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Beyond The Gastrointestinal Tract: Unusual External Findings In A Case Of Fatal Battery Acid Ingestion

Tauheed Anwar[1]*

[1]King George's Medical University, Lucknow, Uttar Pradesh, India

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ABSTRACT

Exposure to caustic agents like sulfuric acid and rodenticides remains a major public health concern. While individual ingestion of either agent is well-documented, simultaneous intake is exceptionally rare and poses a heightened risk of rapid systemic toxicity and gastrointestinal devastation.

We report a fatal case involving simultaneous ingestion of sulfuric acid. The patient showed severe corrosion and necrosis of the lips and perioral area, while the esophagus remained intact, reflecting the known resistance of esophageal squamous epithelium to acid damage. The stomach exhibited extensive coagulative necrosis without perforation, likely due to the presence of approximately 80 ml of semi-digested food that may have lessened tissue injury. Corrosive damage extended to the larynx and upper trachea, causing inflammation and epiglottic swelling. These findings indicate aspiration of acid-contaminated gastric contents, leading to aspiration pneumonia, a major cause of death alongside potential laryngeal edema and respiratory arrest.

This case highlights the grave morbidity and mortality risk from accidental combined ingestion of corrosive and toxic agents. It stresses the urgent need for public education on safe storage and handling of hazardous chemicals.

Introduction

Chemical burns constitute approximately 3% of total burn injuries, predominantly arising in domestic and industrial environments [1]. Among these mineral acids, particularly sulphuric acid employed in lead-acid batteries with a concentration of approximately 29%–32%, are frequent agents involved in corrosive poisoning. Sulphuric acid, also known as battery acid, is a dense, colorless, highly corrosive liquid that causes extensive tissue injury upon contact. Its corrosive nature is amplified by the heat released during exothermic reactions with organic material, leading to coagulative necrosis and secondary thermal burns [2]. Ingestion typically results in widespread gastrointestinal damage, with severity influenced by the concentration, volume ingested, and contact duration.

Although the stomach possesses a naturally acidic environment, large-volume ingestion of sulphuric acid, even as little as 10–15 mL, can overwhelm protective mechanisms, leading to full-thickness gastrointestinal necrosis, particularly affecting the stomach and small bowel [3]. Mortality rates are alarmingly high, with only about 35% of patients surviving such exposures.

In developing countries, like India, poisoning remains a critical public health issue, often driven by the accessibility of toxic household and agricultural substances. Among these, rodenticides represent a significant hazard. Depending on their chemical composition, rodenticides may exert toxicity through cytotoxic, neurotoxic, or coagulopathic pathways. While cases involving individual ingestion of corrosives or rodenticides are commonly reported, simultaneous ingestion of both is extremely rare and portends a high risk of rapidly

Corresponding author: Tauheed Anwar
Email Address: anwertauheed11@gmail.com

progressive systemic and gastrointestinal damage.

This case report describes an unusual and fatal poisoning event following the oral ingestion of battery acid (sulphuric acid). The synergistic toxicity resulted in catastrophic mucosal damage, manifesting as hemorrhagic necrosis of the esophagus, stomach, and small intestine. Through this report emphasize the importance of early recognition and aggressive supportive care in managing such life-threatening toxic exposures.

Case Report

An 18-year-old female was brought to the Department of Forensic Medicine & Toxicology, KGMU, Lucknow, for medicolegal autopsy. As per the inquest report, the case involved suspected ingestion of a corrosive substance. According to the father's account, the deceased was previously healthy with no significant medical history. Patients had mistakenly ingested battery acid that had been stored in a water bottle for domestic cleaning purposes. Immediately after ingestion, patients started complaining intense burning in the throat and epigastrium, followed by repeated episodes of vomiting. The patient succumbed to her injuries while being transported to a healthcare facility. Acid-laden vomitus was accidentally expelled onto the chest, upper limbs, and clothes.

