Case Report

Successful conservative management of a case of caustic esophagitis based on computed tomography assessment

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Case: We describe the case of a female patient who ingested approximately 100 mL of toilet bowl cleaner containing 9.5% hydrochloric acid in a suicide attempt. Upon admission for hematemesis and epigastric pain, she was alert and oriented with stable vital signs. Initial contrast-enhanced computed tomography (CT) demonstrated edematous changes with no evidence of upper gastrointestinal tract perforation. Endoscopy was not performed owing to the high risk of perforation. We managed this patient conservatively. Repeat contrast-enhanced CT revealed mediastinal emphysema on day 2, which resolved by day 6. The patient was subsequently discharged with no apparent strictures of the upper gastrointestinal tract.

Outcome: Surgical interventions are frequently required following the ingestion of large amounts of highly concentrated hydrochloric acid; however, this patient was successfully managed conservatively.

Conclusion: Contrast-enhanced CT is useful in the assessment of the respiratory and digestive systems and the prediction of potential complications.

Key words: Caustic ingestion, computed tomography, mediastinal emphysema

INTRODUCTION

INGESTION OF ACID or alkaline solutions causes tissue damage, resulting in corrosive esophagitis and gastritis. The severity of the injuries caused by caustic ingestion depends on gastric contents and caustic agent properties, including pH, concentration, ingested volume, and tissue contact duration. In general, tissues are more severely damaged by alkaline solutions than by acidic solutions. This is because alkalis cause liquefactive necrosis, while acids cause coagulative necrosis resulting in eschar formation, thereby protecting the mucosal epithelium from deep ulcer formation.¹ Therefore, severe complications, including esophagobronchial fistula and esophageal perforation, are

rare in cases of acid ingestion. In case of alkali ingestion, the most severely injured body parts are those first contacted by the alkali, commonly the oropharynx, hypopharynx, and esophagus. Unlike alkali ingestion, acid ingestion generally results in gastric injury, more commonly than esophageal injury, particularly affecting the pro-pyloric area.

In several countries, including Japan, household products containing concentrated hydrochloric acid are used for cleaning toilets. These products are often responsible for accidental ingestion by children or suicidal ingestion by adults. Previous reports have demonstrated the potential for the ingestion of toilet bowl cleaner (9.5% hydrochloric acid) to cause esophageal or gastric perforation, which requires emergency surgery and often results in unfavorable outcomes.^{2,3}

We describe the case of a female Japanese patient who ingested toilet bowl cleaner containing 9.5% hydrochloric acid and consequently presented with corrosive esophagitis. Although repeat contrast-enhanced computed tomography (CT) demonstrated mediastinal emphysema, conservative therapy resulted in a favorable outcome in this case.

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Furthermore, we summarize previous reports regarding the relationship between the concentration and volume of ingested hydrochloric acid and the severity and extent of the corrosive esophagitis or gastritis. We further discuss screening by endoscopy and CT imaging, and interventions for caustic ingestion.

CASE

A 77-YEAR-OLD FEMALE INGESTED approximately 100 mL of toilet bowl cleaning solution (containing 9.5% hydrochloric acid; Sunpole, Dainihon Jochugiku, Osaka, Japan) in a suicide attempt. She presented to our emergency department with hematemesis (approximately 200 mL) and epigastric pain. She was alert and fully oriented (Glasgow Coma Scale Score, 15) and her vital signs were stable. She had been diagnosed with cancer of the lower urinary tract and had been receiving a course of chemotherapy. Upon admission, she complained of intense pain affecting her throat and upper abdomen. The pharyngeal mucous membrane was erythematous; however, her abdomen was flat and soft. Bowel sounds were normal. Although marked epigastric tenderness was present, there was no rebound tenderness or guarding. Her complete blood count and biochemistry tests were within normal limits, except for a white blood cell count of 13,900 cells/ μ L and

C-reactive protein level of 4.24 mg/dL. She had normal arterial blood gas values and normal blood coagulation.

A chest X-ray followed by contrast-enhanced CT revealed no esophageal or gastrointestinal perforations. However, the CT demonstrated edematous changes in the pharyngeal, laryngeal, esophageal, and gastric submucosa, which were graded as 1 according to the CT grading system for caustic injury.^{1,4} Milk was then administered through a nasogastric tube to dilute the ingested acid following careful irrigation with physiological saline, resulting in alleviation of her epigastric pain. Antibiotic therapy was initiated at admission and was continued until day 6. She was instructed to inhale adrenalin for bronchodilation. Methylprednisolone was administered for severe laryngeal edema. Gastric mucosal protectants were initiated in combination with a proton pump inhibitor. Initial endoscopy was withheld because there was a high risk of esophageal or gastric perforation. The patient subsequently required a tracheostomy and temporary ventilatory support. Glottis edema was observed on fiberoptic bronchoscopy for nasotracheal intubation at the time of tracheostomy. After confirming favorable oxygen saturations, the patient was weaned off ventilator support.

