

# Ammonia Toxicity

## History

The literature on ammonia toxicity in humans largely consists of case reports. In a 1996 literature review, de la Hoz et al found only 94 previously reported cases; of these cases, 20 resulted in fatality and only 35 had clinical follow-up of one year or more. Despite lack of data, most literature is consistent regarding clinical presentation and treatment of ammonia toxicity. Gaseous ammonia effects at various concentrations are as follows:

- 25 ppm or less –TWA
- 25-50 ppm -Detectable odor; unlikely to experience adverse effects
- 50-100 ppm -Mild eye, nose, and throat irritation; may develop tolerance in 1-2 weeks with no adverse effects thereafter
- 140 ppm -Moderate eye irritation; no long-term sequelae in exposures of less than 2 hours
- 400 ppm -Moderate throat irritation
- 500 ppm -IDLH
- 700 ppm -Immediate eye injury
- 1000 ppm -Directly caustic to airway
- 1700 ppm -Laryngospasm
- 2500 ppm -Fatality (after half-hour exposure)
- 2500-6500 ppm -Sloughing and necrosis of airway mucosa, chest pain, acute lung injury (ALI), and bronchospasm
- 5000 ppm -Rapidly fatal exposure

## Inhalation injury

Because of its high water solubility, ammonia has a tendency to be absorbed by the water-rich mucosa of the upper respiratory tract. However, unlike most highly water-soluble irritant gases that tend to affect exclusively the upper respiratory tract, ammonia can damage proximally and distally.

In 1941, Caplin was the first to classify victims of unintentional ammonia exposure; he described them as mild, moderate, and severe. Patients in the mild group presented with conjunctival and upper respiratory inflammation and pain but showed no signs of respiratory distress.<sup>[41]</sup> The moderate group presented similarly but with more exaggerated symptoms. The severe group presented in frank respiratory distress with productive cough, acute lung injury (ALI), and dysphagia. Following a brief ammonia exposure, damage generally is limited to the upper airway mucosa. Brief exposures at very high concentrations, however, can be overwhelming and affect the entire respiratory system. People who are capable of escaping their environment usually are not subject to severe exposures, because they can flee upon detection of ammonia's pungent odor; furthermore, absence of symptoms following inhalational exposure to ammonia essentially rules out significant injury.

- Pain (oropharyngeal, retrosternal)
- Dyspnea, hemoptysis -As expected, individuals with reactive airway disease, such as asthmatics, are particularly sensitive to ammonia inhalation.
- Hoarseness
- Dysphagia
- Loss of consciousness

## **Farming industry**

Industrial injury most often results from either ammonia leaks in fertilizer tanks and hoses or toxic ammonia levels in animal confinement buildings, where ammonia is adsorbed by dust particles that transport it more directly to small airways. Because of this synergistic effect, symptoms have reportedly developed within minutes of entering animal confinement buildings. Symptoms include rhinorrhea, scratchy throat, chest tightness, cough, dyspnea, and eye irritation and usually subside within 24-48 hours.

Farmers exposed to high levels of ammonia may also have a higher likelihood of developing chronic bronchitis and capso, particularly those with underlying atopy.

## **Firefighters**

Firefighters are at risk for exposure to this irritant gas, as ammonia is liberated during combustion of nylon, silk, wood, and melamine.

### **Contact -Burns and cold injury**

Gaseous ammonia combines with water of the skin, eyes, and airways to form ammonium hydroxide. This exothermic reaction results in both heat and chemical burns. Liquid ammonia freezes tissue on contact and may cause full-thickness tissue damage that penetrates deeper than the more conspicuous superficial chemical burns.

Concentrations greater than 10,000 ppm are required to cause skin damage. The eyes begin to feel irritated at concentrations of 50-100 ppm; at 700 ppm, immediate eye damage occurs.

## **Ingestion**

Typical household ammonia products (3-10% ammonium hydroxide) have a pH less than 12.5, although the pH of industrial solutions (up to 30% ammonium hydroxide) is often greater than 13. Because caustic alkali burns generally are thought to occur when pH is greater than 12.5, ammonia ingestions in the home usually do not lead to significant damage. However, Klein et al reported 3 cases of oropharyngeal and esophageal injury following intentional ingestion of household solutions with a pH less than 12.

Patients present with oropharyngeal, epigastric, and retrosternal pain.

Abdominal pain and other gastroenterologic symptoms may occur if ingestion causes viscus perforation (perforation may occur up to 24-72 hours postingestion).

Respiratory symptoms may be present if aspiration pneumonia or pneumonitis complicates ingestion.

Smelling salts are a less common source of household ammonia ingestion. Often in capsule form, smelling salts, which contain approximately 20% ammonia, release a pungent odor when broken. Smelling salts are found in many first-aid kits as a treatment for syncope; unfortunately, children sometimes bite into them, resulting in minor esophageal burns and mild respiratory symptoms.

## Physical

### Inhalation injury

- Head, ears, eyes, nose, throat (HEENT) -Facial and oral burns and ulcerations
- Respiration -Tachypnea, oxygen desaturation, stridor, drooling, cough, wheezing, rhonchi, and decreased air entry
- Central nervous system (CNS) -Loss of consciousness (if exposure is massive)

### Contact -Burns and cold injury

- Skin -Alkali burns to the skin are yellow, soapy, and soft in texture. When burns are severe, skin turns black and leathery.
- HEENT -Burns to the eye penetrate particularly deeply and rapidly, leading to destruction of the inner structures within 2-3 minutes; this may progress to globe perforation. Ammonia typically causes more corneal epithelium and lens damage than other alkalis. Intraocular pressure and pH of the anterior chamber rise, resulting in a syndrome similar to acute narrow-angle glaucoma. Other symptoms include iritis, corneal edema, semi-dilated fixed pupil, and eventual cataract formation.

### Ingestion

- Cardiovascular -With intentional ingestion, hypovolemic shock may occur because of vomiting and thirdspacing of intravascular fluid.
- HEENT -Symptoms include edema of the lips, oropharynx, and upper airway.
- GI -Patient may experience epigastric tenderness; mediastinitis and peritoneal signs may be present with viscus perforation, which can occur as late as 24-72 hours postingestion.
- Respiratory -Aspiration pneumonia and pulmonary edema may occur.