

# Bronchiectasis - A Delayed Sequela of Occupational Hazards: A Rare Case Report

Dr. Muskan Gollen<sup>1</sup>, Dr. Swadip Mishra<sup>2</sup>, Pratima Singh<sup>3</sup>, Pravati Dutta<sup>4\*</sup>

<sup>1</sup>Department of Respiratory Medicine, Kalinga Institute of Medical Sciences (KIMS), Bhubaneswar  
Email: muskangollen8[at]gmail.com

<sup>2</sup>Department of Respiratory Medicine, Kalinga Institute of Medical Sciences (KIMS), Bhubaneswar  
Email: swadip[at]gmail.com

<sup>3</sup>Professor, Department of Respiratory Medicine, Kalinga Institute of Medical Sciences (KIMS), Bhubaneswar  
Email: pratima.singh[at]kims.ac.in

<sup>4</sup>Professor, Department of Respiratory Medicine, Kalinga Institute of Medical Sciences (KIMS), Bhubaneswar  
Email: dr.pravatidutta[at]gmail.com

\*Corresponding Author

Prof. Pravati Dutta

dr.pravatidutta[at]gmail.com

**Abstract:** Ammonia is a highly irritant gas with a pungent odor. Pressurized ammonia is used as a refrigerant for storage and transport purposes. Acute events following ammonia exposure usually are manifested as cough, dyspnoea, laryngeal edema, pulmonary edema, acute respiratory distress, and acute lung injury (ALI) whereas post - exposure events may lead to the development of chronic obstructive pulmonary disease (COPD), bronchiolitis obliterans and bronchiectasis as long term sequelae. In this reported case, a 45 - year - old non - smoker male had accidental workplace exposure to ammonia for an hour. He had been managed with mechanical ventilatory support due to the development of acute respiratory distress syndrome (ARDS). After being recovered from acute complications, he was discharged from the hospital. Then he developed recurrent episodes of cough and dyspnea. He was diagnosed to have developed COPD and bronchiectasis as a long - term sequela of accidental massive inhalational exposure to noxious gas (anhydrous ammonia). Usually mucosa of upper respiratory tract absorbed maximal anhydrous ammonia protecting the lower respiratory tract from injury. But this patient developed bronchiectasis as a chronic manifestation of massive anhydrous ammonia gas exposure whose incidences are not known.

