Case Report
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# Ammonia Inhalation Toxicity: A Case Report

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## Abstract

Inhalational ammonia exposure may occasionally occur in industrial set up. Anhydrous ammonia is a colorless, pungent gas that may be stored or transported in pressurized liquid form. Here we present a case report on accidental ammonia inhalation, its clinical findings and management. A 55 year old male was exposed to inhalation of leaked ammonia gas from the pipeline of a fertilizer factory and came to the hospital after referral within 6 hour after exposure. The patient was presented with mild dyspnea, giddiness, gastrointestinal irritation, conjunctivitis and mucosal burning. The patient was managed aggressively with decontamination, fluid irrigation, IV fluid, steroid, bronchodilators, antibiotics, tear supplement and antibiotics eye drop. The patient was monitored for partial pressure of oxygen, oxygen saturation, pH, ECG, electrolytes, liver enzymes, renal function, CBC and chest X-ray. The patient was discharged in normal condition with follow up advice after 5$^{th}$ day of admission. Immediate decontamination with maintenance of airway, breathing and circulation is crucial followed by monitoring is key for the management. Immediate conservative management of these patients led to good prognosis; otherwise it is usually fatal within first 4-6 hour of exposure.

**Keywords:** Ammonia, inhalation, industry, clinical findings, management, prognosis.

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## 1. Introduction

Ammonia is a colorless irritant gas with a pungent odour. In industry it is used in mining, plastic, explosives and refrigerants etc.[1] About 80% of the ammonia produced by industry is used in agriculture as fertilizer. In pure form it is known as anhydrous ammonia and is hygroscopic. It has alkaline properties and is corrosive. Ammonia gas is easily compressed and forms a clear liquid under pressure.[2] Most people are exposed to ammonia from inhalation of gas or vapors. The wide spread use of ammonia on farms, industry and commercial locations set the exposure to occur from an accidental release or from a deliberate terrorist attack.[2] Ammonia interacts immediately upon contact with available moisture in the skin, eyes, oral cavity, respiratory tract and particularly mucus surfaces to form the very caustic, ammonium hydroxide. Ammonium hydroxide causes necrosis of tissue through saponification leading to cellular destruction.[2]

The immediate effect of ammonia depends upon the route of exposure, whether it is inhalation, skin or eye contact or ingestion. Inhalation produces upper airway irritation with symptoms of lacrimation, cough, dyspnea, convulsion, coma and death. There is glolttic and laryngeal edema, sloughing of bronchial mucosa and chemical pneumonitis with pulmonary edema. Inhalation of the gas at concentration of above 5000 ppm can be rapidly fatal. Ocular exposure can result in lacrimation, conjunctivitis, corneal irritation and temporary or permanent blindness.[1] Exposure to high concentration of ammonia from swallowing results in corrosive damage to the mouth, throat and stomach.[2,3]

So far the management of acute ammonia exposure is concerned, there is no antidote available for its poisoning, but ammonia's effects can be treated and most people recover as per the previous literature. Immediate decontamination of skin and eyes with copious amount of water is important. Treatment consists of supportive

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measures and can include administration of humidified oxygen, bronchodilator, and airway management. Ingested ammonia is diluted with milk or water and managed like a weak alkali corrosive poisoning. Antibiotics and steroids may be used as and when required.[1,2,3]

There is usually no role of laboratory tests of ammonia exposure in emergency management, though it can be detected in blood or urine sample.[3] Inhalational ammonia exposure is rarely found in this part of the country and the present case report may solidify the clinical assessment and management protocol in future exposure.

## 2. Case

A 55yr old male patient was brought to the Emergency Department (ED) of Apollo Hospital, Bhubaneswar, Odisha which was referred from a Government Hospital nearby with a history of exposure to ammonia gas six hours before. The patient was exposed to the ammonia gas for approximately 30 seconds in a fertilizer industry from a leaked pipe line over the ground and preliminary managed at the local hospital, followed by Govt. hospital and finally referred to the ED of Apollo Hospital. A written consent from the patient regarding scientific presentation / publication of his data and photographs was obtained during the hospital stay.

