

Severe upper airway obstruction and mediastinitis after accidental quaternary ammonium ingestion: highlighting the role of early parenteral nutrition

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Abstract

Quaternary ammonium compounds (QACs) are widely used disinfectants and surfactants. Poisoning following ingestion is rare but may threaten both airway and upper gastrointestinal integrity. We report a case of A 21-year-old woman with no prior medical history presented after accidental ingestion of a QAC-based disinfectant at work, with vomiting, hematemesis, hypersalivation, and progressive respiratory distress. Examination showed extensive oral mucosal necrosis and impending laryngeal edema requiring early intubation. Laboratory tests revealed leucocytosis, lymphopenia, hypocalcemia, prolonged prothrombin time, and elevated creatine kinase. Contrast-enhanced Computed Tomography (CT) demonstrated moderate esophagitis, mediastinitis, and gastritis with mild peritoneal effusion but no perforation. Treatment included proton pump inhibitors, corticosteroids, broad-spectrum antibiotics, and a total parenteral nutrition protocol ensuring nutritional intake during gastrointestinal rest. Laparoscopic exploration confirmed inflammatory peritoneal effusion without gastric perforation, and recovery was favorable. This case highlights the emergency medicine relevance of QAC ingestion, which may manifest as life-threatening airway compromise with mediastinitis and peritoneal effusion despite the absence of perforation, requiring rapid multidisciplinary management and tailored nutritional support.

Keywords : Caustic ingestion ; Mediastinitis ; Poisoning ; Quaternary ammonium; Nutrition

INTRODUCTION

Quaternary ammonium compounds (QACs) are cationic surfactants used in disinfectants, detergents, and personal care products, and are increasingly encountered in both domestic and occupational environments. Most reports of QAC toxicity describe inhalational or dermal exposure, while clinically significant ingestion remains rare and poorly characterized(1).

When ingested, QAC-based disinfectants behave as caustic agents and may induce liquefactive

necrosis of the oropharyngeal, esophageal, and gastric mucosa, with potential extension to the mediastinum and peritoneal cavity. Only a few case reports have described severe esophagitis, mediastinitis, and airway compromise after benzalkonium chloride or mixed QAC solutions (2–5).

We present an unusual case of accidental QAC ingestion in a young adult, complicated by severe upper airway obstruction and early mediastinitis

with intraperitoneal inflammatory effusion but without gastrointestinal perforation, and we discuss the diagnostic and therapeutic approach.

CASE PRESENTATION

A 21-year-old woman, working in a cleaning company, with no known medical, surgical, or psychiatric history, was brought to the emergency department after accidental oral ingestion of 75ml of an industrial disinfectant at her workplace. The ingested product was Bacticide Plus, a ready-to-use industrial disinfectant spray for non-porous surfaces, formulated with an alcohol base and benzalkonium chloride, with a declared active concentration of 0.1–0.2% benzalkonium chloride according to the manufacturer's datasheet. She had no history of caustic ingestion, substance misuse, or previous Ear, Nose and Throat (ENT) or gastrointestinal disease, and there was no suicidal intent.

Within a few hours after ingestion, the patient developed multiple episodes of vomiting, followed by moderate hematemesis, sore throat, odynophagia, and progressive dyspnea.

On arrival in the emergency department, initial assessment followed the ABCDE approach.

The patient presented with marked hypersalivation, halitosis, and extensive erosive and necrotic lesions of the oral cavity, while ENT examination revealed severe pharyngo-laryngeal mucosal necrosis with an imminent risk of laryngeal edema and upper airway obstruction. She also developed progressive dyspnea with an oxygen saturation of 86% on room air, tachypnea, and signs of respiratory distress, prompting early endotracheal intubation and mechanical ventilation to secure the airway and ensure adequate oxygenation. Hemodynamically, her heart rate was 110 bpm and blood pressure was 100/70 mmHg under crystalloid infusion, with no clinical evidence of shock, and fluid resuscitation was adjusted to maintain stability.

