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Septic shock and multiple organ dysfunction syndrome following ammonia-induced caustic injury: case report and literature review

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Abstract

Introduction. Ammonia solution is an aqueous alkali with a pungent odor and is widely used in different settings. This clinical case reports a 75-year-old male who ingested 10 milliliters of 10% ammonia solution. The patient suffered from chemical pneumonitis by inhalation exposure and aspiration which led to secondary bacterial infection, progressing to sepsis, septic shock and multiple organ dysfunction syndrome (MODS).

Case presentation. A 75-year-old male was admitted to Hospital of Lithuanian University of Health Sciences Kaunas Clinics following accidental ingestion of 10 ml of 10% ammonia solution. He initially complained of odynophagia, dysphagia, hypersalivation and dyspnea. Esophagogastroduodenoscopy revealed caustic esophageal injury, while imaging findings were consistent with chemical pneumonitis. Initial laboratory evaluation demonstrated impaired renal function with a need for hemodialysis later on. The patient's clinical condition deteriorated, with the development of acute hypoxemic respiratory failure, worsening acute kidney injury and systemic inflammatory response. He was transferred to the intensive care unit, where endotracheal intubation, mechanical ventilation and vasopressor support were started. The clinical course ultimately led to septic shock and multiple organ dysfunction syndrome, resulting in a fatal outcome.

Conclusions. Ammonia ingestion in elderly patients with significant comorbidities carries a high risk of deep alkali-induced gastrointestinal injury, aspiration-related pneumonia and rapid progression to sepsis and multiorgan failure. Early airway assessment, urgent evaluation and close monitoring are essential to improve prognosis in this high-risk population.

Keywords: ammonia poisoning, aspiration pneumonia, sepsis, septic shock, multiple organ dysfunction syndrome.

1. Introduction

Ammonia is a colorless, volatile gas soluble in water, forming ammonium hydroxide. Ammonium hydroxide is used in various environments, from industrial settings handling anhydrous ammonia to household use of ammonia-containing cleaning or medical solutions. Routes of exposure include inhalation, which may cause respiratory tract irritation, inflammation, edema, burns, severe pain and potentially respiratory distress or failure. Ingestion may lead to nausea, vomiting, chest or epigastric pain, caustic burns, ulceration, perforation or mediastinitis, while direct skin or eye contact causes mucosal irritation and injury [1]. As a strong alkali, ammonium hydroxide causes liquefactive necrosis with deep tissue penetration; the severity of injury is determined by concentration, ingested volume and contact time [2]. Complications vary by severity: mild cases may resolve within 2–3 weeks, whereas moderate cases may progress to hemorrhagic gastrointestinal ulceration, shock, esophageal perforation with mediastinitis, airway edema with stridor dyspnea, fever, secondary bacterial or fungal infection and sepsis [2]. Adult cohorts demonstrate high morbidity and non-trivial mortality after caustic ingestion, emphasizing the need for guideline-directed triage, early evaluation and selective endoscopy [3].

2. Case presentation

A 75-year-old male with a history of multiple strokes and myocardial infarction presented to the Emergency Department in Hospital of Lithuanian University of Health Sciences Kaunas Clinics after accidental ingestion of approximately 10 milliliters of 10% ammonia solution. Baseline post-stroke hemiplegia limited reliable communication which was magnified by the swallowing of the ammonia. He reported throat pain, dysphagia, drooling and dyspnea.

Nasopharyngolaryngoscopy showed oral mucosal burns with inflammation of the soft palate and pharynx, laryngeal and epiglottic oedema with copious secretions. Esophagogastroduodenoscopy demonstrated caustic esophageal injury (Zargar grade IIB), erosive gastropathy and hyperemic duodenitis. Early labs showed increased creatinine (148 µmol/L) and low GFR (39.3 mL/min/1.73 m^2) that may have resulted not only from acute kidney injury secondary to toxic exposure, but also from a possible exacerbation or progression of a previously unrecognized chronic kidney disease, potentially aggravated by comorbid cardiovascular conditions (previous myocardial infarctions or strokes leading to systemic hypoperfusion) or age-related renal structural changes. Patient was conscious, stable but needing additional oxygen therapy. Initial chest X-ray showed reactive changes in lungs and chest computed tomography (CT) showed ground-glass opacities and parabronchial thickening which suggested infiltration or chemical pneumonitis. Later, the patient's condition worsened by acute respiratory distress with rising oxygen demand, growing inflammatory markers, rising creatinine levels and uremic markers. The increase in leukocyte count and CRP while on broad-spectrum antibiotics raised concern for chemical pneumonitis (rather than primary bacterial pneumonia), likely triggered by inhalation and subsequent aspiration of the ammonia solution.

