symptoms, Treatments and Outcome: Inhaling ammonia causes irritation in the upper respiratory system. Exposure 50 ppm or more results in immediate irritation to the nose and throat, but tolerance to ammonia develop with repeated exposure. Acute exposure to higher levels of 500 ppm is very dangerous for life. Accidental exposures of high concentrations of ammonia gas have resulted in nasopharyngeal and tracheal burns, airway obstruction and respiratory distress, and bronchiolar and alveolar edema. Ammonia vapor readily dissolves in the moisture present on the skin, eyes, oropharynx and lungs forming ammonium hydroxide which dissociates to yield hydroxyl ions. Chronic occupational exposure to low levels of ammonia in air (<25 ppm) had little effect on pulmonary function but decrease in lung function parameters. Ammonia is also slightly irritating to human eyes in a brief exposure at concentrations of 100 ppm. Acute symptoms are inflamed eyes, swelling abrasions, and of the eyelids, hyperemic conjunctiva, blurred vision, possible transient blindness,

corneal sustained corneal damage. Exposure to an air concentration of 250 ppm is bearable for most persons for 30–60 minutes. The acute ammonia exposure resulted spasms of muscles and also affects the nervous system.

Results: The 38 years old patient is stable and at the time of discharge had normal vitals after treatment.

Discussion: Inhalation injury due to ammonia toxicity can have varied presentations from mild to severe life- threatening respiratory distress and neurological deterioration. Complications can range from upper airway edema to pulmonary edema.

Conclusion: Ammonia inhalational injury may be life threatening. It is important to have a multi- disciplinary approach and in-hospital management of ammonia toxicity as well as to raise awareness regarding the work environment and timely recognition of the possible occupational health hazards.

Keywords: Cryopreservation; Ammonia; Pulmonary Edema

Introduction

Ammonia is a colourless pungent odour highly water soluble irritant gas producing ammonium ion in water. Ammonia is synthesized de-novo within the body as part of many biochemical reactions including bacterial enzymatic activities and is quickly metabolized to non-toxic urea by the liver and excreted by kidneys to neutralize its high toxicity effect. The normal blood level of ammonia in a healthy adult is in the range of 15–45 μg/dL and its toxicity occurs once its blood level surpasses the liver's ability to detoxify it. Ammonia exposure occurs from occupational ammonia gas exposure like in production storage, transportation facilities, exposure to ammonia-based fertilizers, household and industrial cleaners, cooling refrigerants, decaying manure via ingestion, inhalation, direct contact with skin, or contact with the eye. Most of the ammonia toxicities are unintentional with only 9.2% being intentional ones and accidental ingestion was the most common mode of toxicities among children. Ammonia causes tissue damage via exothermic reaction with body tissues producing ammonium ions and liquefactive necrosis with deeper burns. So, exposure to ammonia gas can damage eyes, skin, lungs, and gastrointestinal tract causing mucosal injuries, scarring, and even perforation. The objective of the case report is to create awareness regarding the environment and occupational hazards of accidental ammonia inhalation, its symptoms and the requirement of hospital facilities to promptly manage such cases. In-hospital management consisting of multi-disciplinary approach involving experts from different specialties like pulmonologist, ophthalmologist, otorhinolaryngologist, gastroenterologist and is the most effective management protocol that can prevent the disease burden on patients have been deliberated upon.

Case Report

A 38 years male patient, presented to Emergency with respiratory distress following a gas leak accident in a cold storage, when ammonia gas leaked out of the pipeline. He was unable to speak in full sentences with room air SpO2 85%.He also complained of cough with sputum, blurring of vision, throat pain which was associated with difficulty in eating, drinking & speaking and also had intermittent episodes of vomiting on the same day. Patients was then transferred to Critical Care Unit. Patient was received in ICU on a face mask- SpO2-90% on O2@7L/min.

He was agitated and was persistently coughing. On examination: Respiratory system- On auscultation he had B/L Rhonchi and coarse Crepts. Chest X-Ray (PA View) revealed presence of B/L homogenous infiltrates.

