Essentials of Gastrointestinal and Liver Diseases

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Chapter1: Oesophageal Diseases

Caustic injury of the upper gastrointestinal tract

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Introduction :

Corrosives are a group of substance that have the capacity to cause tissue injury on contact by a chemical reaction. Acid and alkali are the two primary types of agents most often responsible for corrosive injury. Ingestion of corrosive substances is an important public health issue throughout the world especially in developing countries, related to the social, economic, and educational variables and mainly to a lack of prevention. True prevalence of this problem is not known and cannot be extrapolated from the scarce papers or personal experience. In our country, also no definite data is available about the prevalence of corrosive poisoning; but it has been observed that many patients come to emergency department of different medical institution with corrosive poisoning. Most of them are young people with suicidal attempt and few are due to accidental ingestion. We have no guideline for the early and late management of corrosive injury. As a result, complications, morbidity and mortality are high. Moreover, in industrialized and developing countries, the therapeutic approach and management strategies appear to be different, likely because of technology and endoscopic expertise.

Most commonly ingested caustic substances :

Caustic substance	Type	Commercially available form
Acids	Sulfuric	Batteries
		Industrial cleaning agents
		Metal plating
	Oxalic	Paint thinners, strippers
		Metal cleaners
	Hydrochloric	Solvents
		Metal cleaners
		Toilet and drain cleaners eg. Harpic
	Phosphoric	Toilet cleaners
	Nitric acid	Gold workers
Alkali	Sodium hydroxide	Drain cleaners
		Home soap manufacturing
	Potassium hydroxide	Oven cleaners
		Washing powders
	Sodium carbonate	Soap manufacturing
		Fruit drying on farms
Ammonia	Commercial ammonia	Household cleaners
	Ammonium hydroxide	Household cleaners
Detergents, bleach	Sodium hypochlorite	Household bleach, cleaners eg. Clorex solution

Caustic substance Type	Commercially available form
Sodium polyphosphate	Industrial detergents

Factors determining corrosiveness:

- Physical form: solid/ liquid
- Duration of contact with tissue
- Concentration of agent
- Quantity of agent
- pH of agent: pH <2 and >11 are more corrosive
- food: presence or absence of food in stomach
- titratable acid or alkali reserve (TAR): this quantifies the amount of neutralizing substance required to bring the pH of a caustic agent to physiological pH of the tissue.

Patho-physiology:

Traditionally, ingested corrosive substances are either alkalis or acids. Alkaline material accounts for most caustic ingestions in Western countries whereas injuries from acid are more common in some developing countries, like India and also Bangladesh, where hydrochloric acid and sulfuric acid are easily accessible. Harpic is the most common corrosive poisoning in our country.

Acids and alkalis produce different types of tissue damage.

Acid ingestion- causes coagulation necrosis with eschar formation that may limit substance penetration and injury depth.

Alkali ingestion - causes liquefactive necrosis and saponification, and penetrate deeper into tissue. Also causes sub mucosal vascular thrombosis impeding blood flow to already damaged tissue.

Consequences of corrosive injury:

- Necrosis: occurs within minutes of exposure to caustic agent and may persist for hours.
- Ulceration and perforation: four to seven days after ingestion, mucosal sloughing and bacterial invasion occurs. At this time, granulation tissue appears and ulcer becomes covered by fibrin. Perforation occurs during this period, if ulceration exceeds the muscle plane.
- Repair- esophageal repair usually begins on the 10^{th} day after ingestion whereas esophageal ulceration begins to epithelialize approximately 1 month after exposure.
- Fibrosis: occurs within 14-21 days of exposure
- Stricture: scar retraction begins by third week and may continue for several months, resulting in stricture formation and shortening of involved segment of the GIT.
- Gastro-esophageal reflux- develops because of impaired lower esophageal sphincter pressure which in turn accelerates stricture formation.
- Carcinoma formation: occurs after decades of alkali exposure.