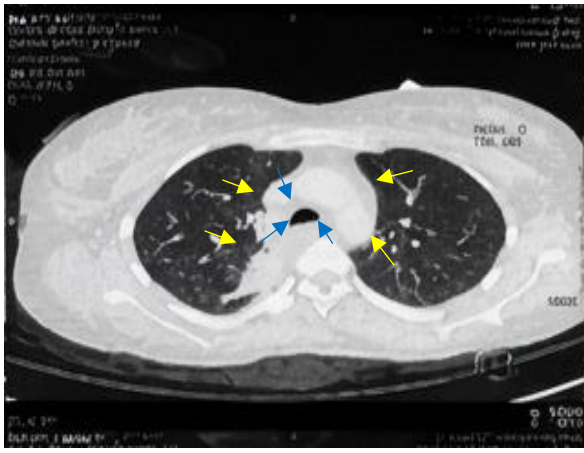
She was initially conscious but anxious, then became progressively drowsy, requiring close neurological monitoring for possible toxic or hypoxic encephalopathy. A complete examination found no cutaneous burns or subcutaneous emphysema, and attention focused on the upper aerodigestive tract and abdominal findings to guide further imaging and possible surgical exploration.

Overall, this clinical assessment prioritized airway protection and respiratory support while simultaneously addressing circulatory status, neurological surveillance, and the extent of caustic exposure, thereby informing subsequent CT imaging, pharmacologic therapy, and nutritional management.

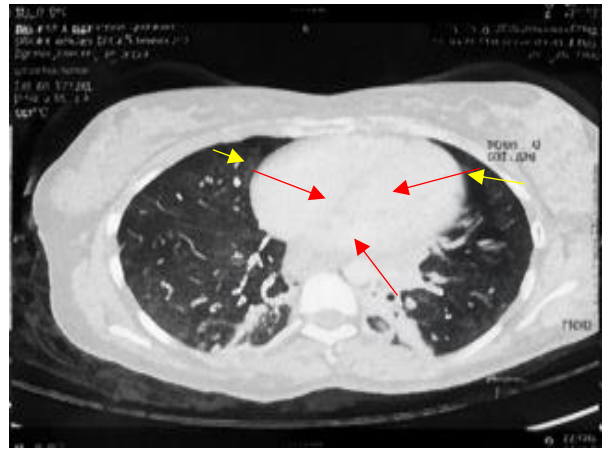
Laboratory evaluation showed leukocytosis with lymphopenia, normal serum electrolytes and creatinine, normal C-reactive protein, prolonged prothrombin time at 51%, mild hypocalcemia at 2.08 mmol/L, and elevated creatine kinase at 312 U/L.

Contrast-enhanced thoracoabdominal CT demonstrated circumferential esophageal wall thickening compatible with moderate esophagitis, mediastinal fat stranding suggestive of mediastinitis, gastric wall thickening with focal mucosal enhancement defects, and a small volume of intraperitoneal effusion, without pneumomediastinum or free intraperitoneal air. CT was used as first-line imaging to assess depth and extension of injury, in line with caustic ingestion recommendations (Figure 1).

Upper endoscopy was deferred because of unstable airway conditions and the risk of worsening mucosal injury in this severely damaged upper tract. No alternative cause such as infectious mediastinitis or peptic perforation was suggested by clinical or imaging findings. Given the progressive clinical and radiological improvement, and after multidisciplinary discussion, no endoscopy was performed during the acute phase, and the patient was scheduled for



A: Upper thoracic CT slice showing esophagitis (blue) and mediastinitis (yellow)



B: Lower thoracic and upper abdominal CT slice showing caustic gastritis (red)

Figure 1. Contrast-enhanced thoracoabdominal CT showing esophagitis, mediastinitis, and gastritis following caustic quaternary ammonium ingestion

delayed endoscopic follow-up to detect late esophageal sequelae.

The patient was intubated early and mechanically ventilated because of progressive upper airway obstruction related to laryngeal edema and persistent hypoxemia. She received high-dose intravenous proton pump inhibitor (omeprazole 80 mg), systemic corticosteroids to reduce laryngeal edema, and broad-spectrum antibiotics (ceftriaxone and metronidazole) to cover mediastinitis and prevent secondary infection.

