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Case Report

Gastrointestinal mucosal damages caused by ingestion of corrosive substances: A case study of hydrochloric acid and sodium hydroxide

Fauzi Yusuf[1], Azzaki Abubakar[1], Desi Maghfirah[1*], Mirza Heltomi[2] and Chinwe C. Isitua[3]

[1]Division of Gastroenterohepatology, Department of Internal Medicine, Dr. Zainoel Abidin Hospital, Banda Aceh, Indonesia; [2]Department of Internal Medicine, Faculty of Medicine, Universitas Syiah Kuala, Banda Aceh, Indonesia; [3]Departement of Biology and Forensic Science, Faculty of Science, Admiralty University of Nigeria, Ibusa, Nigeria

*Corresponding author: desimaghfirah@usk.ac.id

Abstract

Ingestion of corrosive substances is most common in children, while in adults causes more severe damages. Massive ingestion of corrosive substances results in severe damage to the gastrointestinal tract and oropharynx if not treated properly. Corrosive substances with pH<2 or >12 can result in severe esophageal damage with either colliquative (alkaline) or coagulative (acidic) necrosis and, at the same time various gastrointestinal injuries could lead to late post-corrosive complications. The aim of the case study was to report the gastrointestinal mucosal damages due to hydrochloric acid (HCl) and sodium hydroxide (NaOH) ingestion. A 55-year-old male patient was presented to the emergency room with a chief complaint of vomiting an hour before admission. Continuous vomiting with a volume of approximately 10–20 cc per vomit. The vomit was initially bluish and turned in to blackish brown over time. Other complaints included nausea, rapid breathing, heartburn, and burning mouth and throat, and had weakness and dizziness. The patient accidentally drank floor cleaning liquid containing HCl. The patient was diagnosed with hematemesis due to ulceration of esophageal, gastric, and duodenal mucosa induced by HCl. Tracheoesophageal fistula developed later in the patient as a long-term complication. Another a 22-year-old male patient was presented to the emergency room with chief complaints of nausea and vomiting an hour before admission. Headache and slight tightness were also experienced. The patient mouth felt burned pain in the solar plexus and frothy saliva. An hour earlier, the patient attempted suicide by drinking two bottles of floor cleaning liquid due to economic problems. The patient was diagnosed with erosive mucosal esophagogastroduodenum induced by NaOH. These cases highlight that intoxication with corrosive substances can complicate damage to the gastrointestinal mucosal and damage features depend on the type of substance concentration and quantity of the corrosive substance.

Keywords: Ingestion, corrosive substance, hydrochloric acid, sodium hydrochloride, mucosal damage

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Introduction

Ingestion of corrosive substances is most common in children, while in adults, causes more severe damage [1,2]. In children aged 2–6 years old, the most common cause is detergent, and in adults aged 30–40 years old, the most common cause is suicide [3]. Ingestion of significant

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amounts of corrosive substances can result in severe fatal damage to the gastrointestinal tract and oropharynx [1,2]. Mild symptoms caused by ingestion of corrosive substances include pain in swallowing [3]. Corrosive chemicals that are often ingested are basic chemicals and acidic chemicals used in everyday life, such as hydrochloric acid (HCl), sodium hydroxide (NaOH), ammonia, potassium hydroxide, and sodium hypochlorite. Other chemicals include glyphosate, paraquat, phenol, potassium permanganate, mercury chloride, and zinc chloride [4]. Acids or bases usually induce limited mucosal damage. Corrosive substances with pH <2 or >12 can result in extensive esophageal damage, either colliquative necrosis (alkaline) or coagulative necrosis (acidic) [5].