Clinical features in corrosive poisoning:

Clinical features depend on the type of the substance, amount, physical form and time of presentation (early or delayed). Crystals or solid particles may adhere to the mucosa of the mouth, making them difficult to swallow and thereby diminishing the injury produced to the esophagus, but potentially increasing the damage to the upper airway and pharynx. Conversely, liquids are easily swallowed and are most likely to damage the esophagus and stomach, the extent of injury correlating directly with mortality and late sequelae.

GIT-

- Severe pain in lips, mouth, throat, chest and abdomen
- Excessive salivation
- Dysphagia and odynophagia – imply esophageal damage.
- Epigastric pain and hematemesis, melaena- common in stomach involvement.
- Signs-symptoms of perforation- that can occur at any time during the first two weeks after ingestion when mucosal sloughing occurs

Respiratory system:

- Cough, dyspnoea, tachypnoea
- Bronchoconstriction
- Pulmonary oedema
- Chemical pneumonitis
- Hoarseness and stridor- suggest laryngeal or epiglottic involvement.

Eyes and skin:

- Pain and burn at the site of exposure
- Erythema and vesicle formation

Late manifestation-

Dysphagia heralds the onset of oesophageal stricture. Early satiety, weight loss and progressive vomiting suggest gastric outlet obstruction. Although symptoms go on progression, they may disappear and recur after 3-8 weeks with the development of obstruction from scar formation.

Mortality:

Mortality is still quite high in developing countries like ours. Many death result from mediastinitis, peritonitis and subsequent multi organ failure especially in third and fourth degree injuries.

Diagnostic studies:

Laboratory tests:

- CBC- WBC count >20,000/ cu mm and elevated serum C-reactive protein are independent predictors of mortality in corrosive poisoning.
- Serum electrolyte- hypocalcaemia can occur in hydrogen fluoride poisoning.
- Blood grouping and cross- matching
- Renal function test
- Liver function test
- Coagulation profile
- Arterial blood gas analysis: an arterial pH less than 7.22 or a base excess lower than -12 are considered indication of severe esophageal injury and of emergency surgey.

Radiology:

Chest X-ray:

The radiographic signs of early mediastinal leaks are usually subtle. However, chest X-ray helps in detection of pneumothorax, pneumomediastinum and pleural effusion. Air under the diaphragm is suggestive of visceral perforation.

Abdominal X-ray: Can help in the detection of pneumoperitoneum.

Contrast studies:

If it is felt necessary to confirm a clinically suspected perforation, a water-soluble agent, such as iodopamide, gastrograffin, and less irritant than barium sulphate, should probably be used, though both can be equally irritant. Conversely, barium sulfate should be the preferred contrast agent in late barium swallowing, providing greater radiographic details than water-soluble contrast agents.

Endoscopic ultrasound-

Evaluation of esophageal wall caustic damage by endoscopic ultrasound (EUS) using a miniprobe seems safe. The destruction of the muscular layers of the esophagus observed at EUS seems a reliable sign of future stricture formation.

Furthermore, ultrasound examination with a radial probe may predict the response to dilatation, which usually requires more sessions when the muscularis propria is involved at EUS.

CT scan

A CT scan likely offers a more detailed evaluation than early endoscopy about the transmural damage of esophageal and gastric walls and the extent of necrosis. It is more valuable than endoscopy in assessing threatened or established stomach perforation and a CT grading system has been proposed to predict esophageal stricture. With the advantage of not being invasive, CT scan has a promising role in the early evaluation of caustic damage.