Moreover, hypernatremia emerged (serum sodium 149 mmol/L, later progressing to 160 mmol/L) alongside declining kidney function and acute hypoxemic respiratory failure (oxygen saturation remaining 85% on 10 L/min supplemental oxygen with tachypnea). At the same time, blood pressure fell to 74/51 mmHg (MAP 59 mmHg) and mental status worsened. With an escalating immunocompetent cells number, this multitude of symptoms supported a diagnosis of sepsis despite ongoing broad-spectrum antibiotic treatment. He was transferred to the ICU, where he was intubated for airway protection and oxygenation, sedated and a central venous catheter was inserted to permit vasoactive infusions and hemodynamic monitoring. The patient was febrile, and despite fluid resuscitation required a vasopressor (noradrenaline) for persistent hypotension, consistent with septic shock in the context of aspiration-associated sepsis. For the acute respiratory failure, the patient was placed on synchronized intermittent mandatory ventilation (SIMV) to support respiratory function. The positive-end expiratory pressure (PEEP) increased from 5 to 10 cmH_2O, indicating worsening pulmonary compliance and progression of pneumonia. Concomitant with the deteriorating respiratory status, the patient became anuric with progressively rising creatinine and uremic markers (317 to 399 to 539 μmol/L and 26.7 to 31 to 35.5 mmol/L accordingly). Although diuresis was initially achieved with furosemide, the patient then required hemodialysis. The development of complete renal failure and increasing demand for vasopressors, combined with a history of ammonia poisoning, chemical pneumonitis and pneumonia – accompanied by persistently elevated inflammatory markers despite broad-spectrum antibiotic treatment – was consistent with multiple organ dysfunction syndrome (MODS). Arterial blood gas analysis demonstrated a mixed metabolic and respiratory acidosis (pH 7.21, pCO_2 46.5 mmHg, pO_2 83.6 mmHg, HCO^-_3 17.2 mmol/L with base excess of -9.2 mmol/L).

Despite maximal supportive therapy, including sedation, mechanical ventilation and high-dose vasopressor support, the patient's condition was critically severe and continued to deteriorate. He developed sinus tachycardia of 145 beats per minute (BPM) and hypotension (BP 80/35 mmHg). Despite continuous administration of two high-dose vasopressors and fluids, the patient's heart rhythm progressed from sinus bradycardia to asystole, with unsuccessful adult guideline cardiopulmonary resuscitation.

3. Discussion

Accidental ingestion of concentrated ammonia solution leads to liquefactive necrosis, where enzymes from microbes and lysosomes dissolve cellular components and surrounding tissue, resulting in rapid tissue softening and breakdown [4]. Necrosis of the affected organ wall and overall tissue typically last for about three to four days. Damage consists of inflammation, thrombosis and ulcer formation. Stricture risk depends on injury depth and collagen deposition [5].

Following ingestion, the extent of injury may progress rapidly through the mucosa into submucosal and muscular layers, independent of symptom severity, emphasizing that clinical presentation does not reliably predict injury grade. Liquid alkalis like ammonia hydroxide primarily damage the esophagus due to adherence to its mucosa, rarely reaching the stomach. Aspiration may also cause laryngeal and tracheobronchial injury, as observed in this case [5].

Early endoscopic evaluation within 12 to 24 hours remains the gold standard for staging and management. Endoscopy after 48 hours is discouraged because of increased perforation risk due to tissue friability, so it is advised to do an endoscopy right away after initial patient evaluation with the Zargar classification being the most widely accepted tool for stratification of mucosal damage [2, 6–8]. Computed tomography (CT) can complement endoscopy by identifying transmural or extraluminal injury or perforation and helping to guide surgical decision-making [9].