HCl is one of the most common chemicals in iron, food, medicine, and the chemical industry [3]. Intentional or accidental ingestion of HCl is known to cause a variety of clinical symptoms, including severe damage to the upper gastrointestinal tract as well as the tracheobronchial system requiring specialized therapeutic regimens [3]. Usually, esophagogastroduodenoscopy (EGD) results show extensive necrosis of the gastric mucosa [3]. NaOH, another corrosive substantive, is an irritant at low concentrations (0.5–2.0%). As the concentration of the solution increases, the chemical causes burn and the severity and depth of the burn is influenced by concentration and duration of contact [6]. It should be considered that exposure of the skin, eyes, or gastrointestinal tract to NaOH at sufficient concentrations (>2.0%) has the potential to cause harm, regardless of the volume of exposure [6].

Most patients with minor injuries due to corrosive substantive recover without serious complications, but most result in serious chemical injuries have high complications and mortality. Acute symptoms of corrosive ingestion include pneumonia, respiratory failure, bleeding, and perforation while chronic symptoms include strictures and fistulas. Death is most often caused by tracheal necrosis and esophageal or gastric perforation, followed by mediastinitis or peritonitis [5]. Herein, we present cases series of HCl and NaOH ingestion resulting in gastrointestinal mucosal damages.

Case

Case 1: Hydrochloric acid

A 55-year-old male patient was presented to the emergency room of Dr. Zainoel Abidin Hospital, Banda Aceh, Indonesia on February 25, 2023 with a chief complaint of vomiting an hour before admission. Continuous vomiting with a volume of approximately 10–20 ml per vomit. The vomit was initially and changed to blackish brown over time. Other complaints included nausea, rapid respiratory rate, heartburn, and burning mouth and throat. The patient also complained of weakness and dizziness. Continuous chest pain, and severe back and abdominal pain were denied. The family said that two hours ago the patient accidentally drank floor cleaning liquid containing HCL (Porstex), but it was not known how much was drunk. According to the family, from one bottle of cleaning liquid containing 700 ml of floor cleaning liquid, 200 ml was left in the bottle and also spilled on the floor. At home, the family had given milk, but the patient continued to vomit and was weak. The patient had a history of high blood pressure since the age of 40 but was uncontrolled. History of other diseases was denied.

Physical examination found the patient's general condition appeared seriously ill, Glasgow coma scale (GCS) score of 15, blood pressure 170/90 mmHg; pulse 72 x/min, respiratory rate 26 x/min, SpO_2 96% at room, temperature 36.4°C, body weight 66 kg, height 165 cm, BMI 24.2 kg/m^2 (normal weight). The head examination indicated normal sclera and conjunctiva. Thorax examination found rhonchi in 2/3 right lung and heart sounds within normal limits. Abdominal examination showed epigastric tenderness. Examination of extremities within normal limits.

Hematology laboratory examination results obtained hemoglobin 16.8 g/dL, hematocrit 47%, erythrocytes 5.4 x 10^6/mm^3, platelets 273 x 10^3/mm^3, leukocytes 23.92 x 10^3/mm^3, MCV 88 fL, MCH 31 pg, MCHC 36%, RDW 15.9%, MPV 9.6 fL, and PDW 10.3 fL. Blood chemistry indicated sodium 146 mmol/L, potassium 4.50 mmol/L, and chloride 121 mmol/L. Renal function with urea 17 mg/dL and creatinine 1.30 mg/dL. PT ratio was 0.96 seconds and APTT ratio 0.89 seconds. AST 35 U/L, ALT 18 U/L, and HBsAg non-reactive. Blood glucose was 153 mg/dL. Thorax X-ray and ECG results are normal limits. EGD of erosive swelling of the mucosal

esophagus, stomach, and duodenum, with debris and ulceration and EGD suggests dilatation results post proximal esophageal stricture, pylorus stricture and duodenal bulb in **Figure 1**.