Computed tomography grading system for caustic lesions:

Grade	Features
1	No definite swelling of esophageal wall
2	Edematous wall thickening without peri esophageal soft tissue involvement
3	Edematous wall thickening with peri esophageal soft tissue infiltration plus well demarcated tissue interface
4	Edematous wall thickening with peri esophageal soft tissue infiltration plus blurring of tissue interface or localized fluid collection around the esophagus or descending aorta

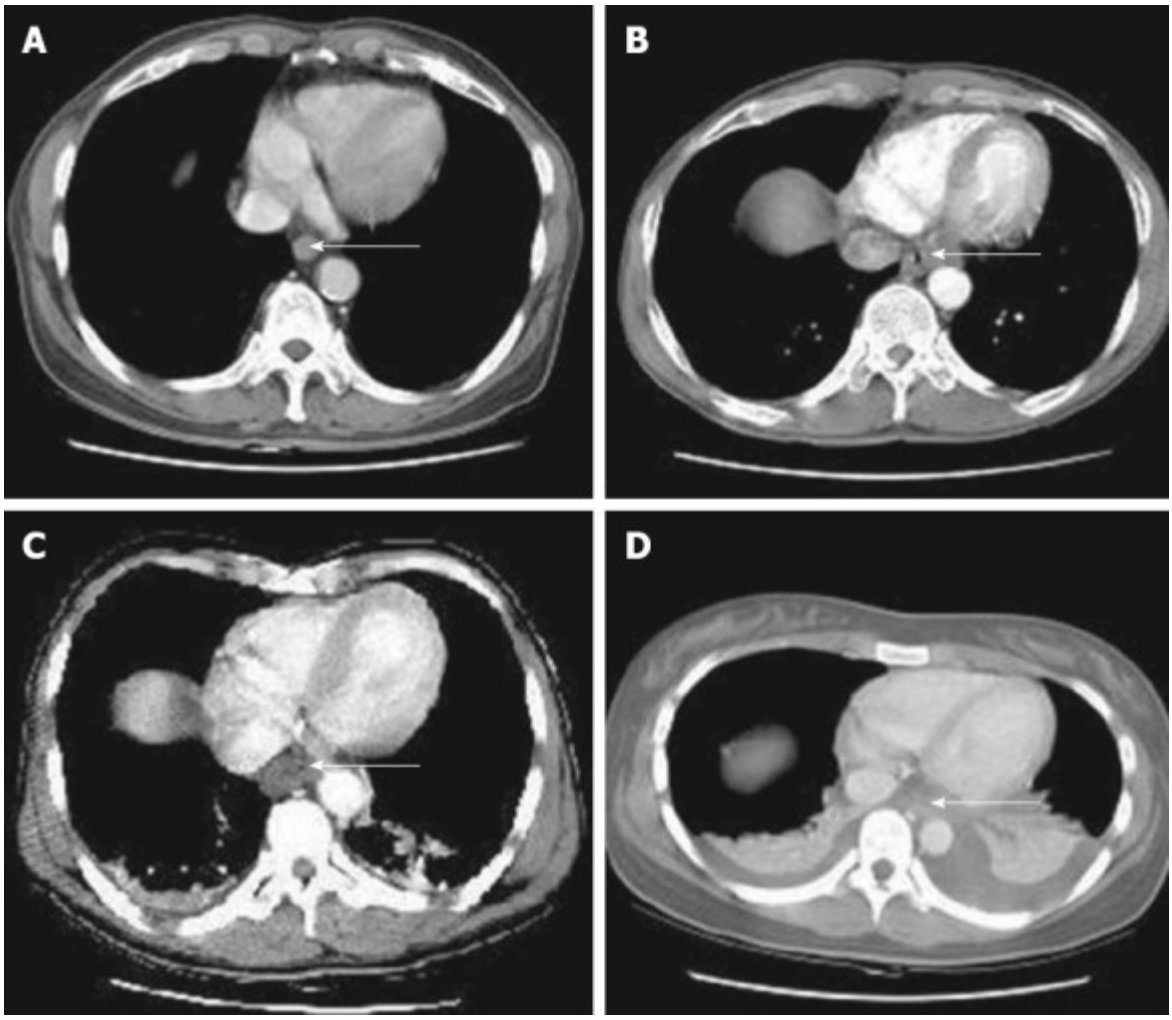

Figure: Computed tomography grading of esophageal caustic injuries. A: Grade 1; B: Grade 2; C: Grade 3; D: Grade 4. Reproduced from Ryu et al. Arrows show the esophageal wall.

