INVITED ARTICLE

Corrosive Ingestion

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Abstract

Corrosive ingestion remains a common problem in developing countries, such as India due to the lack of strict laws that regulate the sale of caustics. While appropriate treatment of the acute phase can mitigate tissue damage improper management of the acute corrosive injury is widely prevalent due to the limited experience of the individual physicians in managing this condition. The aim of this review is to summarize the epidemiology and pathophysiology of corrosive ingestion, principles in the management of acute phase injury, long-term effects of caustic ingestion, and prevention of corrosive ingestion.

Keywords: Caustic injury, Corrosive, Esophageal stricture, Gastric stricture, Poisoning.

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Corrosive ingestion, although rare in developed countries due to the enforcement of strict regulatory measures, remains a common problem in developing countries, such as India. It can pose a significant management challenge due to a devastating effect on the upper gastrointestinal tract in the acute and chronic phases of the injury. While patients with established strictures are usually treated in tertiary referral centers, emergency management of acute corrosive injury happens in the peripheral centers. Although significant tissue damage occurs immediately after caustic ingestion, appropriate treatment of the acute phase can prevent aggravation of the injuries, and facilitate future management in the chronic phase. However, improper management of the acute corrosive injury is widely prevalent due to the limited experience of the individual physicians in managing this condition. The aim of this review is to summarize the epidemiology and pathophysiology of corrosive ingestion, principles in the management of acute phase injury, long-term effects of caustic ingestion, and prevention of corrosive ingestion.

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Epidemiology

The term "corrosion" derived from the Latin verb corrodere that translates as "to gnaw" underscores how corrosive substances "gnaw" their way through the flesh. Commonly ingested corrosives are broadly classified into acids and alkali. Sodium hydroxide containing bathroom cleaners and dishwashing agents are the often-implicated alkali while toilet cleaning agents containing sulfuric or hydrochloric acid and goldsmith's solvent, which contains hydrochloric and nitric acid in 3:1 proportion are commonly implicated acids.[1–3] The causative agents for corrosive poisoning are not uniform worldwide. According to the 2013 annual report of the American Association of Poison Control Centers, sodium hypochlorite, a natural alkali constituent in household bleach, was the most commonly implicated corrosive agent.[4] Similar reports from the European countries also implicated alkalis as the common cause of corrosive poisoning.[1]

In contrast to western data, in developing countries such as India, where acids are commonly used in the toilet cleaners compared to more expensive caustic soda, acids contribute to most of the corrosive injury.[2,3] Corrosive ingestion could be accidental or suicidal. Accidental corrosive ingestion is frequent in children who unintentionally ingest household cleaning products. A multicenter study of poisoning among Indian children reported caustic ingestion as an important cause of poisoning ranking behind kerosene, drugs, and pesticide poisonings.[3] Adults usually ingest corrosives with suicidal intent, although accidental ingestion has been reported in adults in an inebriated state. Suicidal ingestion tends to be associated with higher grades of tissue injury in the oral cavity, oropharynx, and proximal esophagus as they tend to ingest it with some hesitancy.[2,3] Accidental intake is often associated with the rapid consumption of corrosives in more substantial amounts without knowing that they are corrosives and are frequently associated with gastric injuries.

Pathophysiology

In corrosive ingestion, the factors that determine the extent of the injury are nature (acid/alkali), physical form (liquid/solid) and amount of corrosive ingested. Acids, except for hydrofluoric acid, causes coagulation necrosis with the formation of a coagulum that limits tissue penetration and transmural spread, thereby reducing the incidence of full-thickness injury. However, alkalis by producing liquefaction necrosis increases the likelihood of transmural injuries often accompanied by periesophageal injury with damage to adjacent organs like the respiratory tract.[1–3] The preferential involvement of the stomach in acid ingestion is due to the eschar formation limiting the esophageal damage and reflex pylorospasm increasing the gastric contact time, particularly in the prepyloric zone when the ingested corrosive quantity is minimal.