Repeat contrast-enhanced CT imaging was performed on day 2 and detected subcutaneous and mediastinal emphysema between the nasopharynx and upper mediastinum (CT grade 3, Fig. 1A). CT performed on day 6 demonstrated

Fig. 1. Computed tomography of the chest demonstrating mediastinal emphysema (white arrows in A) on day 2 that resolved by day 6 (B). Endoscopic images on day 19 demonstrating erythema and erosion of the upper gastrointestinal tract (C: esophagus; D: stomach). No esophageal strictures were observed. However, there was a mild deformity of the gastric wall.

satisfactory resolution of both emphysema and previously observed edematous changes (Fig. 1B). Parenteral nutrition was initiated through a central venous catheter and she was converted to enteral feeding on day 13. No evidence of upper gastrointestinal tract strictures was observed using either upper gastrointestinal tract radiography performed on day 15 (images not shown) or endoscopic screening performed on day 19 (Fig. 1C–D). The patient was discharged from the surgical ward on day 21.

DISCUSSION

IN TABLE 1, A SUMMARY of 31 previously reported cases of Sunpole ingestion with ingested amounts documented (1973–2015), mainly based on reports by Ito *et al.*² and Yamamura *et al.*,³ is presented. Out of these, five patients (16.1%) died. Among the fatal cases, four ingested ≥ 300 mL of Sunpole. Although one patient died following ingestion of 100 mL, this patient had a past history of Sunpole ingestion and had undergone resection of the lower esophagus and gastric tube reconstruction. In this fatal case, the authors suggested reingestion of Sunpole may have caused injury of the small intestine leading to peritonitis.

One of six cases with severe complications (including perforation, fistula, and/or hemoperitoneum) survived. Four of 11 cases of ingestion of no more than 100 mL, including our case, had no apparent complications without the need for balloon dilatation or other surgical procedures. However, Okuda *et al.* reported a case of acidic detergent (22% hydrochloride) ingestion with no apparently abnormal physical or laboratory findings at the initial emergency room visit who subsequently presented 10 days later with epigastric pain and hematemesis requiring surgical intervention (gastrectomy and Roux-en-Y anastomosis).⁵ Patients with no apparent symptoms following caustic ingestion should be carefully followed up. In general, particularly in suicide attempts, ingested volumes may not always be determined. However, information regarding the amount of caustic material ingested is useful for prognostication and treatment plan formation. Seven previously reported cases had pyloric stenosis or ulcer without esophageal complications supporting the common conception that acid ingestion tends to cause more serious injury to the stomach than the esophagus, particularly the pro-pyloric area.

In the present case, the patient ingested approximately 100 mL of 9.5% hydrochloric acid. Although repeat CT

Table 1. Summary of 31 cases of toilet bowl cleaner (Sunpole) ingestion in Japan in which the ingested amount was documented (1973–2015)

Ingested volume (mL)	Esophageal complications				
	None	Ulcer or stenosis	Perforation	NA	
≤100	4	6	1	0	
100 – 300	5	7	0	3	
≥300	0	2	3	0	
Ingested volume (mL)	Gastric complications				
	None	Ulcer or stenosis	Necrosis or perforation	NA	
≤100	3	7	1	0	
100 – 300	0	12	1	2	
≥300	0	0	4	1	
Ingested volume (mL)	Requirement of surgery (including balloon dilatation)			Number of deaths (%)a	Number of cases (%)b
	None	Yes	NA		
≤100	4	7	0	1 (9.1)	11 (35.5)
100 – 300	5	7	3	0 (0)	15 (48.4)
≥300	2	2	1	4 (80)	5 (16.1)

This table is predominantly summarized from reports by Ito *et al.*³ and Yamamura *et al.*⁴ The numbers in the table represent the number of cases unless otherwise stated. The numbers in parentheses represent the mortality rate (%) in each category of ingested volume (a), and the percentages relative to total 31 cases (b), respectively. NA, not applicable.

demonstrated mediastinal emphysema (Fig. 1A), emergency surgery was considered unnecessary based on symptomatic evaluation and laboratory and CT findings. In addition, no apparent strictures of the esophagus or stomach developed (Fig. 1C–D), despite the ingestion of a relatively large volume of the caustic agent. The major short-term complications of hydrochloric acid ingestion are reportedly gastrointestinal perforation, airway obstruction, and infection, frequently resulting in fatal outcomes. Acid ingestion, a known cause of profound systemic acidemia in addition to massive hemolysis, correlates with injury severity and adverse outcomes. In particular, ingestion of high concentrations of hydrochloric acid may result in non-anion gap metabolic acidosis.⁶ However, arterial blood gas evaluation demonstrated normal blood pH and no hemolysis in our patient who had ingested a lower concentration of hydrochloric acid (9.5%).