**Keywords:** Inhalational injury, noxious gas, anhydrous ammonia, bronchiectasis, COPD

## 1. Introduction

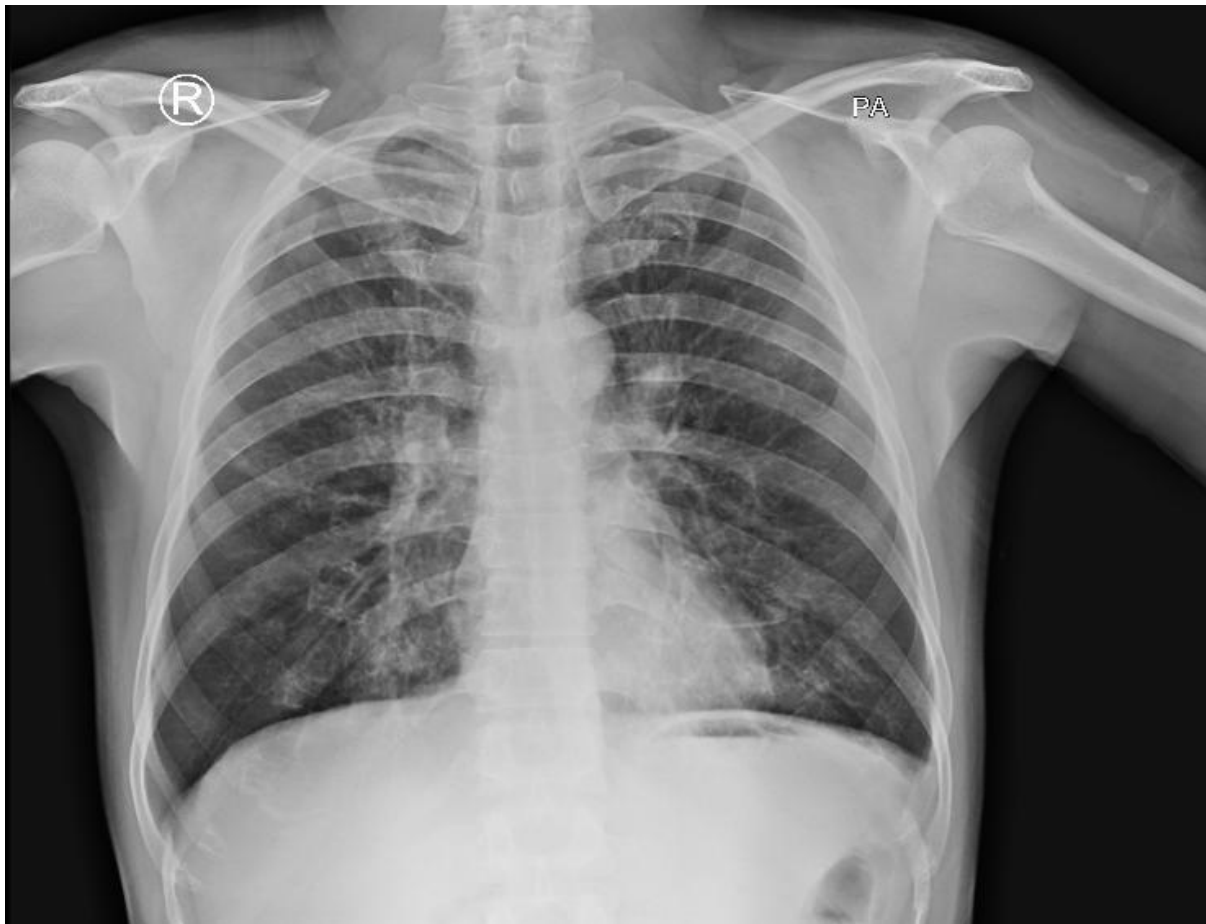
Ammonia (NH<sub>3</sub>) is a highly irritant gas with a pungent order. Ammonium ion (NH<sub>4</sub><sup>+</sup>) is formed when ammonia dissolves in water [1]. It is commonly used as a refrigerant and fertilizer in agriculture. Pressurized ammonia is used for storage and transport purposes. Patients with acute exposure to anhydrous ammonia complain of eye irritation, running nose, scratchy throat, cough, chest tightness and dyspnea. Acute complications include tracheobronchitis, pulmonary edema, laryngitis, bronchopneumonia, ALI and ARDS. Patients who recovered from acute complications, develop long - term sequelae such as airway hyper reactivity, bronchiectasis [2-5], COPD [6, 7], and bronchiolitis obliterans [8, 9]. This reported case developed bronchiectasis after massive exposure to noxious gas (anhydrous ammonia) as long - term sequelae [4].

## 2. Case

A 45 years old male, non - smoker, laborer nondiabetic and normotensive presented with complaints of cough with expectoration, shortness of breath and low - grade intermittent fever for 5 days. 14 years back at the age of

31 years, he had accidental exposure to noxious gas (ammonia) for an hour while working in the cold storage area of a factory in the southern part of Odisha. He lost consciousness, got admitted to a nearby hospital and was managed with mechanical ventilatory support. After recovery and discharge from the hospital, the patient repetitively had dyspnea and cough. Over the years he frequently suffered from cough with foul - smelling expectoration along with shortness of breath. There was history of frequent hospitalization at various health care centers.

14 years after exposure he got admitted to our hospital (KIIMS, Bhubaneswar) with acute exacerbation of respiratory symptoms. On physical examination, he had tachypnea, pallor and digital clubbing. On auscultation, there were bilateral coarse leathery crepitations and polyphonic rhonchi over the entire lung fields. Arterial blood gas analysis was suggestive of respiratory acidosis with adequate compensation at 96% saturation with PaO<sub>2</sub> - 85 mm of Hg. Sputum culture suggested *Pseudomonas aeruginosa*. Chest X - ray was suggestive of bilateral hyperinflation (Fig.1).