Chief complain at the time of presentation to the ED were burning sensation of eye, oral cavity, urethra and anus, difficulty in breathing and foreign body sensation within oral cavity. On examination of the patient there was bilateral reddening of eye with lacrimation, mucosal surface of oral cavity, urethra and anus were redden, uvula was red and edematous. There was hoarseness of voice. Other findings were BP= 130/84 mm Hg, pulse-94/min. respiratory rate=20/min, $SpO_2$= 98% with 100% $O_2$, normal sinus rhythm,. On systemic examination CNS- conscious, oriented, pupil normal size and reacting to light, GCS= 15. CVS= $S_1$ $S_2$ (+ve), no murmur. RS= B/L VBS, no added sound (crepitation, ronchi, stidor, wheeze). Abdomen= Soft, non-tender, BS(+ve).

The patient was advised for CBC, ECG, ABG, serum electrolytes, LFT, RFT, ophthalmic consultation, chest X-ray and urine analysis. The immediate treatment administered to the patient was decontamination (removal of exposed garments and undergarment), irrigation of the exposed parts (eye, urethra, anus) with normal saline periodically, tear supplement with antibiotics eye drop, nebulization with salbutomol and ipratropium bromide with humidified O2, IV fluids, antacids, hydrocortisone and antibiotics. Patient was advised nil orally.

ECG demonstrated as sinus tachycardia, chest X-ray was normal without any infiltration. Complete blood count showed normal findings. Random blood sugar was found 108 mg%. LFT and RFT showed normal level of parameters. Serum-electrolytes were within normal range. Urine analysis showed normal findings. ABG analysis showed pH=7.374, $PO_2$=88, $PCO_2$=39.1, HCO3=22.7 mmol/L, SpO2=98.7% , BE= 2.1mmol/L.

Tear supplement and antibiotics eye drop advised every hourly and monitoring of ECG, ABG, serum electrolytes every four hourly for the first 24 hour. Adequate oxygenation and saturation was maintained. Respiratory obstruction or dyspnea was taken care off. The patient was shifted to indoor after six hour of emergency management. Upper GI endoscopy was done which was normal except some inflammation on proximal mucosa.

The same conservative management was continued for the next 48 hour and all the symptoms subside except some gastrointestinal irritation and conjunctivitis. Oral feeding with liquid food then started and the patient was discharged on 5$^{th}$ day of admission without any symptoms except some conjunctivitis. Ophthalmic consultation was given and was uneventful and the patient was discharged with ophthalmic drop, multivitamins with advice of soft diet, rest and follow up after 15 days or whenever required.

## 3. Discussion

Widespread use of ammonia in industry and agriculture led to its exposure to humans in various forms.[4] The clinical manifestations of acute ammonia exposure are usually immediate in presentation and its toxic effects are mediated through its irritant and corrosive properties.[5] The exposure may be in the form of inhalation, ingestion and local (dermal, ocular).[4,5,6,7] In the current case report the incident occurred in a fertilizer industry leaked from a gas pipe leading to inhalational exposure of ammonia.

After inhalation of ammonia the damage to the respiratory tract depends on the concentration, pH, duration of exposure and depth of inhalation.[8] Concentration of $NH_3$ < 700 ppm is irritating to skin, eye and mucous membrane, concentration > 1500 ppm shows pulmonary edema, coughing and laryngospasm, concentration 2500-4500 ppm is fatal within 30 minutes

of exposure and conc. > 5000 ppm is rapidly fatal due to airway obstruction.[2,6] In our case the inhalation exposure was from the ground level pipe for 30 seconds. The exposure may be less than 700 ppm as per the clinical findings suggest.

Prompt irrigation of eyes for 5-10 seconds is required and immediate treatment of airway and pulmonary injury required after removal from the exposure site.[4] In this case the patient was removed from the exposed site in 30 sec and first aid given at the local hospital before referring to our hospital.