Endoscopy:

Direct evaluation by endoscopy is useful in grading severity of tissue injury, planning for nutritional support and long-term management of strictures.[6]

It is considered crucial and usually recommended in the first 12-48 h after caustic ingestion, though it is safe and reliable up to 96 h after the injury; very early (<6 hours) endoscopy may not reveal the full extent of injury. Gentle insufflation and great caution are mandatory during the procedure.

Though endoscopy is considered by most a cornerstone in the diagnosis of corrosive ingestions, which patients would clearly benefit from it is still debated. Significant lesions at endoscopy are not usually observed when symptoms are absent after unintentional ingestion of less aggressive substances, thus making routine post-ingestion endoscopy questionable in this group of patients.

All adult patients must undergo endoscopy after suicidal ingestion, because of the larger amount of more corrosive agents swallowed.

Contra indications to endoscopy:

- Haemodynamic unstability
- Radiologic suspicion of perforation
- Supraglottic or epiglottic burn with oedema
- Third degree burn of the hypopharynx
- Between 5-15 days after corrosive ingestion.

The findings on upper GI endoscopy are based on Zargar's modified endoscopic classification of burns due to corrosive ingestion.[6] They are graded as below:

Endoscopic classification of caustic injuries:

Grade	Features
Grade 0	Normal
Grade 1	Superficial mucosal edema and erythema
Grade 2	Mucosal and submucosal ulcerations
Grade 2A	Superficial ulcerations, erosions, exudates
Grade 2B	Deep discrete or circumferential ulcerations
Grade 3	Transmural ulcerations with necrosis
Grade 3A	Focal necrosis
Grade 3B	Extensive necrosis
Grade 4	Perforations

Endoscopic classification is important for prognosis and management. Generally, grade 0 and 1 lesions do not develop delayed sequels, such as esophageal strictures or gastric outlet obstruction.

The degree of esophageal injury at endoscopy is an accurate predictor of systemic complications and death, with each increased injury grade correlated with a 9-fold increase in morbidity and mortality.

Clinical Approach in Management of Corrosive Poisoning

Goals of therapy are to prevent perforation and to avoid progressive fibrosis and stricture. Approach to the management of corrosive poisoning is based on the clinical features of the patient with caustic ingestion-

1. Asymptomatic patient:
If there is history of minimal corrosive ingestion and no oropharyngeal burns on examination, then the patient requires only observation in the Emergency Room.

2. Symptomatic patient:
If there is history of ingestion of large volume of corrosive along with signs like stridor, hoarseness of voice and respiratory distress, then the patient requires admission in intensive care unit (ICU) and management as detailed below-

Protection of airway: In the presence of respiratory distress and airway edema, urgent endotracheal intubation should be done as airway edema may rapidly progress over minutes to hours. Supraglottic edema leads to acute upper airway obstruction and cricothyrotomy or tracheostomy is needed in such a situation. Delay in prophylactic airway protection may make subsequent attempts at intubation or bag mask ventilation difficult or impossible. There is no clear role for systemic steroids in decreasing airway edema and of intravenous adrenaline or nebulization in reducing the need for endotracheal intubation.

Hemodynamic status: Acute circulatory compromise usually occurs due to hypovolemia. The reasons for hypovolemia are hemorrhage, vomiting and third-space sequestration. Hemodynamic correction can be done by replacement with crystalloid fluids. Invasive hemodynamic monitoring is indicated in unstable patients.

Decontamination: Any attempt at gastric emptying, gastric lavage or dilution of compound is contraindicated in corrosive poisoning. Emetics should not be given as they increase the risk of re-exposure to the corrosive agent followed by mucosal injury and subsequent perforation. Nasogastric tube should not be inserted since it may cause esophageal perforation and increase the risk of aspiration.

Dilution and neutralization: Dilution and neutralization of corrosive by nasogastric tube lavage generates heat and increases the risk of aspiration. Both have no proven benefit and hence are contraindicated.