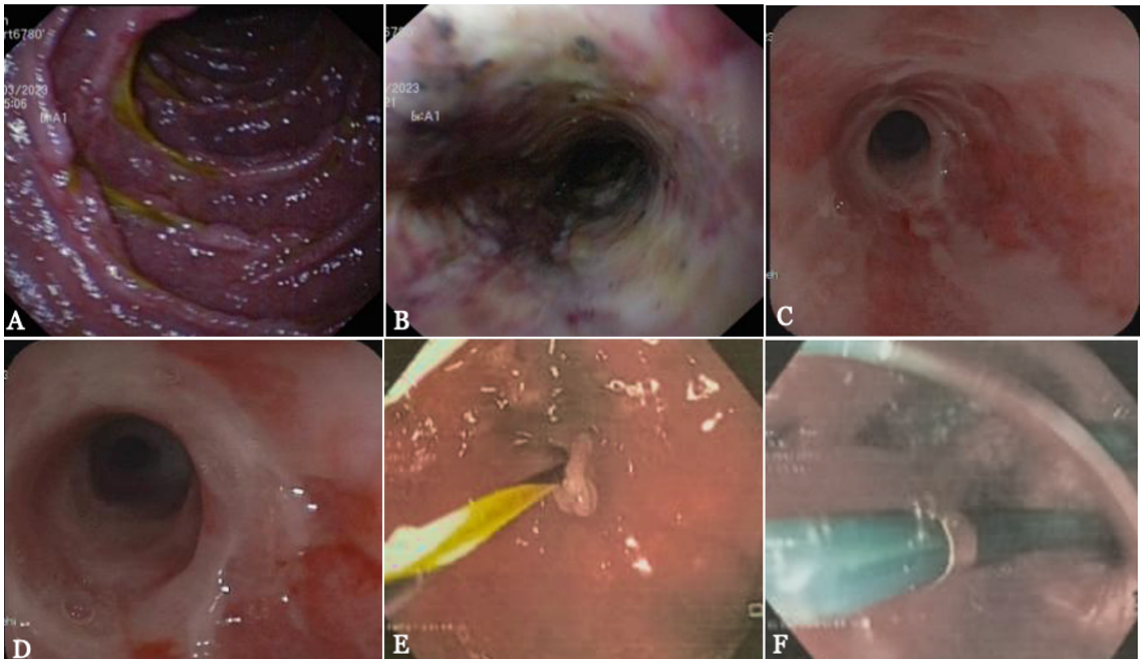

Figure 1. Serial of esophagogastroduodenoscopy (EGD) of the patient. On March 2, 2023, EGD indicatives erosive swelling of the mucosal esophagus, stomach, and duodenum, with debris and ulceration (A and B). On March 20, 2023, EGD indicates a distal esophageal stricture, while the gaster and duodenum could not be assessed because the scope could not enter (C and D). On April 25, 2023, the EGD suggests dilatation results post proximal esophageal stricture, pylorus stricture and duodenal bulb perforation (E and F).

The patient was diagnosed with hematemesis et causa ulceration of esophageal, gastric, and duodenal mucosa induced by HCl, aspiration pneumonia, and stage II hypertension. The management given to the patient was bedrest, nasogastric tube (NGT) insertion, 2.000 kcal/day, O_2 nasal 3L per min, intravenous (IV) Ringer's lactate, lansoprazole 60 mg drip in 50 cc NaCl 0.9% at 5 cc/h, IV ceftriaxone 2 g/12 h, metronidazole 500 mg/8 h drip, IV tranexamic acid 500 mg/8 h, acetylcysteine infusion 3 gr/24 h drip for three days, rebamipide 2x100 mg, amlodipine 1x 10 mg and sucralfate syrup 30 cc after every gastric lavage. The patient condition was stable and able to eat and swallow well and the bleeding in the digestive tract had stopped, so the patient was discharged on March 6, 2023.