Because of severe odynophagia, extensive upper gastrointestinal mucosal injury, and the high risk of esophageal and gastric complications, oral and enteral feeding were initially contraindicated. Total parenteral nutrition (TPN) was started within the first 24 hours through a central venous catheter, using a standard three-in-one admixture providing approximately 25 kcal/kg/day and 1.2–1.5 g/kg/day of amino acids, with 20–30% of non-protein calories as lipids and the remainder as glucose. Micronutrients (electrolytes, vitamins, and trace elements) were added according to current critical care recommendations, and infusion was delivered over 24 hours using an infusion pump with strict aseptic precautions. Daily reassessment of weight, fluid balance, serum electrolytes, liver

and renal function, and capillary glucose allowed progressive adjustment of TPN composition and rate, with reduction of parenteral intake as soon as cautious enteral feeding became feasible.

Despite the absence of pneumomediastinum or free intraperitoneal air on CT, the combination of circumferential esophagitis, mediastinitis, gastric wall thickening with mucosal defects, and intraperitoneal effusion raised concern for occult transmural injury. In a multidisciplinary meeting involving intensivists, surgeons, and gastroenterologists, we considered that the risk of missed perforation or rapidly progressing necrosis outweighed the morbidity of a minimally invasive procedure, and diagnostic laparoscopy was therefore selected as a proactive strategy.

Laparoscopic exploration confirmed inflammatory peritoneal effusion without gastric perforation or transmural necrosis, and submucosal edema of the lesser curvature, which was managed by lavage and close monitoring (Figure 2, Figure 3).

ICU care focused on ventilatory support, fluid and electrolyte balance, analgesia, and prevention of nosocomial infections, following current principles of caustic ingestion management.

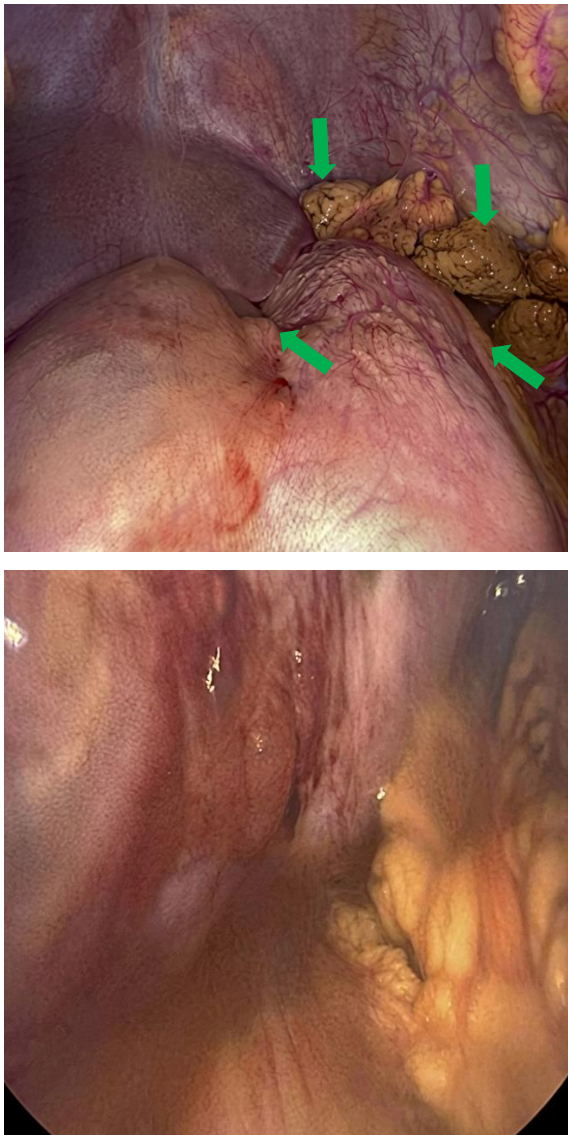


Figure 2. Laparoscopic view showing inflammatory intraperitoneal effusion without visible gastrointestinal leakage (green)

On postoperative day 1, the patient remained hemodynamically stable, with no new respiratory or abdominal complications. Follow-up CT showed regression of esophageal and gastric wall edema and partial improvement of mediastinal and abdominal fat stranding, without new collections or perforation.