3. Stabilized patient:
Initial evaluation of a stabilized patient aims to identify the acute complications of corrosive ingestion and stratify the risk for acute and long-term complications mainly by endoscopic grading of corrosive lesions.

Role of corticosteroids: While there is no role of systemic steroids in the management of caustic ingestion, intralesional steroids (triamcinolone injection) can be given to prevent stricture but optimal dose, frequency, and best application technique are still to be defined.

Role of antibiotics: Tissue destruction from caustic injury increases the risk of infection by enteric organisms. Antibiotics are not recommended prophylactically in corrosive poisoning. They are recommended in GI perforation and if lung involvement is identified.

Role of proton pump inhibitors (PPIs) and H2-blockers: Gastroenterologists routinely recommend PPIs and H2-blockers in caustic ingestion.

Nutrition: Endoscopic grade of lesions needs to be assessed for planning nutritional support in patients with caustic ingestion. Patients with Grade 1/2a lesions on endoscopy can tolerate oral feeds, while those with Grade 2b/3a lesions will need nasoenteral feeding. Patients with Grade 3b lesions require gastrostomy for enteral feeding and rarely need total parenteral nutrition (TPN).

Nasogastric tube: Though a nasogastric tube may be helpful to ensure patency of the esophageal lumen, the tube itself can contribute to the development of long strictures and routine use is not uniformly recommended. Any esophageal catheterization may be a nidus for infection and nasogastric placement may worsen gastroesophageal reflux in this patient population, with a consequent delay in mucosal healing. However, positioning a nasogastric tube has the advantage of providing a lumen for dilatation should a tight stricture develops. Therefore, after caustic injuries the placement of a nasogastric tube may be considered, but the decision should be made with caution and done on a case-by-case basis.

Mitomycin C: Mitomycin C, a chemotherapeutic agent with DNA crosslinking activity, when injected or applied topically to the esophageal mucosa, may be valuable in preventing strictures, but this drug has deleterious adverse effects, especially if systemic absorption occurs across the intact mucosa.

Intraluminal stent: Specially designed silicone rubber or, more recently, polyflex stents have been found helpful in preventing stricture formation but the efficacy is less than 50%, with a high migration rate (25%). Patient selection remains a challenge and the development of hyperplastic tissue is a concern.

Other modalities for stricture prevention under evaluation: Intraperitoneal injection of 5-fluorouracil has been effective in preventing strictures experimentally. Anti-oxidant treatment (vitamin E, H_1 blocker, mast cell stabilizer, methylprednisolone) and phosphatidylcoline inhibit collagen production and stricture formation by decreasing tissue hydroxyproline, the ultimate product of collagen degradation, but no human study is available.

Role of endoscopy:

Early Admission
Within 48-72 hours of corrosive ingestion:

- Upper GI endoscopy should be performed on Day 1-2. (ideally between 12-24 hours of ingestion).
- If endoscopy reveals only mild lesions, then the patient can be discharged and clinical follow-up should be done at one month.
- If severe lesions are found on endoscopy, then surgical gastrostomy is indicated, which should be followed by repeat endoscopy and dilatation after three weeks.

Delayed Admission
Within 72 hours to three weeks of corrosive ingestion:

- No endoscopy is indicated.
- Gastrostomy should be done if there is severe dysphagia.
- Endoscopy and dilatation of stricture (if present) should be done three weeks after ingestion.

Late Admission
More than three weeks of ingestion:

- Requires endoscopy and dilatation of stricture.
- If the procedure is successful, then follow-up endoscopy should be done at one month.
- If the procedure is unsuccessful, then surgical gastrostomy is performed, which is followed by
- retrograde dilatation of stricture after 10 days of operation.

Complications

Acute:

- Airway compromise;
- shock (due to hemorrhage, vomiting or third-space sequestration);
- GI perforation (can cause esophageal leak/rupture and
- mediastinitis or gastric leak/bleed leading to peritonitis).

Late:

- Stricture- Following a grade 2B and a grade 3 esophageal burn, stricture incidence may be 71% and 100%, respectively. Strictures usually develop within 8 wk after the ingestion in 80% of patients, but it can happen as early as after 3 wk or as late as after 1 year.