A similar association between ammonia exposure and kidney injury was reported in a young, previously healthy adult who developed reversible acute tubular necrosis (ATN) requiring short-term dialysis after accidental ammonia poisoning. In that case, renal recovery occurred once the direct toxic and ischemic event resolved. In contrast, our patient's kidney injury developed in the context of concentrated ammonia ingestion, severe caustic injury, aspiration and evolving systemic inflammation – the kidney injury was likely multifactorial, driven by hemodynamic instability, systemic inflammatory response and probable exacerbation of a previous kidney disease. Additionally, advanced age and pre-existing neurologic impairment significantly reduced renal reserve, making the kidney injury more severe. Thus, while both cases demonstrate the nephrotoxic potential of ammonia, the pattern in our patient reflects a broader MODS-related kidney injury, rather than the isolated, fully reversible ATN seen in the younger individual [10].

Prospective research in France containing data of 3544 adult cases with ingestion of substances causing caustic trauma showed that in up to 24% of all observed corrosive ingestion cases, patients suffered from pulmonary complications. Overall, in-hospital mortality rate was 8%. Importantly, older age, comorbidities and proper care administration were the most important factors in general patient mortality and morbidity [11]. Ultimately, another literature review of ingestion of caustic agents described complications across a broad spectrum. The most common complication of GI tract were strictures, hemorrhage and perforation, while systemic complications included disseminated intravascular coagulation (DIC), multi-organ system failure and sepsis [12].

Another study included a total of 176 patients who had ingested various caustic agents, including alkalis, acids and other corrosive substances. Among them, 61 were elderly (over 60 years). The elderly group demonstrated considerably poorer clinical outcomes across multiple parameters. Respiratory involvement occurred in 19 of 61 elderly patients (31.1%), compared with 20 of 115 non-elderly patients (17.4%), making it a leading cause of morbidity in older individuals. Respiratory failure requiring mechanical ventilation was particularly frequent among the elderly, affecting 17 patients (27.9%), nearly twice the rate seen in younger adults. Although the overall occurrence of gastrointestinal complications was similar between the two age groups, older patients experienced a higher proportion of peritonitis or mediastinitis, which developed in 8 elderly patients (13.1%) versus 6 younger patients (5.2%). Antibiotic therapy was required more often among the elderly (39 patients, 63.9%). In addition, mortality was higher, with 14 deaths (23.0%) among the elderly versus 13 (11.3%) in the younger group. Overall, these results highlight that elderly patients are significantly more vulnerable to severe respiratory and systemic complications, prolonged hospitalization, and higher mortality following ingestion of any type of caustic agent [13].

Chemical pneumonitis secondary to ammonia exposure can result from aspiration or inhalation of vapors during ingestion or emesis. Even though aspiration can occur in otherwise healthy individuals, certain comorbidities significantly increase the risk. Post-stroke patients with hemiplegia, as in the presented case, are particularly vulnerable due to impaired bulbar function, weak or absent cough and gag reflexes, and reduced consciousness or coordination of swallowing muscles. Aspiration in this particular population often goes unnoticed. Additional risk factors include advanced age and immobility; all of which were presented in the case [14, 15]. Ammonia, as mentioned before, is a strong tissue irritant soluble in water and mainly damages the upper airways and can extend to the bronchi and alveoli, causing irritation of respiratory tract, chemical burns, bronchospasm, pneumonitis, edema and acute respiratory distress syndrome (ARDS). Management is mainly supportive and it contains additional oxygen therapy, airway protection and ventilation with small tidal volumes if ARDS occurs [1, 16].

In animal (mice) model of ammonia poisoning by inhalation, researchers demonstrated acute respiratory and lung damage. Various inflammatory mediators (such as IL-1β and IL-6) and neutrophils rose in both bronchoalveolar fluid and blood. On day 7, the overall mice condition worsened, showing macrophage infiltration, pulmonary hemorrhaging and endothelial dysfunction with coagulation deviation, while no respiratory collagen increase was observed [17].

In humans, the pathophysiology involves alkaline agents producing protein hydrolysis and saponification of damaged mucosa through interaction with tissue lipids and proteins minutes after ingestion. This chemical reaction causes liquefactive necrosis, allowing the alkali to penetrate progressively through tissue layers and greatly increasing the risk of full-thickness tissue wall injury. Esophageal damage is mainly localized in the middle and lower thirds of the organ, as well as the stomach. The process generates heat and hemorrhagic swelling regarding thrombosis of small vessels, which provokes bacterial colonization to the injured tissue site. Later on, inflammatory response is initiated, further promoting bacterial infiltration. Studies have shown that connective tissue formation of damaged organ is seen only after a few weeks, to which leads to a stricture [18]. The greater surface tension of alkaline substances prolongs their interaction with the esophageal lining, while the stomach's acidic environment partially neutralizes their effect. This difference accounts for the esophagus sustaining more extensive injury than the stomach. For these first two to three weeks, the injured organs' walls are the weakest, so while ulcerations penetrate beyond the muscular layer of the organ, the structural integrity of the wall is compromised, making tissues completely delicate and susceptible to perforation [19].