However, the familiar dictum that "Acid licks the esophagus and bites the stomach" is not always accurate as esophageal injury is frequently reported even after acid ingestion. Solid corrosive agents adhere to the oropharynx and hypopharynx, producing extensive damage to these areas, whereas liquid agents transit rapidly, causing injury to the esophagus and stomach. Airway injury occurs with the concomitant vapor aspiration of ammonia or formaldehyde.[1] Damage to the small bowel is uncommon after corrosive ingestion as reflex pyloric spasm limits the passage of the caustic into the small intestine. However, this protective mechanism is lost in patients with ingestion of a large quantity of corrosive or history of prior gastric surgeries like pyloroplasty or gastric bypass, resulting in damage to the small bowel.[5] While the amount of ingested corrosives influences the extent of damage, reliable information of the ingested quantity is seldom available. Also, it is important to understand that corrosives like phosphoric and hydrofluoric acid can cause systemic effects such as severe hypocalcemia, hypokalemia, and acidosis.[1]

The clinical outcome of corrosive ingestion depends upon the extent and depth of the initial injury. Mild injuries involving only the mucosa usually heal without any sequelae, whereas moderate injuries extending beyond mucosa results in esophageal stricture. Severe transmural injuries manifest as perforation in the acute phase or tight undilatable stricture in the recovery phase. The tissue injury following corrosive ingestion goes through three phases.[6] The acute necrotic phase (phase 1) characterized by cell necrosis lasts for 24–72 hours. The second phase of mucosal sloughing with ulceration and fibroblast colonization with granulation lasts for 3–12 days. The stricture formation that occurs in cicatrization and scarring (phase 3) phase begins approximately 3 weeks after the initial injury and can continue for 3–6 months or more. As the esophagus is at its weakest point during the ulceration and granulation phase (phase 2), invasive diagnostic procedures, such as endoscopy and therapeutic procedures like dilatation or stenting should be avoided.

Clinical Features

Clinical manifestations depend upon the extent of the injury. In the mildest form, the patient may be asymptomatic or presents with mild symptoms like throat pain with normal or mild erythema of the oral cavity mucosa. However, in moderate to severe injuries, the patient presents with significant symptoms. Odynophagia and dysphagia signify extensive esophageal involvement, whereas stridor or hoarseness suggests upper airway involvement.[2] Epigastric pain and hematemesis point to gastric involvement. However, it is crucial to understand that a significant overlap of symptoms can occur, and symptoms cannot reliably predict the site of injury. Respiratory distress with tachycardia and drooling of saliva suggests severe injuries. The presence of abdominal tenderness and peritoneal signs is an ominous sign indicating gastric necrosis. Gastric or esophageal perforation can occur at any time during the first 2 weeks, although the incidence is maximum between days 3 and days 12 following ingestion.[6] Therefore, change in the patient's clinical course in the form of worsening abdominal pain (indicating gastric perforation) or development of chest pain (indicating esophageal perforation) requires immediate radiological evaluation.

Management

Management of acute corrosive ingestion is focused on initial resuscitation, evaluation of the grade of injury, treatment of early complications, maintenance of nutrition, and prevention of stricture formation.

Initial Resuscitation

Initial resuscitation aims to minimize the aggravation of injuries and treat potential systemic complications. The basic principles of resuscitation with the maintenance of airway and circulation should be followed. Tracheostomy with mechanical ventilation might be required in patients with laryngeal injuries. The supine position, gastric lavage, and induced emesis are contraindicated as this result in additional esophageal injury secondary to reexposure of the esophagus to the corrosive agent.[1,2] Neutralization of acids with alkali and *vice versa* is not recommended as heat generated by the exothermic chemical reaction could aggravate tissue damage.[7,8] In addition, dilution of the corrosive with milk or water has a little role, as the extent of the injury is usually determined within minutes after ingestion of corrosive agent. Supportive care, rather than specific antidotes, is the mainstay of acute-phase treatment. Intravenous proton pump inhibitors are usually given in patients with higher-grade injuries to minimize damage to the injured gastric mucosa and avoid the exacerbation of esophageal injury by superimposed gastroesophageal reflux, although their efficacy is not proven. It has been well documented that corticosteroids do not prevent the development of strictures following corrosive ingestion and its use should be reserved for patients with airways involvement.[7–9] Routine use of broad-spectrum antibiotics is not recommended except in patients with high-grade injuries and those receiving systemic steroids. 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.[2,7]