Patient was tachypnoeic which was persistently increasing and was not maintaining saturation on face mask, so he was kept on HFNC O2 @50L/min with FiO2 80%. P/F ratio after 12 hours was same as on the time of admission (P/F ratio= 100), so patient

was kept on HFNC for 4 days with FiO2 and flow adjusted as per respiratory rate , P/F ratio and other respiratory findings. Serial Chest X-Rays were done. On Day-3, patient developed new pneumonic patch on right side and Oxygen requirement increased with P/F ratio dropping to 64. Antibiotics were revised (Meropenam, Clindamycin and Azithromycin) and Ketamine nebulisation was started on 8 hourly basis. Inj Acetylcystine was added.

The use of pre-operative ketamine nebulisation reduced the incidence and severity of POST during early post-operative period in patients receiving GA with tracheal intubation. This technique adds to the armamentarium of the anaesthetist in management of the 'little big problem' of POST. Ketamine nebulization significantly attenuated the incidence and severity of POST, especially in the early post-operative period, with no adverse effects. While Acetylcysteine is used for respiratory disease with excessive mucus. It acts as mucolytic (loosen mucus in airways) when used in oral preparations while when used as injection it treats overdose toxicity. Acetylcysteine is a mucolytic medication. It thins and loosens mucus (phlegm) making it easier to cough out. It also works as an antidote in paracetamol poisoning by replenishing the body's stores of glutathione, a chemical that removes toxic substances.

Gradually after 3-4 days of Ketamine nebulisation along with other conservative management, Chest X-Ray, ABG and other respiratory parameters (Oxygen support decreased : HFNC Face-mask nasal prongs) improved. Incentive Spirometry was started and antibiotics tapered.

After 5 days, patient was maintaining well on Room air, B/L Chest Clear on auscultation. Chest X ray and P/F ratio also improved. Patient was then discharged with no respiratory complaints and SpO2 96% on Room Air after 12 days of ICU stay.

On admission-	**Day-3**	**Day-7**	**On Discharge**
B.P-132/96 mmHg	BP- 126/90 mmHg	BP- 116/84 mmHg	BP- 110/74 mmHg
H.R- 111/min	HR-120/min	HR-90/min	HR-82/min
SpO2-90% on O2@ 7L/min	SpO2- 94% on HFNC on O2 @50L/min with FiO280%.	SpO2- 95% on HFNC on O2@30L/min with FiO250%.	SpO2- 96% on Room Air
RR- 40/min	RR- 42/min	RR-24/min	RR- 16/min
GCS-E4V5M6	GCS-E4V5M6	GCS-E4V5M6	GCS-E4V5M6
P/F RATIO			
100	64	218	>400
Chest Auscultation			
B/L coarse crepts and ronchi	B/L coarse crepts	Rightsidedfinecrepts	B/L clear

Table 1: Vitals at Different Times

Discussion

Ammonia toxicity is not an uncommon poisoning that is often seen in the occupational setting as accidental exposure, and so is this case. This is a severe ammonia gas intoxication following several minutes of exposure resulting into irritation of the upper airways, acute b/l infiltrates, pneumonic patch and irritant conjunctivitis. Severe acute exposure to ammonia gas may cause significant damage to respiratory tissues which may be fatal or lead to severe pulmonary impairment often requiring mechanical ventilatory assistance to rarely needing lung transplantation. Our patient did not require such measures despite severe injuries resulting from the prolonged inhalation exposure and managed conservatively. Nevertheless, complications did occur, despite the fact that the victim had been removed quickly from the contaminated environment, admitted to the hospital and treated intensively with

corticosteroids in oral and parenteral administration. In fact, it is known that the lesions caused by ammonia gas inhalation are followed by the installation of a chronic pulmonary disease. The use of steroids is debated however, in patient with respiratory symptoms such as ours, may be justified with the clinical improvement with its use. In a case report by Bhalla et al. They discussed a case of accidental ammonia gas intoxication who was managed with steroids, prophylactic antibiotics, and other supportive measures only and was discharged after 72 hours of ICU stay. Our patient initially showed some improvement but there was deterioration on the third day which subsequently improved after additional antibiotics indicating that superinfection should be considered and managed accordingly in such cases.