Mild inhalation exposure show coughing, hemoptysis pharyngitis, laryngitis, tracheobronchitis. Severe injury led to pulmonary edema, bronchiectasis, and ocular injury in the form of lacrimation, conjunctivitis, palpebral edema, blepharospasm, and photophobia.[6] Chemical burns are also caused due to anhydrous ammomia especially in high concentration. In our case we found there was lacrimation and conjunctivitis on eyes, burning sensation over the mucosa (nasal, oral, urethral, anal). Foreign body sensation was felt due to edematous uvula and hoarseness of voice due to laryngitis. Systemic affects following acute exposure to high concentration of ammonia include an elevated pulse and blood pressure, cardiac arrest, cyanosis and hemorrhagic necrosis of the liver.[2] In our patient we found sinus tachycardia and slightly raised blood pressure.

Clothing must be removed quickly, and irrigation with water initiated at the scene and continued for the first 24 hour. Patient with significant facial or pharyngeal burns should be incubated and eyes irrigated.[6,7] In this case the clothing was not removed until the patient reaches our centre. So the patient complained of burning pain of urethral and anal orifices. The irrigation of affected part including eye was continued for the next 24 hour. The patient was managed conservatively and did not required intubation with monitoring of ABG, ECG, X-Ray ophthalmic consultation, UGI Endoscopy.

Complications may develop in the form of chemical pneumonia, chronic lung disease, bronchopneumonia[9], severe metabolic acidosis, convulsion, respiratory failure[7], persistent airway obstruction, bronchiolitis obliterans, bronchiectasis[6], acute respiratory failure with differ lung parenchyma and airway involvement.[10] In our case the patient did not show any sign/symptoms of respiratory failure or airway obstruction. It was probably due to lower concentration and or less time of exposure.

Prognosis depends upon concentration, pH, duration of exposure and depth of inhalation[8], presence or absence of abnormal chest findings on admission[4], early decontamination/ irrigation of the effected part.[6] In our case the patient was exposed for less time and normal chest X-ray findings on admission probably led to good prognosis. The patient should be discharged with follow up instruction as attached format.[2] In our case the patient was discharged on 5$^{th}$ day of admission with proper follow up instruction.

## 4. Conclusion

Accidental exposure of leaked gases in industrial set up may be a suspected ammonia inhalation poisoning. Patients usually present with mucosal irritation, conjunctivitis and dyspnea. Prompt decontamination with irrigation of the exposed parts at the scene and subsequently is required. The basic approach is to stabilize the airway, breathing and circulation along with monitoring of $pO_2$, $SpO_2$, electrolytes, ECG, chest-X ray etc. Prognosis may depend upon the concentration of ammonia, duration of exposure, early decontamination and prompt management of airway.

## 5. Conflict of Interest

None Declared

## 6. References


1. Pillay VV. Modern Medical Toxicology. Asphyxiant Poisons. Toxic Gases.4th Ed.2013. Jaypee. India: 351-354.
2. Agency for Toxic Substances and Disease Registry. Accessed on Jan 2016. (Available at http://www.atsdr.cdc.gov/substances/toxsubstance.asp?toxid=2).
3. Centers for Disease Control and Prevention. Accessed on Jan 2016. (Available at http://emergency.cdc.gov/agent/ammonia/index.asp).
4. Arood R, Hammond J, Ward GG. Ammonia Inhalation. Journal of Trauma-Injury Infection & Critical Care. 1985; 25: 444–447.
5. Public Health England. Ammonia Toxicological Overview. Published: August 2015. (Available at https://www.gov.uk/government/publications/ammonia-properties-incident-management-and-toxicology).
6. Amshel CE, Fealk MH, Phillips BJ, Caruso DM. Anhydrous ammonia burns case report and review of the literature. Burns, 2000; 26(5): 493–497.

7. Koksal O, Almacioglu ML, Ozdemir F et al. A Case Report of Ammonium Sulfate Inhalation. JAEM.2011; 10: 180-182.
8. National Poisons Information Service (NPIS). Ammonia. TOXBASE®, 2014 (accessed 04/2014.)
9. Price SK, Hughes JE, Morrison SC et al. Fatal Ammonia Inhalation A Case Report With Autopsy Findings. S Afr Med J.1983; 64: 952-955.
10. Flury KE, Dines DE, Rodarte JR et al. Airway Obstruction Due to Inhalation of Ammonia. Mayo Clinic Proceedings. 1983; 58(6): 389-393.