Evaluation of the Grade of Injury

The evaluation conducted simultaneously with the initial resuscitation is aimed at distinguishing patients with high-grade injuries who require emergency surgery from patients with mild injuries who require conservative treatment. Blood biochemical parameters suggestive of transmural necrosis are metabolic acidosis with elevated lactate levels, leukocytosis, thrombocytopenia, high CRP level, and deranged liver function tests.[7,8] Plain X-ray is of value in patients with clinical suspicion of perforation as they may show the presence of free air. Traditionally, early upper gastrointestinal endoscopy performed within 48 hours of ingestion has been recommended as the mainstay investigation to decide further management. The findings on endoscopy are usually graded using the classification proposed by Zargar et al.

- Grade I—only erythema and edema.
- Grade IIa—hemorrhage, erosion, blisters, and ulcers with exudate.
- Grade IIb—circumferential esophageal ulceration.
- Grade IIIa—scattered deep ulcers with brown, black, and gray discoloration.
- Grade IIIb—extensive deep ulcers with brown, black, and gray discoloration.
- Grade IV—an esophageal perforation.

Grades III and IV injuries are classified as high-grade injuries as they often require emergency surgery and invariably result in stricture formation (Fig. 1).[10] The major drawback of endoscopy

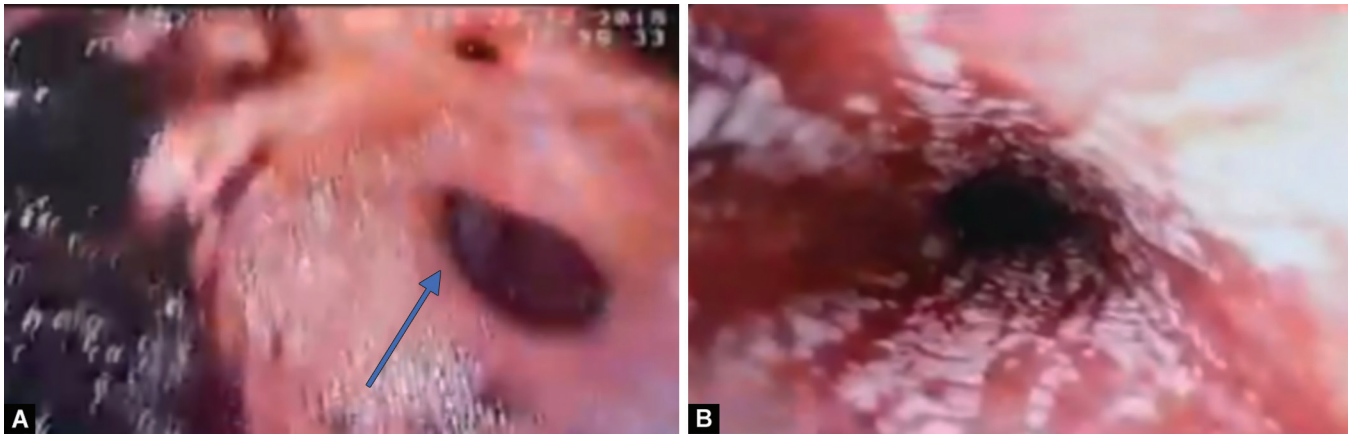
is its inability to accurately determine the depth of necrosis, which could lead to futile surgery or inappropriate nonoperative management, and adversely affecting survival. Also, endoscopic grading is observer-dependent. Due to the increased risk of iatrogenic perforation, upper gastrointestinal endoscopy is better avoided between 5 days and 15 days after ingestion. To overcome the limitations of endoscopy contrast-enhanced computed tomography (CECT) based grading of corrosive injuries has been proposed.[1]

- Grade I—normal appearing organs.
- Grade II—enhancement of internal mucosa with hypodense wall due to edema, with surrounding soft tissue inflammatory change and enhancement of outer esophageal wall (target sign).
- Grade III—the absence of postcontrast wall enhancement.