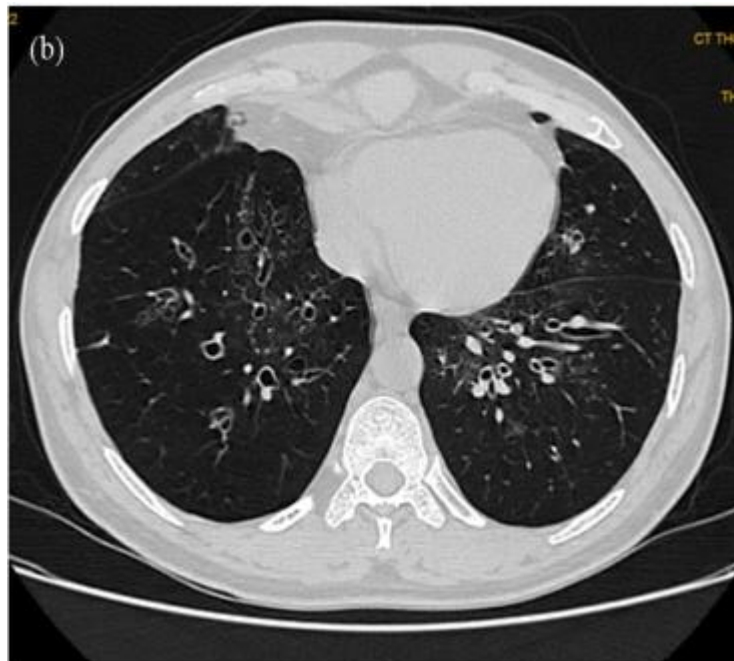


**Figure 1:** Chest X - Ray (PA View) - Showing hyperinflation.

HRCT thorax showed evidence of bronchiectatic changes and mosaic attenuation with bronchial wall thickening in bilateral lung fields (**Fig 2 a**) and centrilobular nodules with tree-in-bud distribution in left lower lobe (**Fig 2 b**). Spirometry was done which showed severe obstruction with no reversibility. Serum alpha-1 antitrypsin level,

rheumatologic panel and sweat chloride test were negative. Sputum culture for fungi and mycobacteria showed no growth. *Aspergillus precipitans* were not detected. After ruling out all possibilities of central bronchiectasis, he was diagnosed to have COPD with bronchiectasis due to noxious gas exposure (anhydrous ammonia).





**Figure 2:** (a) HRCT - Thorax suggestive of centrilobular bronchiectasis and mosaic attenuation in the bilateral upper lobe and (b) HRCT - Thorax suggestive of centrilobular bronchiectasis changes in bilateral lower lobe and tree in bud appearance in left lower lobe

### 3. Discussion

At the tissue level, ammonium ions are formed when ammonia reacts with water. This reaction is exothermic. The formation of alkaline solution causes liquefactive necrosis of the surrounding tissues via saponification of fat and protein denaturation. Thus causing significant thermal injuries to surrounding tissue [10, 11].

Moderate to massive exposure to anhydrous ammonia gas causes severe dermal, ophthalmic and life - threatening respiratory injuries. Severity of inhalation injury is determined by concentration of toxic gas and duration of exposure time to it [11]. Exposure to small concentrations of gas causes tracheobronchitis and laryngitis whereas massive exposure leads to non - cardiogenic pulmonary edema, respiratory arrest, severe laryngeal edema and even can cause ALI / ARDS. ALI cases might die from other complications like bacterial pneumonia [3, 4]. But long - term respiratory complications include COPD, bronchiolitis obliterans, bronchiectasis, and interstitial pulmonary disease [2, 12, 13]. Usually mucosa of upper respiratory tract absorbs maximum anhydrous ammonia, thus protecting the lower respiratory tract from injury. But this patient developed bronchiectasis as a chronic manifestation of massive anhydrous ammonia gas exposure whose incidences are not known [14].

In this reported case, he had accidental exposure to anhydrous ammonia gas followed by a syncopal attack, which led to significant respiratory injuries. As a sequelae this patient developed COPD and bronchiectasis which caused severe obstructive ventilatory dysfunction, confirmed via computed tomography of the chest and spirometry.

### 4. Conclusion

Within two weeks of massive exposure to anhydrous ammonia gas, most of the acute manifestations resolved but few may develop long - term sequelae such as bronchiectasis and fixed obstructive ventilatory dysfunction. These complications result from chemical or infectious injuries or tissue destruction, where the causes are unclear. These patients need long - term frequent hospitalization. The patients diagnosed with bronchiectasis of unknown origin, questioning regarding noxious gas (e. g. ammonia, hydrogen chloride, phosgene, sulfur dioxide, ozone) exposure should be enquired.

#### Acknowledgment

We take this opportunity to express our gratitude towards, Department of Radiodiagnosis, Kalinga Institute of Medical Sciences, Bhubaneswar.

#### Declaration of Interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

#### Funding Details

There are no funding details to be declared.