- Gastric outlet obstruction- Gastric outlet obstruction has an incidence of 5% mainly in the prepyloric area, where prolonged contact with the antral mucosa due to pyloric spasms and to resulting pooling of the caustic agent in this region

- late achlorhydria,
- protein-losing gastroenteropathy,

Remote:

- Carcinoma of esophagus.
- Patients who develop esophageal strictures after alkali consumption have high-risk (1,000 times more risk than the general population) for the development of squamous cell carcinoma of esophagus.
- The mean latency period is 40 years after ingestion and in 84% of the patients, the malignancy is located in the area of the bifurcation of trachea.

Management of Complications:

Laparotomy

Laparotomy is indicated in patients with:

- Endoscopic or radiologic evidence of perforation
- Severe abdominal rigidity
- Persistent hypotension
- Respiratory distress
- Ascites or pleural effusion
- pH < 7.2 on arterial blood gas (ABG) analysis

Laparotomy permits tissue visualization, resection and repair of perforation.

Stricture management

Stricture formation begins weeks to months after injury and is the most important consequence of corrosive poisoning. Procedures used for prevention and treatment of strictures are:

1. Dilatation therapy: This is done 3-6 weeks after injury with balloon or bougies (usually Savary); Savary bougies are considered more reliable than balloon dilators in consolidated and fibrotic strictures such as old caustic stenosis or in long, tortuous strictures, and may offer the operator the advantage of feeling the dilatation occurring under his hands. Repeated sessions may be required.

   The interval between dilatations varies from less than 1 to 2-3 wk and usually 3-4 sessions are considered sufficient for durable results.

2. Surgery: Esophageal strictures resistant to dilatation therapy may require surgery that includes resection of stricture surgically and esophageal bypass surgery.

Late surgery:

Surgery for non-responding esophageal stricture-

esophageal replacement by retrosternal stomach or, preferably, right colonic interposition should be considered.

Surgery for stomach injuries-

Elective surgery earlier than 3 mo has been considered risky because of poor nutritional state and the presence of adhesions and the edematous gastric wall.

Pyloroplasty has been recommended for moderate strictures, but progressive fibrosis causing recurrent stricture occurs frequently.

Gastrojejunostomy is a safer alternative to gastric resection in the presence of extensive perigastric adhesion, an unhealthy duodenum, and poor general condition;

Partial gastric resection is preferred by many for the long-term risk of malignant transformation.

Conclusion:

Prevention has a paramount role in reducing the incidence of corrosive ingestion especially in children, yet this goal is far from being reached in developing countries like Bangladesh. Rather, ingestion of corrosive substances is increasingly reported in our country due to lack of prevention. The relationship between symptoms and severity of injury may be vague, and patients should be carefully monitored, since esophageal or gastric perforations can occur at any time during the first 2 wk after ingestion. Endoscopy is considered a cornerstone in the diagnosis of corrosive ingestions, yet the indication for early endoscopy should likely be made on a case-by-case basis. A CT scan may offer a promising role in assessing the evolution of the injury and impending perforations.

Main late sequelae include esophageal strictures, often accompanied by undernourishment. The likelihood of a gastric outlet obstruction should always be kept in mind. The presence of severe gastroesophageal reflux and of esophageal dysmotility may worsen the prognosis. Stricture prevention by stents seems promising but the experience is still limited. Systemic corticosteroids offer no role. Endoscopic dilatation is usually successful in achieving a patent esophageal lumen, but in complex strictures several attempts must be carried out, and in such patients bougies may be preferred to balloon dilatation.Both an improvement in nutritional status and a sustained esophageal patency should be considered reference points for a successful dilatation. Gastrostomy may be lifesaving in this perspective.

Mortality and morbidity of esophageal replacement in patients not responding to dilatation are low in expert hands. Risk of esophageal cancer after caustic ingestion might be overestimated, yet endoscopic screening is still recommended.