Her respiratory status improved, allowing stepwise ventilator weaning, and nutritional support was progressively adapted from parenteral to enteral feeding.

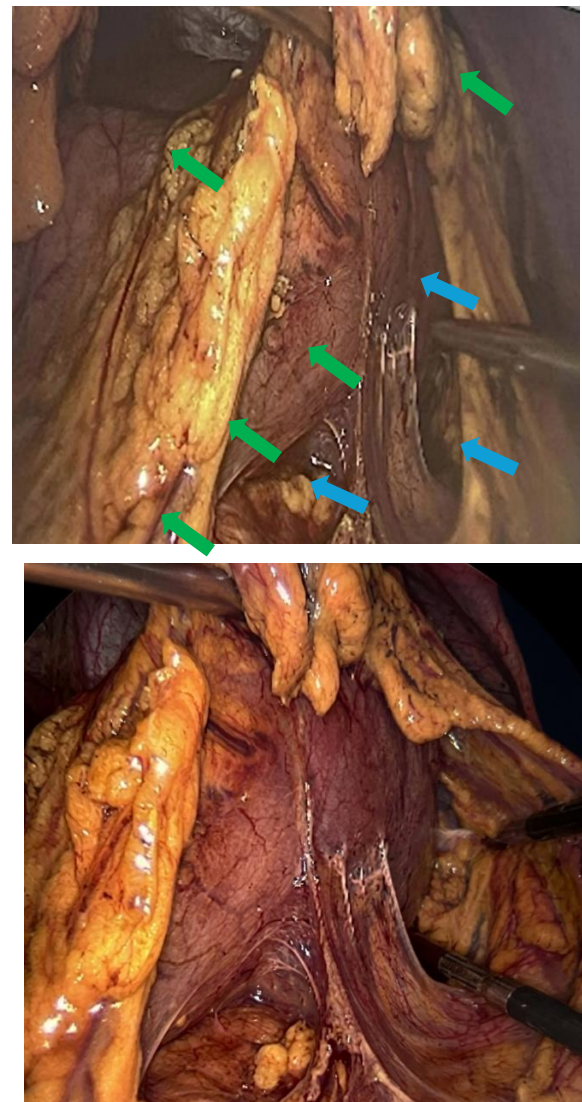


Figure 3. Laparoscopic exploration of the lesser sac confirming the absence of perforation of the posterior gastric wall and the posterior cavity of the omentum (orange)

Long-term endoscopic surveillance was planned to detect late esophageal sequelae.

At 3-month clinical follow-up, the patient reported normal swallowing function, with no dysphagia for solids or liquids and no odynophagia. Her body weight and nutritional markers remained stable, and she had resumed her usual occupational activities without functional limitations. A delayed upper endoscopy performed at month 6 showed no significant esophageal strictures, only mild residual mucosal irregularities in the distal

esophagus and antral region, requiring no endoscopic intervention.

DISCUSSION

This case illustrates an uncommon but severe presentation of QAC-based disinfectant ingestion, combining extensive oropharyngeal burns, high-grade risk of laryngeal edema, mediastinitis, and intraperitoneal inflammatory effusion without documented perforation. Most published QAC poisonings involve inhalation or dermal exposure, while clinically significant ingestion is rarely reported and often associated with benzalkonium chloride or mixed biocide solutions(1,2,4–6).

Several case reports have described benzalkonium chloride or mixed QAC ingestion with severe esophageal or airway injury across different age groups : Kumar et al. reported an adult with dilute benzalkonium chloride ingestion complicated by severe esophagitis and chemical pneumonitis, Civan et al. described corrosive esophagitis due to benzalkonium chloride in a neonate, Nițescu et al. detailed a pediatric case with a particularly severe course, Perera et al. reported laryngeal edema and metabolic acidosis after ingestion, and Spiller described a fatal ingestion of a 10% benzalkonium chloride solution (3,4,6,8).