On March 15, 2023 the patient was re-admitted with complaints of nausea and vomiting, accompanied by fever, decreased appetite, and difficulty swallowing. The results of the physical examination showed a moderate general condition, blood pressure 140/80 mmHg, pulse 80x/min, respiratory rate 20x/min, temperature 38^{0}C, weight 62 kg, height 165 cm with BMI 22.7 kg/m^2 (normal weight). Laboratory examination results obtained hemoglobin 10.8g/dL, hematocrit 32%, erythrocytes 3.6 106/mm3, platelets 642x10^3/mm^3, leukocytes 12.86x 10^3/mm^3, MCV 87 fL, MCH 30 pg, MCHC 34%, RDW 14.6%, PDW 9.5 fL, MPV 9.4 fL, and ESR 130 mm/h. Blood chemistry examination indicated calcium 8.9 mg/dL, fasting blood glucose 90 mg/dL, ureum 26 mg/dL, creatinine 1.14 mg/dL, sodium 136 mmol/L, potassium 4.40 mmol/L, chloride 100 mmol/L. Esophagography examination result appeared to be tracheoesophageal fistula contains are presented in **Figure 2**. The patient was diagnosed as dysphagia due to esophageal stricture with a history of HCl intoxication, and grade I hypertension. The patient was given bedrest therapy, NGT, sonde diet 2,000 kcal/day via NGT, IV Ringer's lactate, IV lansoprazole 30 mg/24 h, IV ondansetron 4 mg/8 h, paracetamol 3 x 500 mg, curcuma 3 x 20mg, amlodipine 1x10 mg and sucralfate syrup 3 x 30ml. The patient was treated for eight days.

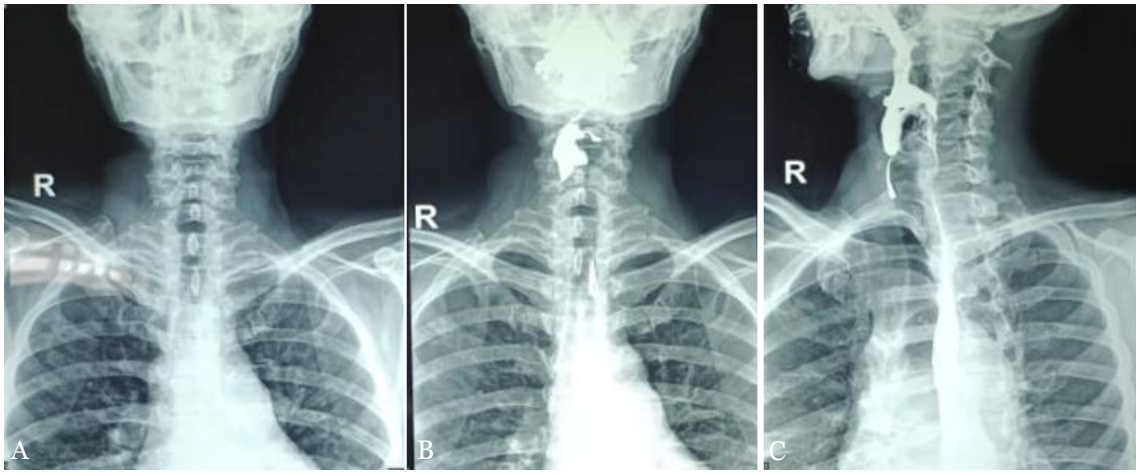

Figure 2. Esophagography examination results found to be good bone, trachea in the middle, and no soft tissue mass swelling. The conclusion appeared to be tracheoesophageal fistula (March 20, 2023). Plain photo before contrast (A); contrast in the proximal 1/3 esophagus (B); and contrast coming out of the esophageal lumen out of a small opening depicting a fistula (C).