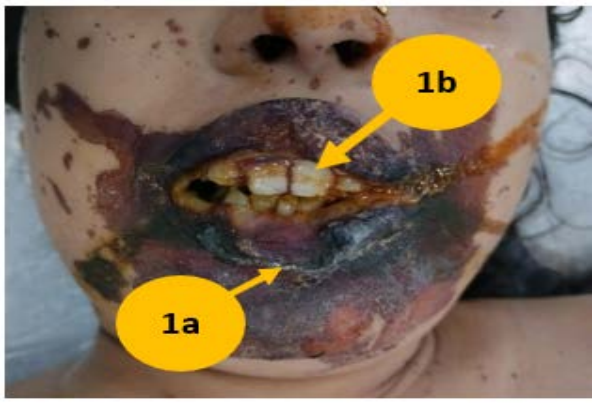

Fig 1a:- Dry, Corroded, necrotic, Brownish black appearance of lips and perioral region

Fig 1b:- Chalky white discoloration of teeth

Fig 2:- superficial burn present on front of chest

External examination revealed charred and excoriated lips and perioral tissues, with brown-black trickle marks extending from the angles of the mouth to the chin as shown in (Fig no. 1a) and anterior chest (Fig no. 2). Acidic vomitus stains were evident on the clothing. Multiple linear streaks of corrosive injury were noted on both hands and feet. Rigor mortis was fully established, and postmortem lividity was present over the back, sparing pressure points. Cyanosis was noted on nail beds. The lips, buccal mucosa, and perioral skin were corroded, appearing dark brown to black. Chalky white discoloration with brownish stains was noted on the dentition (Fig no. 1b). A superficial parchmentized chemical burn measuring 18 cm × 7 cm was observed on the anterior chest (Fig no. 2). Additional superficial burns were present on the dorsum of the right hand. The tongue was corroded and appeared yellowish-brown.