Compared with these observations, our case is distinctive in combining high-grade risk of upper airway obstruction with CT-documented mediastinitis and intraperitoneal inflammatory effusion in the absence of radiological perforation. Also, in highlighting the role of early laparoscopic exploration and structured nutritional support in an emergency setting, with early mediastinal and peritoneal involvement suggesting deep mucosal and submucosal necrosis with inflammatory spread rather than open transmural rupture and raising questions about thresholds for surgical exploration when CT findings are equivocal, as also discussed in caustic esophagogastritis with mediastinitis managed conservatively (6).

In our patient, the estimated ingested volume of around 75 ml is consistent with severe caustic injury, as larger volumes of QAC-based disinfectants are more likely to produce deep mucosal damage and systemic inflammatory response. Although precise dose–response relationships for benzalkonium chloride ingestion are not well established, this quantity clearly exceeds incidental exposure and supports the severity of the clinical course.

Airway compromise is a major determinant of prognosis in caustic ingestions. In our patient, hypersalivation, extensive oral necrosis, and dyspnea were early warnings of impending airway obstruction, justifying prompt intubation before complete diagnostic workup. This decision is consistent with recommendations that emphasize airway and hemodynamic stabilization as the first priorities in caustic injuries(6,8).

In emergency practice, a systematic ABCDE approach is crucial in caustic ingestions, as it ensures rapid identification and treatment of life-threatening airway and breathing compromise before secondary diagnostic procedures. In our patient, early recognition of airway obstruction risk at the A and B steps led to prompt intubation and ventilation, while simultaneous evaluation of circulation, neurological status, and overall exposure guided fluid management, monitoring, and the decision to proceed to CT imaging and laparoscopy. Emphasizing ABCDE in such toxicological emergencies may therefore improve time-critical decisions and reduce delays in securing the airway and assessing the extent of injury (9).

CT now plays a central role in evaluating caustic injuries, as it can detect wall thickening, fat stranding, and free air or fluid, and help stratify the risk of perforation and guide surgical decisions. In our case, CT findings prompted laparoscopic exploration, which confirmed inflammation without perforation and avoided unnecessary resection (6,8).

In fact, in the absence of radiologically proven perforation, conservative management could have been considered, as described in other series of caustic esophagogastritis with mediastinitis. However, in our case, the coexistence of mediastinal and intraperitoneal inflammation with focal gastric enhancement defects led the team to favor early surgical exploration to avoid delayed recognition of perforation, while still aiming to preserve the stomach and esophagus whenever possible. This decision illustrates the delicate balance between active surgical assessment and close conservative monitoring in severe caustic injuries, and supports an individualized, CT-guided approach.

Management of QAC ingestion is mainly supportive, as no specific antidote exists. Core measures include airway protection, acid suppression, pain control, infection prevention, and early nutritional support. Systemic corticosteroids remain controversial for esophageal injury but may be justified for severe laryngeal edema threatening airway patency, as in this case (1,4,7,9).

In this context, early TPN was used as a temporary bridge to cover energy and protein needs while keeping the upper gastrointestinal tract at rest, in line with recommendations that reserve parenteral nutrition for situations where enteral nutrition is contraindicated or impossible. The chosen regimen, targeting around 25 kcal/kg/day and 1.2–1.5 g/kg/day of amino acids with a balanced lipid–glucose ratio, is consistent with current expert guidelines for critically ill patients and aimed at limiting both catabolism and metabolic complications (11).

CONCLUSION

This case shows that accidental ingestion of quaternary ammonium–based disinfectant can cause severe caustic injury with combined upper airway obstruction, mediastinitis, and significant nutritional vulnerability. Early airway protection, rapid CT-based assessment, and timely surgical evaluation were essential to rule out perforation

and to guide a conservative but closely monitored strategy. In this context of extensive upper digestive tract damage and odynophagia, early, carefully supervised parenteral nutrition played a key supportive role by covering energy and protein needs while allowing functional rest of the injured mucosa. Overall, this observation underlines the importance of a multidisciplinary approach integrating toxicology, emergency medicine, intensive care, surgery, ENT, and nutrition to reduce morbidity and improve short-term outcomes after severe caustic ingestions.

Informed consent

Informed consent was obtained from the patient for publication of this case report and its anonymized images.

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