The patient came to treatment for the third visit (April 3, 2023) with main complaint of swallowing difficulty. Complaints accompanied by nausea and vomiting every time the patient eats and weight loss of about 20 kg for approximately the last two months. Swallowing pain was denied. The results of the physical examination obtained a moderate general condition, blood pressure 138/64 mmHg, pulse 71x/min, respiratory rate 20x/min, temperature 37^{o}C, body weight 44 kg, height 165 cm and BMI 16.16 kg/m2 (underweight). The patient was diagnosed as distal esophageal stenosis, tracheoesophageal fistula, grade I hypertension, and malnutrition. The patient was given bedrest therapy, NGT, sonde diet 1,000 kcal/day via NGT, IV Clinimix/24 hours, IV ceftriaxone 2gr/24 h, IV omeprazole 40 mg/12 h, IV ondansetron 4 mg/8 h, oral amlodipine 1 x 5 mg, and sucralfate syrup 3 x 30 ml. The patient was treated for 19 days. On April 22nd, the patient was referred to Dr Cipto Mangunkusumo Hospital in Jakarta for further treatment.

Case 2: Sodium hydroxide

A 22-year-old male patient was presented to the emergency room of Dr. Zainoel Abidin Hospital, Banda Aceh, Indonesia with chief complaints of nausea and vomiting an hour before admission. Headache and slight tightness were also experienced. The patient's mouth felt like it was burning, the stomach felt full, pain in the solar plexus, and frothy saliva. Continuous chest pain, and severe back and abdominal pain were denied. An hour earlier, the patient attempted suicide by drinking two bottles of floor cleaner (Wipol) due to economic problem. The patient was immediately taken to the hospital. History of previous suicide attempts was denied.

On physical examination, patient had a GCS score of 15, blood pressure 130/80 mmHg, pulse 100 x/min, respiratory rate 28x/min, SpO_2 97% at room air and temperature 36.4^{o}C. Thorax examination found no rhonchi and heart sounds within normal limits. On abdominal examination, epigastric tenderness was found, but tenderness in all abdominal quadrants was not found. Examination of extremities was within normal limits.

Hematology laboratory examination indicated hemoglobin 15.2 g/dL, hematocrit 42%, erythrocytes 5.2 105/mm3, platelets 237 x 10^3/mm^3, leukocytes 20.57 x 10^3/mm^3, MCV 83 fL, MCH 29 pg, MCHC 35%, RDW 12.3%, MPV 8.3 fL, eosinophil type count 1%, 0%, rod neutrophils 0%, segment neutrophils 85%, lymphocytes 8% and monocytes 6%. PT ratio 1.01 seconds, APTT ratio 0.98 seconds. AST 16 U/L, ALT 11 U/L, albumin 4.g/dL, calcium 9.0 mg/dL, random glucose 131 mg/dL, urea 16 mg/dL, creatinine 0.90 mg/dL, sodium143 mmol/L, potassium 3.90 mmol/L and chloride 113 mmol/L. EGD examination of patient indicated erosive on all mucosal surfaces of the esophagus and stomach (**Figure 3**).