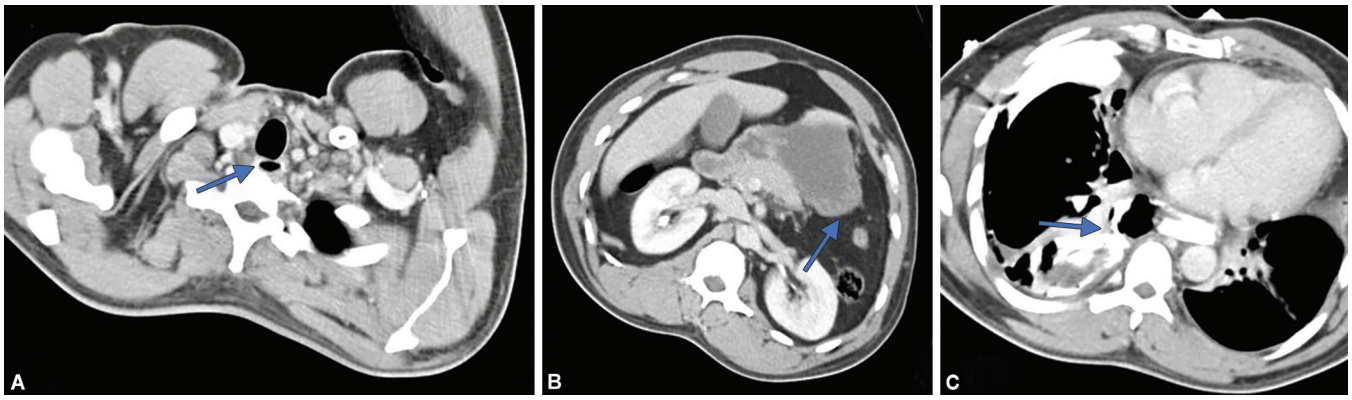
CT grade I corresponds to endoscopic grades I–IIa injuries, grade II with more severe endoscopic grades IIb–IIIb injuries and grade III invariably corresponds to very severe endoscopic grade IIIb injuries (Fig. 2). Recent studies have shown that CECT neck, thorax, and abdomen performed 3–6 hours after corrosive ingestion is superior to endoscopy to detect transmural injuries of the gastrointestinal tract.[7,8] A current emergency treatment algorithm is based on CECT and endoscopic evaluation should be reserved for patients in whom CECT is contraindicated (renal failure and contrast allergy) or interpretation of CT findings is difficult or uncertain (Flowchart 1). Also, in the pediatric population to minimize radiation exposure, endoscopy is preferred over CECT for evaluation of the grade of injury.[1,7,8]

Emergency Surgery

Emergency surgery is indicated in patients in whom initial evaluation suggests transmural necrosis of the gastrointestinal tract. The aim is to remove all the necrotic tissue. Laparotomy is the standard approach, although minimally invasive surgery can be selectively used in high volume centers (Fig. 3). A diagnostic laparoscopy before laparotomy may be selectively employed in patients with suspicious findings. For patients with combined esophageal and gastric injury, esophagogastrectomy with cervical esophagostomy is performed through a combined abdominal and cervical approach with a transhiatal esophageal stripping technique (Fig. 4).[1,2] The thrombosis of perivisceral vessels, corrosive induced periesophageal edema, and the lack of mediastinal adhesions in the acute phase facilitates blunt esophageal stripping. In patients with suspected airway injury and those with significant mediastinal contamination, the transthoracic approach is used. Total gastrectomy is performed in patients with isolated gastric wall necrosis.[11] Although immediate reconstruction with esophagojejunostomy has been reported, external drainage of the distal esophageal stump followed by delayed reconstruction is recommended as most of the patients are in sepsis with hemodynamic instability. A partial gastrectomy is not advocated even in patients with segmental gastric involvement