Management of ammonia poisoning is restricted due to extended inner tissue damage. A few keys to maintain patient‘s stable condition is managing the airway, especially when upper airway is severely damaged on nasopharyngoscopy. If the injured expresses stridor, voice changes, respiratory distress and the saturation is dropping, intubation needs to be performed before airway swelling or collapse. In this case, videolaryngoscopy should be considered to minimize further mucosal injury. Once the airway is secured, persistent hypoxia and a rising alveolar–arterial oxygen gradient should prompt early bronchoscopy to assess lower airway injury. Fluid resuscitation is essential, as oropharyngeal and gastrointestinal burns from caustic ingestion can lead to hypotension due to fluid shifts from the intravascular to the interstitial compartment. Intravenous access must be established, and an initial bolus of isotonic crystalloid solution should be administered. Hemodynamic status should be monitored using standard indicators. Patients who are conscious and can safely swallow, small amounts of water may be given after ingestion to mildly dilute the corrosive substance but, since tissue injury occurs almost immediately, later dilution offers no benefit. Chemical neutralization using weak acids or alkalis should never be attempted, as it can trigger exothermic reactions that exacerbate tissue damage. Use of corticosteroids is clinically inconsistent [20].

In otherwise severe clinical cases, sepsis or multiple organ dysfunction syndrome (MODS) may develop as a consequence of extensive mucosal necrosis, translocation of bacteria or superinfection of pulmonary injury. According to Sepsis-3 definitions and World Health Organization (WHO), sepsis is defined as a life-threatening organ dysfunction caused by a dysregulated host immune response to infection [21, 22]. As per Sepsis-3 and Surviving Sepsis Campaign (SSC) 2021, Sequential Organ Failure Assessment (SOFA) clinically optimizes organ dysfunction, and sepsis is suspected when a suspected or confirmed infection leads to new organ dysfunction (SOFA ≥ 2) [23].

Current SSC 2021 guidelines emphasize the importance of time and within 1 hour, specialists should measure lactate, do early blood cultures, prompt broad-spectrum antibiotic therapy, administrate crystalloids, and point early vasopressor use (preferably norepinephrine) to maintain MAP ≥65 mmHg [24].

In contrast to the clinical case, a case report of a 19-year-old woman who ingested hydrochloric acid (Harpic) and developed a reversible episode of chemical pneumonitis with short-term ventilatory support. The difference of both cases lies both in the type of caustic agent and the patient's vulnerabilities. Hydrochloric acid typically causes superficial coagulative necrosis, and despite presenting with Zargar IIIA injury and transient respiratory failure, the young patient recovered fully without long-term consequences. In our case, ingestion of concentrated ammonia, a potent alkali, resulted in liquefactive necrosis, deeper tissue penetration, and a higher risk of transmural injury, perforation and secondary infection. Moreover, the elderly age, post-stroke bulbar dysfunction, and impaired protective airway reflexes significantly increased the likelihood and severity of aspiration, leading to rapid respiratory deterioration and systemic complications. This comparison highlights how agent chemistry and host factors – especially advanced age and neurologic impairment – profoundly influence the progression, severity and outcomes in caustic ingestion [25].

4. Conclusions

Ammonia ingestion in elderly patients with significant comorbidities and reduced physiological reserve carries a high risk of rapid clinical deterioration. Due to the alkali nature of ammonia and its ability to cause liquefactive necrosis, these patients are particularly vulnerable to deep gastrointestinal injury, aspiration-related chemical pneumonia and progression to sepsis, septic shock and multiple organ dysfunction syndrome. Age-related factors, such as impaired airway reflexes, post-stroke bulbar dysfunction and diminished organ resilience further amplify the severity of complications. Therefore, such patients require urgent evaluation, early airway assessment, and close inpatient or ICU monitoring. Awareness of the pathophysiology and potential outcomes of caustic alkali exposures is crucial for timely intervention and improving the prognosis of this high-risk population.

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