Case Report

Accidental exposure to ammonia gas: what should one do?

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ABSTRACT

Ammonia (NH_3) is a highly water-soluble, colourless, irritant gas with a unique pungent odour. Liquid ammonia stored under high pressure is still widely used for refrigeration in cold stores used for storing grains. Severe toxicity following accidental exposure is a likely possibility. We report accidental exposure to ammonia gas in workers at a cold storage and its management.

Key words: Ammonia, toxic gas, inhalational injury, vocal cord injury, hoarseness of voice, toxic gas inhalation.

INTRODUCTION

Ammonia as gas is widely used in the production of explosives, pharmaceuticals, pesticides, textiles, leather, plastics, pulp and paper. It also finds its use in rubber industry, petroleum products, and is a major component of many common household cleaning / bleaching products (e.g., glass cleaners, toilet bowel cleaners, metal polishes, floor strippers, wax removers, smelling salts).[1,2,3] Ammonia use as refrigerant gas has declined in developed counties but liquid ammonia under high pressure is still widely used for refrigeration in cold stores. Ammonia (NH_3) is a highly water-soluble, colorless, irritant gas. It has a unique odour which helps its identification in case of spills / accidents. Although the pungent odour prevents very severe exposure but severe toxicity following accidental exposure to a very high concentration of ammonia is a likely possibility.

We report seven cases of accidental exposure to ammonia treated at our centre after a spill at a cold storage.

Case Report

Seven male patients reported to the emergency medical OPD attached to the Nehru Hospital at PGIMER, Chandigarh after the pipe carrying ammonia burst in the cold storage where they were working. All the victims ran away from the place of accident (primary decontamination) and were taken to an open space by their colleagues working outside the place of accident. They all started complaining of severe burning in the eyes and throat. Secondary decontamination was performed at the site by using water to wash the eyes. Within few minutes some of them complained of difficulty in breathing. They were brought to the emergency by their colleagues. There was a delay of approximately 2 hours between the development of first symptoms and reporting to our centre.

All the victims were male in the second and third decade of lives. The mean age was 32.2 years. All of them complained of severe burning in the eyes, skin and difficulty in breathing immediately. Four patients were chronic smokers and exhibited severe bronchospasm on examination. The rest had mild wheezing. Three patients had excessive drooling of secretions from the oral cavity. All the patients complained of hoarseness of voice which was due to vocal cord oedema documented on indirect laryngoscopy performed by experienced ENT specialist. In all the patients skin was decontaminated with fresh water and the eyes were decontaminated with saline till symptomatic relief. All of them received injectable steroids (hydrocortisone), beta 2 agonists by nebulisation and high flow oxygen. One patient required shifting to the high dependency unit because of severe hypoxia secondary to severe bronchospasm. The CT scan of Chest and Pulmonary function tests was normal in all patients after 48 hours. All except one were discharged after 72 hours of observation and one patient was discharged on day 5 of admission.

DISCUSSION

Injury from ammonia most commonly is caused by inhalation, it also may follow ingestion or direct contact with eyes or skin.[1] Ammonia gas causes damage when anhydrous ammonia (liquid or gas) reacts with tissue water to form the strongly alkaline solution, ammonium hydroxide. This exothermic reaction causes significant thermal injury. It may also result in severe alkaline

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chemical burns to skin, eyes, and upper respiratory tract.[1,2] Mild exposures primarily affect the upper respiratory tract. Prolonged exposure to low concentration of the gas tends to affect the entire respiratory system.[4] the most important step in prevention of injury is decontamination. The primary decontamination includes shifting the patient away from the site of exposure. All the victims in our cohort did perform primary decontamination by running away from the site of the accident, therefore, prolonged exposure was prevented in all of them. Since the gas vapours may remain trapped in the clothes and ammonium hydroxide present on the skin / mucous membranes will continue to cause damage, secondary decontamination is as important. Removal of the clothes and washing of the skin with copious amount of water will remove most of the toxin. This was undertaken as the patients arrived in our emergency. Decontamination of eyes and skin was carried out till there was symptomatic improvement.

Ammonia is highly water soluble, it has a tendency to be absorbed by the water-rich mucosa of the upper respiratory tract. Unlike most highly water-soluble irritant gases that tend to affect exclusively the upper respiratory tract, ammonia can damage the respiratory tract both proximally and distally.[1] On exposure to ammonia gas, ammonium hydroxide is formed in the upper airway causing tissues to breakdown and liberates more water, thus perpetuating the conversion of ammonia to ammonium hydroxide. This can result in continued exposure and irritation of the respiratory tract resulting in excessive salivation. Excessive secretions can compromise the airway and may also result in drooling of saliva.

Since all the victims had a mild exposure they all had predominant upper respiratory tract symptoms. Four patients with history of exposure to cigarette smoke had severe bronchospasm and excessive secretions from the upper respiratory tract. One of them required admission to the high dependency unit. The possible explanation for this could be exaggerated response to the ammonia gas due to the hyper reactive airway. None of our patients demonstrated severe airflow limitation on pulmonary function tests. The possible reason for this could be the timing of the test. Since pulmonary function tests could be performed in all after a delay of 48 hours and during this period all the manifestations reversed on treatment.

In case of a brief ammonia exposure, damage generally is limited to the upper airway mucosa. Brief exposures at very high concentrations and continuous exposure at low concentration, can be overwhelming and affect the entire respiratory system.[1,4] In the respiratory tract, this results in the destruction of cilia and the mucosal barrier to infection. Furthermore, secretions, sloughed epithelium, cellular debris, oedema, and reactive smooth muscle contraction cause significant airway obstruction especially in a person having a reactive airway disease.[4] None of our patients developed symptoms referable to lower respiratory tract. This was also corroborated by the normal findings on the chest x ray and the CT scan done in these patients.

Oropharyngeal and retrosternal pain, hoarseness of voice, dysphagia and loss of consciousness are the other reported symptoms. Hoarseness and dysphagia were present in our patients too. Although mild laryngeal involvement is invariably seen in all, patients with severe and prolonged exposure are at high risk for developing life threatening laryngeal oedema.

The priority in severe toxicity is to keep the airway patent, which can be achieved by medical or surgical methods.[1] Indications for intubation include severe respiratory distress (hypoxemia, hypercapnia), stridor, hoarseness, deep facial burns, burns identified by bronchoscopy or endoscopy, and depressed mental status. In case of severe laryngeal oedema, intubation may be difficult and one has to resort to cricothyrotomy or tracheostomy to open the airway.

One of our patients had stridor, dyspnoea, excessive secretions and altered mental state but we could avoid intubation in him by using a combination of steroids and glycopyrrolate. Inhaled β_2 agonists (salbutamol) in our patients helped in reversing the bronchospasm.

CONCLUSION

We conclude that exposure to ammonia gas can lead to severe respiratory embarrassment. Primary and secondary decontamination remains the cornerstone of therapy. Steroids, oxygen and bronchodilators remain the main stay for managing such patients. Anticholinergic agents can be a useful adjunct to the standard supportive treatment.

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