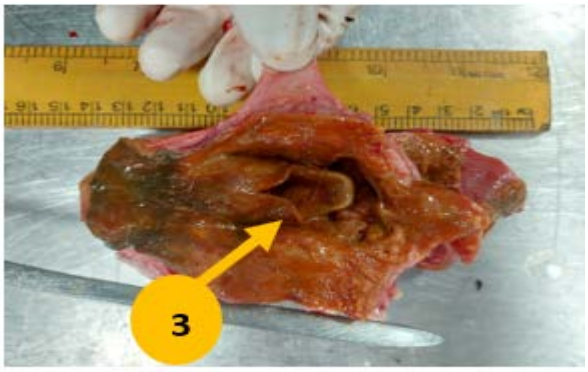

Fig 3:-Yellowish brown discoloration with inflammation and swelling of larynx

Fig 4:- Carbonized and necrotic appearance of gastric mucosa

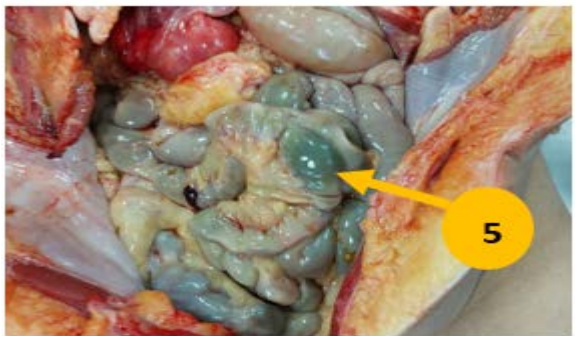

Fig 5:- Necrotic changes seen in intestine

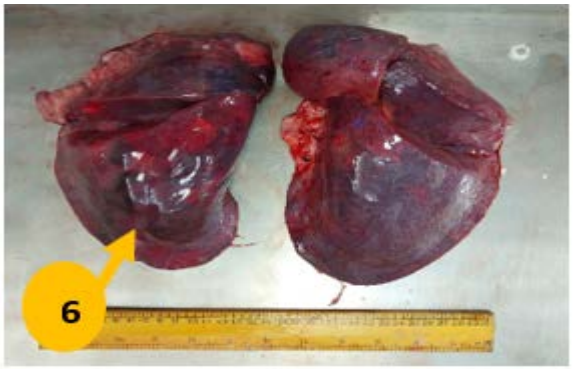

Fig 6:- Multiple hemorrhagic areas on the surface of lungs

On internal examination, the esophageal mucosa was congested and corroded, though without perforation. The larynx wand upper third of the trachea showed corrosive injury and inflammatory edema of the epiglottis (Fig no. 3). The gastric mucosa demonstrated extensive carbonization and necrosis, particularly along the lesser curvature (Fig no. 4). with diffuse interstitial hemorrhages but no evidence of perforation. The stomach wall was soft and friable. The small and large intestines also exhibited patchy necrosis (Fig no. 5). Multiple petechial hemorrhages were noted over the pleural surfaces of both lungs (Fig no. 6). The liver, kidneys, urinary bladder, and uterus were grossly unremarkable.

The final cause of death was established as acute respiratory failure secondary to aspiration pneumonitis following battery acid ingestion.

Discussion

Chemical burns represent a significant form of mechanical trauma, typically resulting from direct contact, inhalation, or ingestion of corrosive agents. Although less common than thermal or electrical injuries, acid ingestion is associated with high morbidity and mortality. According to WHO (2017), burns account for approximately 1,80,000 deaths annually, with acid burns comprising nearly 15% of global burn injuries. In India, the National Crime Records Bureau (NCRB) reported 1,070 acid attacks between 2017 and 2021, while accidental acid ingestion cases continue to rise, averaging 250–300 incidents per year [4].

Workplace exposure, improper storage, and easy accessibility of corrosive substances, particularly in low- and middle-income households, are major contributing factors. Accidental ingestion is often facilitated by the practice of storing acids in unlabelled water or soda bottles, increasing the risk of confusion, especially among children and vulnerable individuals [5].

Acids produce tissue damage primarily via coagulative necrosis, forming a protective eschar that limits deep penetration, whereas alkalis induce liquefactive necrosis and deeper injuries [6]. The extent of injury is determined by several factors including concentration, volume ingested, contact duration, and gastric contents [7]. In the present case, extensive corrosion and necrosis were noted on the lips, perioral region, larynx, and upper trachea, while the esophagus remained unaffected—likely due to the protective nature of its stratified squamous epithelium. No gastric perforation was observed, possibly due to the buffering effect of approximately 80 ml of semi-digested food content in the stomach. Aspiration of acidic material into the lungs resulted in chemical pneumonitis, with laryngeal edema contributing to respiratory arrest.

Zargar et al. (1991) identified esophageal strictures (24.18%), aspiration pneumonitis (11.36%), and respiratory failure (7.69%) as common complications following corrosive ingestion [8]. However, in our case, stricture formation did not occur due to the short survival period. Similarly, Rajan et al. (1985) reported high rates of pneumonitis (84%), renal failure (38%), and hypotension (32%) in formic acid ingestion cases [9].

This rare presentation of sulfuric acid ingestion underscores the need for enhanced preventive strategies, early identification, and targeted toxicological research. It contributes valuable insight into the pathophysiology of dual-agent poisoning and may guide future clinical management and forensic investigations.

Conclusion

This case highlights the fatal consequences of sulfuric acid ingestion though relatively uncommon, results in rapid and extensive tissue damage with multisystem involvement, as demonstrated in the present case. The unusual external and internal autopsy findings highlight the aggressive corrosive nature of the chemicals and the diagnostic challenges it may pose. In view of the increasing incidence of acid ingestion and acid-related violence due to easy accessibility, the Supreme Court of India has directed all states to regulate acid sales through mandatory licensing of retailers and prohibition of over-the-counter sales without proper maintenance of sale registers. Strict enforcement of these regulatory measures, along with improved public awareness regarding the lethal potential of corrosive substances, is essential to prevent such fatalities. This report underscores the urgent need for public awareness, regulatory control on acid availability.

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