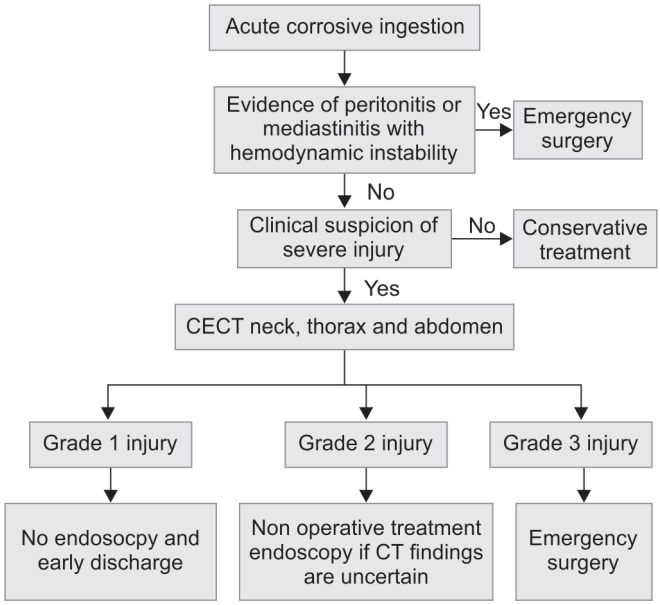


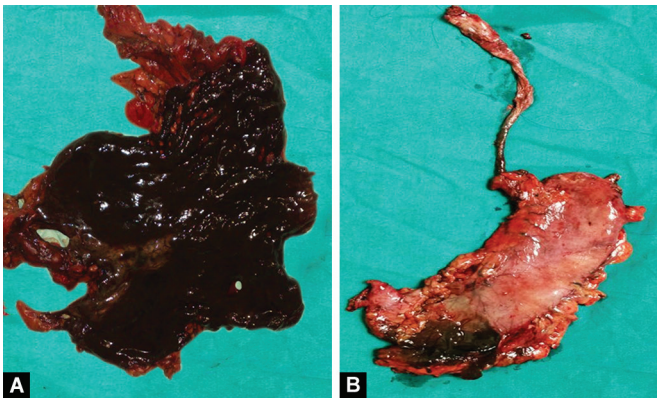
Figs 1A and B: Upper gastrointestinal endoscopy findings in a patient with acute corrosive esophagogastric injury; (A) Grade IIIb injury of the gastric antrum. Pyloric opening marked with arrow; (B) Grade IIa/IIb injury of the esophagus



Figs 2A to C: Contrast-enhanced computed tomography neck, thorax and abdomen of a patient with acute corrosive esophagogastric injury; (A) Pharyngeal mucosa shows normal contrast enhancement (arrow) suggestive of grade I injury); (B) Absent gastric wall enhancement (arrow) suggestive of grade III injury; (C) Esophageal perforation with contrast extravasation (arrow)

Flowchart 1: Emergency treatment algorithm for a patient with acute corrosive injury



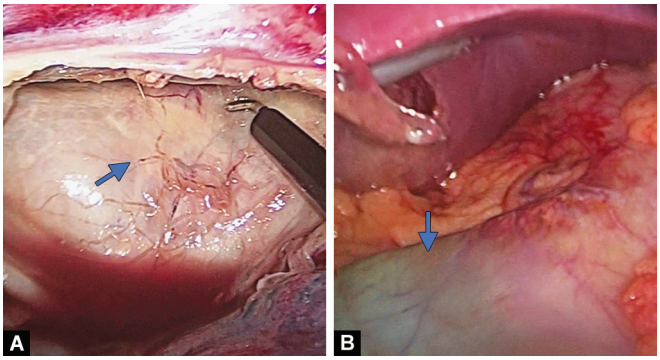


Figs 4A and B: (A) Specimen of total gastrectomy showing transmural necrosis of the gastric wall; (B) Esophagogastrectomy specimen showing esophageal and gastric transmural necrosis

as ongoing necrosis can affect the remnant stomach, resulting in postoperative complications. Resection of other abdominal organs such as colon, spleen, and pancreas may be required in patients with an extension of transmural necrosis to adjacent viscera. However, extensive resections like pancreatoduodenectomy increase postoperative morbidity and mortality.[12] A feeding jejunostomy should be performed in all patients undergoing emergency surgery to facilitate early enteral nutrition and to optimize nutritional status before definitive reconstruction procedure.