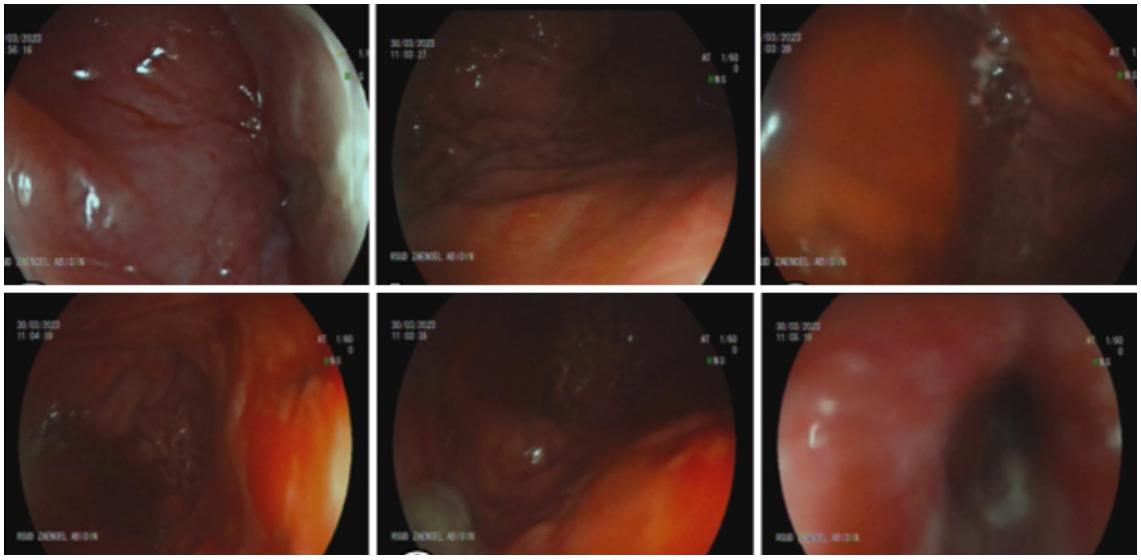

Figure 3. Esophagogastroduodenoscopy (EGD) examination in the patient indicates erosive on all mucosal surfaces of the esophagus and stomach.

The patient was diagnosed with erosive mucosa esophagogastrodudodenum induced by NaOH. Management givens including bedrest, NGT and gastric lavage 200–300 ml NaCl, fasting 6 h, gradual diet starting 50 cc, oxygen 3 L per minute, IV NaCl 0.9%, IV lansoprazole 60 mg and sucralfate syrup 3x30ml. The patient was treated for three days, the patient was discharged with improved condition and a repeat EGD evaluation was not done since the patient lost contact.

Discussion

Poisons are substances that can cause illness, injury, or death. Poisons can be swallowed, inhaled, or absorbed through the skin. Some toxic substances in small doses can have harmful effects, and other substances that are normally harmless can become toxic if exposed to large enough quantities [7].

Poisoning can occur in almost any place, from any substance, and in any form (liquid, solid, or gas) [7]. According to the American Poison Control report, there are about 200,000 cases of corrosive poisoning each year, which most often occurs in acids and bases used as cleaning agents in the home, with 95% of poisoning with suicidal intent. Oral poisoning by corrosives usually occurs by accidentally or intentionally drinking acidic solutions (hydrochloric, acetic, sulphuric, lactic, oxalic, carbolic), basic solutions (sodium and potassium, soaps, detergents), heavy metal salts (sublimate), formalin, feeding solutions, and many other chemicals. The most commonly misused acid solution is HCl (more than 50%) which is easily used as a cleaning agent. In addition to acidic solutions, basic solutions are also misused such as NaOH [8]. The patient in the first case had a history of drinking HCl floor cleaner (Porstex). The patient in the second case had a history of drinking NaOH floor cleaner (Wipol). The contains of both types of floor cleaners are presented in **Table 1**. Porstex contains a strong acid, namely 20% HCl, while Wipol contains a strong base, namely NaOH <1%. So, both floor cleaners contain corrosive ingredients that can cause gastrointestinal damage.

Table 1. Content of Porstex and Wipol

Porstex	Wipol
Hydrochloric acid (HCl) 20%	Soap <6 %
Glycol 20%	Sodium hydroxide (NaOH) < 1 %
Sulfonic ingredients 2%	Pine oil <3 %
Mineral oil 57%	
Additives 1%	

Adopted from [9,10].

Clinical manifestations of corrosive injury to the upper gastrointestinal tract depend on the physical condition, type, and quantity of corrosive substances. Typically burning and pain in the mouth and throat, chest and back abdominal pain, nausea, vomiting, and often blood. These symptoms may occur acutely or several days after exposure. Hypersalivation, difficulty swallowing (dysphagia), odynophagia and edema, ulceration or whitish plaques in the oral cavity, palatal mucosa, and pharynx are common phenomena [8,11,12]. Persistent chest or back pain indicates esophageal perforation and mediastinitis. Severe abdominal pain is associated with gastric perforation and peritonitis. Respiratory manifestations can be observed in the form of dyspnea, cough, hoarseness, stridor, pneumomediastinum and pneumothorax or endotracheal or bronchial necrosis with mediastinitis [12]. The clinical manifestations of both acid and alkaline poisoning in patients are not much different (**Table 2**).