Similarly, a case reported by Maries et al [2] developed irritation syndrome of the upper airways, occlusive bronchiolitis and acute toxic pulmonary edema and managed symptomatically including steroids and prophylactic antibiotics, and did not require ventilatory assistance. While cases reported by Koksal et al. and Lali´c et al required mechanical ventilation and lung transplantation respectively.

The case was signalled and declared acute intoxication, occupational disease and work accident. We emphasize the importance of preventing such situations. The workers should be familiar with the work environment, and comply with occupational safety measures; we also stress the importance of the presence of well-trained and equipped rescue teams, able to act quickly if an accident were to occur. Acute respiratory distress syndrome (ARDS) and acute lung injury are common complications following acute moderate to severe exposure which may progress to chronic obstructive airway disease, this often occurs following chronic mild to moderate ammonia gas exposure Thus, following ammonia gas toxic exposure, the long-term follow-up of a patient is required to diagnose and manage the long-term effect of toxic exposure.

The presentation of a patient varies with acute vs chronic intoxication, causing tracheobronchitis, asphyxiation, and even death on acute exposure and bronchiectasis, optic neuropathy on chronic exposure via inhalation. Its diagnosis is done based on circumstances of possible exposure, physical examination findings, and basic and body system focused lab tests while there are no tests for assessing the extent of its toxicity. The management of ammonia toxicity is symptomatic and supportive as there's no antidote for it.

Conclusion

Ammonia gas intoxication is not uncommon and mostly happens as accidental exposure in an occupational setting and has a debilitating effect on different body systems and might cause long morbidity as it lacks a proper antidote. Hence, it is important to raise awareness regarding the environment and occupational safety measures such as proper ventilation of workplace for prevention and availability of hospital facilities to promptly manage such cases. In-hospital management requires multi - disciplinary approach involving experts from differentspecialities like pulmonologist, ophthalmologist, otorhinolaryngologist, gastroenterologist, and is the most effective management protocol that can prevent the disease burden on patients. The medical team and the emergency department play a major role in the diagnosis and management of patients exposed to ammonia inhalation. The key to successfully managing ammonia exposure and reducing long-term sequelae is timely interventions. The team can obtain valuable insights into the exposure environment and may provide the first clue to ammonia poisoning. They can initiate decontamination before arriving in the emergency department. The emergency clinician, after primary and secondary surveys, should initiate early consultation for ophthalmology, gastroenterology, a burn nurse, and plastic surgery depending on the need. Early specialist intervention improves patient outcomes. Based on the exposure, the patient may need long-term follow-up care. Follow-up with a pulmonologist may be necessary for patients with severe acute inhalation, as the development of chronic lung disease is common. Long-term follow-up with a gastroenterologist may be necessary for patients with ingestion to screen for the development of stricture.

In patients with intentional or suicidal exposure, psychiatric consultation may be advisable early in the management of such cases.

Nursing will provide inpatient care and monitoring, evaluate the patient progress on follow-up outpatient visits, and report their findings to the treating clinician. Ammonia toxicity requires an interprofessional team approach, including primary clinicians, specialists (based on exposure type), and specialty-trained nurses all collaborating across disciplines to achieve optimal patient results.

References

1. Bhalla A, Mahi S, Sharma N, Singh S (2011) Glycopyrrolate in toxic exposure to ammonia gas. Journal of Emergencies, Trauma, and Shock 4: 140-1.

2. Maries L, Maries M (2018) Ammonia intoxication at the workplace - a case report. J. Ecophysiol. Occup. Health 18: 103-5.

3. Koksal O, Almacioglu ML, Ozdemir F, Bulut M, Akkose Aydin S (2011) A case report of ammonium sulfate inhalation. J. Acad. Emergency Med 180-2.

4. Lalic H, Djindjic-Pavicic M, Kukuljan M (2009) Ammonia intoxication on the workplace - case report and a review of literature. Coll. Antropol 33: 945-9.