Caustic ingestion frequently causes perforation of the upper gastrointestinal tract, which may occur at any time during the first 2 weeks post-ingestion, resulting in deterioration of general symptoms and the possible requirement of emergency surgical intervention. Currently, the majority of endoscopists recommend early endoscopy to determine the severity and extent of injury and thereby predict potential complications in stable patients without evidence of perforation.^{7,8} Proposed indications for endoscopy following caustic injury are presented in the review article by Ramasamy *et al.*⁷ In the present case, an endoscopist estimated a high risk of GI tract perforation for the following reasons: (i) the patient presented with a relatively large amount of hematemesis; and (ii) follow-up CT on day 2 detected subcutaneous and mediastinal emphysema between the nasopharynx and upper mediastinum. Until relatively recently, caustic esophagogastritis was considered a contraindication to endoscopy due to a perceived high risk of iatrogenic perforation with this procedure. However, the safety of early endoscopy for caustic injury has been confirmed by several studies to date.⁹

Comprehensive guidelines informing the timing and patient selection for endoscopy following ingestion of caustic agents have yet to be established. The timing recommended in the literature remains particularly controversial. Numerous studies recommend evaluation by endoscopy should be performed within 12–24 h of caustic ingestion for early assessment and treatment.⁷ It is believed that, after 48 h, there is progressive weakening of the esophageal wall resulting in increased risk of perforation. Conversely, other studies recommend endoscopy should be performed between 24 h and 48 h as damage will have yet to mature before 12 h and will therefore be underestimated by examination.⁸ Controversy regarding indications for endoscopy following

caustic injury may be a reason for endoscopists hesitating in performing early endoscopy in patients with caustic injuries. Endoscopy is preferably avoided between 5 to 15 days following injury due to the initiation of fibroplasia, followed by granulation tissue formation conferring increased risk of perforation during this period.⁹

The diagnosis on mediastinal emphysema can generally be made earlier and more accurately by CT than by plain chest radiography. In the present case, initial contrast-enhanced CT imaging was used to assess the severity and extent of injury with specific evaluation of esophageal wall thickness and presence of perforations. Follow-up CT on day 2 detected subcutaneous and mediastinal emphysema between the nasopharynx and the upper mediastinum, indicating possible perforation. We decided to manage our patient's condition conservatively based on symptomatic evaluation and laboratory and CT findings. On day 19, no apparent strictures were identified by endoscopy. A CT grading system^{1,4} has been proposed for the prediction of esophageal complications following caustic ingestion. In a retrospective study, Lurie *et al.* reported that CT has lower sensitivity and higher specificity than endoscopy for predicting major outcomes.⁴ Because CT is non-invasive, it has a promising role in the early assessment and careful follow-up of caustic lesions.⁴ CT remains useful, particularly when endoscopy is unavailable.

The management of caustic agent ingestion predominantly comprises close clinical monitoring, symptomatic therapy, and emergency surgery in cases where perforations are detected or suspected. However, the following related conservative therapeutic interventions remain controversial. Neutralization of pH by either weak alkali or weak acid solution is not recommended because exothermic reactions induced by the neutralization reaction may cause thermal injury and exacerbation of tissue injury. Corticosteroids may have utility as prophylaxis against stricture formation in cases of caustic injury. There is, however, no evidence demonstrating administration of steroids results in more favorable outcomes such as a reduction in the rate of esophageal strictures.¹ The induction of vomiting to remove acidic solutions is contraindicated as re-exposure of the esophageal mucosa to the caustic agent may aggravate tissue injuries. Oral administration of milk (or water) in patients with caustic ingestion is controversial. Numerous authors have suggested benefits as long as small amounts are administered, with the aim to dilute the causative solution and irrigate and protect the esophagogastric epithelium. Oral administration of milk is likely beneficial when administered within 30 min of caustic agent ingestion.¹⁰ In contrast, numerous authors are against this approach because: (i) oral administration of a diluent may cause vomiting resulting in

injury exacerbation due to re-exposure of tissues to caustic materials; and (ii) milk administration in particular may interfere with the performance of endoscopy.¹ We carefully performed immediate gastric lavage in the present case. Administration of charcoal is not recommended because caustic materials will not bind to activated charcoal and charcoal may impede visualization during endoscopy.¹⁰ Insertion of a nasogastric tube is recommended under endoscopic guidance. Blind insertion of a nasogastric tube is contraindicated in suspected cases of perforation, particularly in cases of alkaline injury.

Initial endoscopic screening is crucial for determining affected areas and injury extent in cases of caustic ingestion. However, when endoscopic screening is unavailable, contrast-enhanced CT can be used for detecting edematous changes and perforations affecting the respiratory and digestive systems, thereby enabling the prediction of potential complications. Close clinical monitoring and symptomatic therapy is important in such cases and management strategies should be decided on a case-by-case basis.

CONFLICT OF INTEREST

NONE.

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