Table 2. Clinical manifestations in patients

Case 1	Case 2
Nausea and vomiting	Nausea vomiting
Hematemesis	Shortness of breath
Shortness of breath	Dizziness
Burning in the mouth	Burning in the mouth
Heartburn	Heartburn
	Bubbly saliva

Alkaline has a pH >7, it is tasteless and odorless, and therefore, the amount ingested is greater. Alkalis cause deeper wounds. Alkalis are also available in solid form NaOH, the amount ingested is usually limited, hence more oropharyngeal and supraglottic injuries. Liquid alkali if ingested in large enough quantities can cause extensive, circular esophageal burns [14]. Acids have a pH <7 and have a pungent odor and noxious taste, which may limit the amount ingested, resulting in acidic solutions causing coagulation necrosis. Acids cause esophageal injury in conjunction with gastric injury. Although alkali ingestion causes liquefaction necrosis and acid ingestion causes coagulative necrosis, ingestion of concentrated acid or alkali can cause similar pathological changes [14].

Esophageal injury begins within minutes of ingestion, early tissue injury is characterized on day two, small blood vessel thrombosis occurs, on days four to seven mucosal sloughing occurs, and bacterial invasion and fibroblastic migration are the main findings. After week two, collagen buildup occurs, and by week three, scar retraction begins leading to stricture formation (occurring over weeks to years) [13,14].

Four factors make a person susceptible to aspiration through acid ingestion: (1) rapid ingestion of large amounts of acidic fluids, which may be the main reason such patients experience more acidemia (lower pH, HCO_3) and higher base deficit); (2) laryngeal dysfunction in the elderly; (3) retention of acid in the mouth before swallowing, which will lead to choking on one further gulp of acid into the trachea and increase the severity of the laryngeal injury and risk of laryngotracheal sequelae; and (4) NGT irrigation-induced vomiting [16].

Patients in HCL poisoning cases were found to have pneumonia thorax photos, with the results of sputum examination found no pathogen development. Aspiration pneumonia in case I patients is due to inhalation of HCL exceeding the exposure threshold which exceeds 2 ppm (Parts per million). While in patients with NaOH intoxication, normal thorax photos were obtained.

Factors that determine the degree of damage are the nature (acid/alkali), physical form (liquid/solid), and amount of corrosive ingested. Solid corrosives adhere to the oropharynx and hypopharynx, producing extensive damage to these areas, whereas liquid materials move rapidly, causing injury to the esophagus and stomach. Damage to the small intestine is rare after ingestion of corrosives as the pyloric reflex will limit caustic entry into the small intestine. However, this protective mechanism is lost in patients who ingest large amounts of corrosive or have a history of previous gastric surgery such as pyloroplasty or gastric bypass, resulting in damage to the small intestine [14]. It has been found that the fatal dose of HCl is 30-40 ml [18].

The annual report of the American Association of Poison Control Centers National on the volume of fatal exposure to corrosive substances when ingested NaOH with a fatal dose in the period 2001-2013. Half a cup of NaOH liquid with a pH of 13, 120-170 ml of bathroom cleaning

liquid containing NaOH, and three sips of cleaning liquid containing NaOH. There are no case studies that mention how much concentration of NaOH in bathroom cleaners is fatal if ingested. However, 100–200 ml of concentrated NaOH (about 20% concentration) can be fatal in some cases of NaOH ingestion [6].

The damage and clinical manifestations in these cases are influenced by the quantity of the corrosive substance taken. In the first case, the patient drank a substance containing HCl with a concentration of 20% but the volume of the substance consumed was unknown, from the clinical manifestations and examination it can be estimated that the amount of liquid consumed exceeds the fatal dose. Whereas in case 2 the patient drank 2 bottle caps of liquid containing NaOH, estimated at 15 ml with a concentration of NaOH < 1%, the amount drunk was still below the fatal dose [6].