#### Consent

A written informed consent was obtained from the patient for publication of this case report and accompany images.

#### Declaration of Patient Consent

The authors certify that they have obtained all appropriate patient consent forms. In the form the patient (s) has/have given his/her/their consent for his/her/their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be

published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

## References

- [1] A. J. L. Cooper, F. Plum, Biochemistry and physiology of brain ammonia, *Physiol. Rev.*67 (1987) 440–519. <https://doi.org/10.1152/physrev.1987.67.2.440>.
- [2] K. E. Flury, D. E. Dines, J. R. Rodarte, R. Rodgers, Airway obstruction due to inhalation of ammonia, *Mayo Clin. Proc.*58 (1983) 389–393. <https://pubmed.ncbi.nlm.nih.gov/6855275/> (accessed August 9, 2023).
- [3] R. E. De La Hoz, D. P. Schlueter, W. N. Rom, Chronic lung disease secondary to ammonia inhalation injury: A report on three cases, *Am. J. Ind. Med.*29 (1996) 209–214. [https://doi.org/10.1002/\(SICI\)1097-0274\(199602\)29:2<209::AID-AJIM12>3.0.CO;2-7](https://doi.org/10.1002/(SICI)1097-0274(199602)29:2<209::AID-AJIM12>3.0.CO;2-7).
- [4] K. Miller, A. Chang, Acute inhalation injury, *Emerg. Med. Clin. North Am.*21 (2003) 533–557. [https://doi.org/10.1016/S0733-8627\(03\)00011-7](https://doi.org/10.1016/S0733-8627(03)00011-7).
- [5] C. S. White, P. A. Templeton, Chemical pneumonitis, *Radiol. Clin. North Am.*30 (1992) 1231–1243. [https://doi.org/10.1016/s0033-8389\(22\)00864-8](https://doi.org/10.1016/s0033-8389(22)00864-8).
- [6] I. Kass, N. Zamel, C. A. Dobry, M. Holzer, Bronchiectasis following ammonia burns of the respiratory tract. A review of two cases., *Chest*.62 (1972) 282–285. <https://doi.org/10.1378/chest.62.3.282>.
- [7] R. Sobonya, Fatal anhydrous ammonia inhalation, *Hum. Pathol.*8 (1977) 293–299. [https://doi.org/10.1016/S0046-8177\(77\)80026-9](https://doi.org/10.1016/S0046-8177(77)80026-9).
- [8] A. R. Tonelli, A. Pham, Bronchiectasis, a long-term sequela of ammonia inhalation: A case report and review of the literature, *Burns*.35 (2009) 451–453. <https://doi.org/10.1016/j.burns.2008.02.007>.
- [9] F. G. B. G. J. Van Rooy, J. M. Rooyackers, M. Prokop, R. Houba, L. A. M. Smit, D. J. J. Heederik, Bronchiolitis obliterans syndrome in chemical workers producing diacetyl for food flavorings, *Am. J. Respir. Crit. Care Med.*176 (2007) 498–504. <https://doi.org/10.1164/rccm.200611-1620OC>.
- [10] A. H. Osmond, C. J. Tallents, Ammonia Attacks, *Br. Med. J.*3 (1968) 740. <https://doi.org/10.1136/bmj.3.5620.740>.
- [11] R. Arwood, J. Hammond, G. Gillon Ward, Ammonia inhalation, *J. Trauma - Inj. Infect. Crit. Care*.25 (1985) 444–447. <https://doi.org/10.1097/00005373-198505000-00014>.
- [12] N. Brautbar, M. P. Wu, E. D. Richter, Chronic Ammonia Inhalation and Interstitial Pulmonary Fibrosis: A Case Report and Review of the Literature, *Arch. Environ. Heal. An Int. J.*58 (2003) 592–596. <https://doi.org/10.3200/AEOH.58.9.592-596>.
- [13] I. Kim, H. Lee, B. H. Kang, D. H. Lee, Y. H. Nam, M. S. Roh, S. - J. Um, Chronic obstructive lung disease after ammonia inhalation burns: a report of two cases, *Kosin Med. J.*37 (2022) 354–360. <https://doi.org/10.7180/kmj.22.004>.
- [14] M. Gorguner, M. Akgun, Acute Inhalation Injury, *Eurasian J. Med.*42 (2010) 28–35. <https://doi.org/10.5152/eajm.2010.09>.