Long-term Sequelae of Corrosive Ingestion

Stricture is an important late sequel following corrosive ingestion. It begins within the first 2–3 weeks and may progress rapidly. Endoscopic dilatation using either bougie (like Savary–Gilliard dilators) or balloon dilators is recommended in patients with short dilatable strictures.[2] If endoscopic dilatation is not feasible, then definitive surgical treatment is performed after 6–12 months,



Figs 3A and B: (A) Thoracoscopic esophagectomy performed for a patient with transmural necrosis of the esophageal wall (arrow) with esophageal perforation; (B) Laparoscopic total gastrectomy performed for a patient with transmural necrosis of the gastric wall (arrow) with gastric perforation

depending upon the level of the stricture with a longer delay preferred for pharyngeal stricture. The type of surgery is determined by the extent of gastric and esophageal stricture.[13–17] In patients with isolated long esophageal stricture, midcolon bypass based on the ascending branch of the left colic artery is preferred. The retrosternal route is the preferred route as the native esophagus is generally not resected. Large series have shown that the risk of malignancy in the native esophagus and esophageal mucocele is negligible as the esophageal mucosa is destroyed by the ingested corrosive.[14] Patients with pharyngeal strictures refractory to dilatation usually require a staged procedure in the form of the pectoralis major or sternocleidomastoid myocutaneous flap to create neo cervical esophagus, followed by a colonic bypass.[15,16] Similarly patients with coexistent gastric stricture require a staged procedure. In patients with distal gastric stricture, Billroth I gastrectomy is preferred as gastrojejunostomy can make future colonic bypass difficult. If gastrojejunostomy must be performed retrocolic route should be avoided because it can potentially damage the colonic vascular arcade. In patients with high gastric or total gastric stricture jejunum is used for distal colonic anastomosis instead of the stomach. Total gastrectomy is preferred in preserved patients, whereas in patients with poor general condition stomach can be left *in situ* without major long-term sequences. The feasibility of laparoscopic Billroth I gastrectomy and laparoscopic colon bypass has been reported.[18] However, these procedures, especially colonic bypass is technically challenging and should be restricted to centers with extensive experience in advanced laparoscopic procedures.

Prevention of Stricture Formation

As the management of corrosive esophagogastric stricture is technically challenging various modalities were tried to prevent stricture formation. Early oral feeding in patients with superficial esophageal injury could prevent synechiae and stricture formation. Early nasogastric tube placement is an option as it facilitates enteral feeding and can provide a lumen for dilatation if a tight stricture develops particularly in high esophageal or pharyngoesophageal strictures.[2] However, placement of a nasogastric tube itself can facilitate the development of long strictures. Hence, the decision for nasogastric tube placement must be taken on a case to case basis. Early placement of polyflex stents or biodegradable stents made of poly-L-lactide or polydioxanone has been advocated

to prevent stricture formation.[19] However, it is associated with a limited success rate (<50%) and a high incidence of migration (up to 25%). Considering the cost, limited success rate and association with potential complications like migration, stents are not routinely recommended in the acute management of corrosive injuries. Many pharmacological agents including mitomycin C, 5-fluorouracil, vitamin E, octreotide, and interferon-alfa-2b have been tried in an experimental and clinical setting to prevent stricture formation; however, none of them have been proven to be of significant benefit.[20]

Prevention of Corrosive Ingestion

Immediate and late sequelae of corrosive ingestion requiring numerous diagnostic and interventional procedures put a significant social and economic burden on the family and healthcare system. As it is a preventable menace, active measures should be taken to reduce the incidence of corrosive ingestion and its consequences. The decreasing incidence of corrosive ingestion in developed countries suggests that strict laws and regulatory measures can prevent corrosive ingestion. Stringent legislation is necessary for developing countries to curtail the sale of caustics in unlabeled containers and limit unrestricted access of adults to dangerous corrosive agents. The packing of these agents should be made childproof to prevent accidental ingestion by children. Also, parents need to be educated to keep household corrosives safely away from children. As most of the corrosive ingestion in adults is with suicidal intent, a sound social support system should be developed to assist in relieving some of the stresses that predispose adults to suicides.

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