For patients who initially have peritoneal signs or extreme abdominal or chest pain, with or without unstable vital signs, hollow organ perforation is highly suspected, therefore CT should be performed immediately [19]. Patients in the first and second cases did not find signs of organ perforation such as extreme abdominal and chest pain, so after initial treatment and improvement of the general condition, an initial EGD examination was performed. The first case with EGD results of grade IIIb mucosal damage and the second case with grade IIa so conservative therapy and monitoring of the patient's condition were carried out.

Early resuscitation aims to minimize damage to the gastrointestinal tract and treat systemic complications that may occur. The basic principles of resuscitation are airway and circulation maintenance. Intravenous proton pump inhibitors are usually given to patients with high-grade injuries to minimize damage to the injured gastric mucosa and avoid exacerbation of esophageal injury by gastroesophageal reflux through which corrosive fluids pass. Administration of broad-spectrum antibiotics is not recommended except in high-grade injuries. Intravenous antibiotics active against oral and intestinal flora should be used as the oropharynx and upper esophagus are home to many virulent bacteria, which can cause systemic sepsis in high-grade injuries [14,20].

Duodenal ulcer treatment, sucralfate is FDA approved for the treatment of duodenal ulcers for up to 8 weeks (short-term). Sucralfate forms a protective film and protects the gastric mucosa from pepsin, pectic acid and bile salts. Sucralfate binds to positively charged proteins in the exudate, locally forming a viscous substance. Sucralfate is a drug used to treat duodenal ulcers, epithelial wounds, chemotherapy-induced mucositis, radiation proctitis, ulcers in Behcet's disease, and burns. Sucralfate exhibits its action by forming a protective layer, increasing bicarbonate production, exhibiting anti-gastric effects, and promoting tissue growth, regeneration, and repair [21]. Both patients underwent initial management with gastric lavage, and were given lansoprazole intravenously and drip and sucralfate syrup.

The clinical outcome of corrosive ingestion depends on the extent and depth of the initial damage. Mild injuries involving only the mucosa usually heal without sequelae, whereas moderate injuries that extend beyond the mucosa result in esophageal strictures. Severe transmural injury manifests as perforation in the acute phase or irreversible stricture in the convalescent phase [14,22]. Tissue injury after corrosive ingestion goes through three phases. The acute necrotic phase (phase 1) characterized by cell necrosis lasts for 24–72 hours. The second phase of mucosal exfoliation with ulceration and fibroblast colonization with granulation lasts for 3-12 days. The stricture formation that occurs in the cicatrix and scarring phase (phase 3) begins about 3 weeks after the initial injury and may continue for 3-6 months or longer. As the esophagus is at its weakest during the ulceration and granulation phase (phase 2), invasive diagnostic procedures, such as endoscopy and therapeutic procedures such as dilatation or stenting should be avoided [14]. In addition to strictures, complications that can occur are gastric obstruction, trachea-esophageal fistula, hiatal hernia, gastroesophageal reflux disease (GERD), carcinoma, and chest complications [14,23].

Conclusion

Intoxication with corrosive substances can result in damage to the gastrointestinal mucosa. Corrosive substances can be either acids or bases. The acidic substances most commonly used in everyday life include hydrochloric acid (HCl) while the basic substances that are often found

include sodium hydroxide (NaOH). These substances can be ingested accidentally or intentionally by people who try to commit suicide. Damage features depend on the type of substance concentration and quantity of the corrosive substance ingested.

Ethics approval

The patients provided written informed consent to be published as case report.

Competing interests

The authors declare that there is no conflict of interest.

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Underlying data

All data underlying the results are available as part of the article and no additional source data are required.

How to cite

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