Corrosive Poisoning

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ABSTRACT

Corrosive poisoning is a common emergency as corrosive agents are easily available for household use. Emetics and neutralizing agents should be avoided in treatment. Management of corrosive poisoning includes parenteral hydration and nutrition, H₂-receptor antagonists or proton pump inhibitors. Upper gastrointestinal (GI) endoscopy should be done once the patient is hemodynamically stable and there are no signs of perforation. Urgent surgery is required in the event of perforation. Patients with Grade 0-1 injuries do not need hospitalization, while patients with Grade 2 and 3 injuries require intensive care unit (ICU) management.

Keywords: Corrosive agents, proton pump inhibitors, perforation

Corrosives are a group of chemicals that have the capacity to cause tissue injury on contact by a chemical reaction. They most commonly affect the gastrointestinal tract (GIT), respiratory system and eyes. Corrosives and caustics are synonyms, both mean ‘something that eats away’. Acids and alkalis are the two primary types of agents most often responsible for caustic exposures. Exposure to corrosive agents continues to be a leading toxicological source of injury for children and adults. An average home contains a dozen different cleaning products. These account for a large number of accidental and intentional poisonings. The estimated prevalence of corrosive poisoning is 2.5-5% while the morbidity is above 50% and the mortality is 13%. Eighty percent of corrosive poisoning occurs in children below five years. But, adult exposure has more morbidity and mortality due to significant volume of exposure and possible co-ingestion.

COMMON CAUSTIC AGENTS

The common caustic agents include:
- Strong acids and alkalis
- Concentrated weak acids and alkalis
- Oxidizers (with neutral pH)
- Alkylating agents
- Dehydrating agents
- Halogens and organic halides
- Phenol

Acids
- Car battery fluid (sulfuric acid)
- Descalers (hydrochloric acid)
- Metal cleaners (nitric acid)
- Rust removers (hydrogen fluoride)

Alkalis
- Bleach (hypochlorite)
- Sodium hydroxide (liquid lye)

Uses of Common Caustic Agents
- Hydrochloric acid-metal/toilet bowl cleaner
- Sulfuric acid-automobile batteries
- Sodium hydroxide-paint remover/drain cleaner
- Phenol-antiseptic

Factors Determining Corrosiveness

Factors that determine corrosiveness include:
- 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.

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MECHANISM OF ACTION OF CORROSIVE AGENTS

- Alkali ingestion: Causes liquefaction necrosis. This process includes protein dissolution, collagen destruction, fat saponification, cell membrane emulsification, submucosal vascular thrombosis and cell death.\(^3\)
- Acid ingestion: Causes coagulation necrosis. In this process, hydrogen (H\(^+\)) ions desiccate epithelial cells producing an eschar. This process leads to edema, erythema, mucosal sloughing, ulceration and necrosis of tissues.\(^3\)

Both acids and alkalis cause fibrosis and cicatrization (striction formation).

CONSEQUENCES OF CAUSTIC INJURY

Caustic injury may cause the following:\(^4\)

- Necrosis: Occurs within seconds of exposure to caustic agent
- Ulceration and perforation: Occurs within 24-72 hours of exposure
- Fibrosis: Occurs within 14-21 days of exposure
- Stricture: Occurs after weeks to years of exposure
- Carcinoma formation: Occurs after decades of alkali exposure.

CLINICAL PRESENTATION IN CORROSIVE POISONING

**GIT**

- Severe pain of lips, mouth, throat, chest and abdomen
- Excessive salivation
- Dysphagia and odynophagia
- Epigastric pain and hematemesis
- Symptoms and signs of GI perforation

**Respiratory system**

- Cough
- Dyspnea
- Bronchoconstriction
- Pulmonary edema
- Chemical pneumonitis

**Eyes and skin**

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

INVESTIGATIONS

**Laboratory Tests**

- Hemogram: WBC count >20,000/mm\(^3\) is an independent predictor of mortality in corrosive poisoning.
- Serum electrolytes: Hypocalcemia can occur with hydrogen fluoride poisoning.
- Blood grouping and cross-matching
- Renal function tests
- Liver function tests
- Coagulation profile
- Arterial blood gas analysis: Arterial blood pH and base deficit correlate with severity and adverse outcomes.

**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. A lateral view is more sensitive than PA view for detecting intraperitoneal air.\(^5\)
- Abdominal X-ray: Can help in the detection of pneumoperitoneum.
- Contrast studies: Barium studies have low sensitivity in detecting perforation and high-risk of aspiration and inflammation.
- CT scan: CT scan of neck/chest/abdomen should be considered if there is a high-risk of suspicion for perforation despite negative plain X-rays. Contrast-enhanced CT (CECT) is used to assess esophageal wall thickness, which can be used to predict the response to dilatation of stricture and the number of sessions required to achieve adequate dilatation. CT studies done with water-soluble contrast will allow localization of leak of air.

**Endoscopy**

Endoscopy has been called 'sine qua non' for evaluating patients with corrosive poisoning. Direct evaluation by endoscopy is useful in grading severity of tissue injury, planning for nutritional support and long-term management of strictures.\(^6\)

**Indications for upper GI endoscopy**

- Corrosive ingestion by small children
- Symptomatic older children and adults
- Patients with altered mental status
Patients with intentional ingestion
- Patients with ingestion of large volumes
- Patients with ingestion of concentrated products.

**Contraindications for upper GI endoscopy**
- Hemodynamic compromise
- Peritonitis and mediastinitis
- Mild ingestion (asymptomatic patients with normal oral/upper airway examination).

Endoscopy done very early (<6 hours) may not reveal the full extent of injury. The commonest practice is to perform endoscopy on Day 1-2.

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

<table>
<thead>
<tr>
<th>Grade</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>Normal mucosa</td>
</tr>
<tr>
<td>1</td>
<td>Erythema/Hyperemia</td>
</tr>
<tr>
<td>2a</td>
<td>Superficial ulcer/erosion/friability/hemorrhage/exudates</td>
</tr>
<tr>
<td>2b</td>
<td>Findings in 2a + deep discrete/circumferential ulcers</td>
</tr>
<tr>
<td>3a</td>
<td>Scattered necrosis (black/grey discoloration)</td>
</tr>
<tr>
<td>3b</td>
<td>Extensive/circumferential necrosis of mucosa</td>
</tr>
</tbody>
</table>

**MANAGEMENT**

Management is based on the presenting clinical features on admission to the hospital. This can be divided into emergency management, management of stable patient and long-term management.

**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.

**CLINICAL APPROACH IN MANAGEMENT OF CORROSIVE POISONING**

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 or dilution of compound is contraindicated in corrosive poisoning. Emetics should not be given as they increase the risk of mucosal injury and subsequent perforation. Nasogastric tube should not be inserted since it may cause esophageal perforation and increase the risk of aspiration. Exceptions to general rules of decontamination are zinc chloride and mercury chloride poisoning because both cause systemic toxicity.
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.

Corticosteroids: While there is no role of systemic steroids in the management of caustic ingestion, intralesional steroids can be given.

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.

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).

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; obstruction

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:

- Dilatation therapy: This is done 3-6 weeks after injury, progressively larger bougies are passed over endoscopically placed guide wires for dilatation. But, the risk of perforation, aspiration and dysphagia is high.
- Surgery: Esophageal strictures resistant to dilatation therapy may require surgery that includes resection of stricture surgically and esophageal bypass surgery.

REFERENCES

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