Caustic Ingestions in Pediatric Patients

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Abstract

Caustic ingestions continue to cause significant morbidity in children. The extent of mucosal injury from caustic ingestion depends on the concentration and pH of the substance in addition to the viscosity, location of contact, and contact time. Esophageal stricture is one of the most common sequelae of caustic injury. Review of proper household storage should be considered at well child visits as a measure of caustic ingestions prevention.

Keywords: Caustic; Esophagus; Pediatrics

Introduction

Caustic agents are a group of chemicals that have the ability to cause an injury of the tissues they contact. It means 'something that erodes'. The caustic agents' sound effects can vary from no apparent injury to hazardous outcomes due to respiratory and gastrointestinal burns, lifelong complications and potentially fatal sequelae at the extreme scale. Severe complications, such as oesophageal perforations as well as strictures requiring multiple dilatations or oesophageal replacement, have been described [1]. Acids and alkalis are the two principal types of caustic agents.

Exposure to caustic agents continues to be a leading toxicological source of injury for children despite continuous educational programs [2], this could be because the average home contains many cleaning products such as dishwashing liquids, window cleaning agents, and drain cleaners. These count for a large number of accidental and intentional poisonings.

In this article, we aim to provide an overview of accidental ingestion of caustic substances in pediatrics age group.

Epidemiology

Incidence varies with nation and culture. The 2015 annual report of the American Association of Poison Control Centres (AAPCC) which documented more than 1 million substance exposures in children <6 years. Roughly 25% of those exposures were to cosmetics/personal care products and household cleaning substances [3]. Evidence also suggests that nearly eighty percent of caustic ingestions occur in children less than 5 years [4].

Ingestion of caustic chemicals remains an important public health problem in many countries and it’s still a significant issue in developing countries, where it is still a major cause of morbidity and mortality in children [5,6]. For example, In some parts of Palestinian authorities and according to unpublished work by Sultan M et al., it was found that caustic agents constitute the second most common accidentally ingested substance (27.2%) among children <14 years (n=202). On the other hand, it has been declining in developed countries through education and safety measures such as warning labels and child safety caps. The Poison Prevention Packaging Act of 1970 stated that caustic agents should have a “special packaging” which is designed to be difficult for the children who are younger than 5 years of age to open, this was followed by a decline in accidental exposures and esophageal injuries [7].

Pathophysiology

The extent of mucosal injury from caustic ingestion depends on the agent's pH, concentration, tissue contact time, location of contact, amount ingested, viscosity, and the ingested form of the agent (liquid, gel, or solid) [8].

Caustic ingestions are mostly caused by alkali, which are typically colourless, odorless liquids posing an increased risk of a high volume, and they bind with tissue proteins producing liquefactive necrosis and saponification, allowing deep penetration to submucoosa and muscularis, resulting in scarring of tissue, and thrombosis of vessels and impeding blood flow to already damaged tissue, and to the extreme extent leading to perforation [9]. Free radical damage with consequent lipid peroxidation may also contribute to esophageal damage. Alkali often cause damage to the mouth, pharynx, esophagus, and stomach, along with the trachea if aspirated. Unless they are of low concentration, most alkali are considered highly caustic agents if they are of a pH >12.

On the other hand, acids constituted less than 5% of all toxic ingestions in the 2014 AAPCC annual report. Acidic liquids tend to have a bitter taste, decreasing their volume of ingestion with either accidental or intentional ingestions. Strong acids with pH <2 produce tissue injury because of coagulation necrosis as the result of ischemia [10]. The depth of injury is minimized by the fact that acids characterized by eschar formation, which limits further acid penetration [11]. Gastric injury is more common than esophageal injury due to the low viscosity and specific gravity of acids that result in rapid transit to the stomach [12]. Common household acids include hydrochloric acid (toilet bowl cleaner), sulfuric acid (stain removers, car battery), and phosphoric acid (hair dye).

Clinical Presentation

The clinical manifestations of caustic ingestion do not predict presence or severity of esophageal lesions in children, so that the relationship between symptoms and severity of injury is uncertain. For example, in
one study, it was found that 82% of patients with symptoms had grade 0 or 1 esophageal injury on esophagoscopy versus 12% of asymptomatic patients who had a grade 2 lesion [13].

In children with symptoms, the most common presenting symptoms are drooling, vomiting, refusal of intake by mouth, and abdominal pain. The patient may also experience an oropharyngeal burn. Oral burns include lip or tongue erythema and edema, leukoplakia, or ulceration. Rarely do patients present with respiratory symptoms, such as hoarseness and stridor which suggest upper airway and or epiglottic involvement; when these symptoms are present, they are associated with more significant injury.

The presence or absence of oral lesions is a poor indicator of esophageal injury; Dogan et al. [9] illustrated that in a 2006 review of 473 pediatric patients admitted to the hospital with suspected significant caustic ingestion, oropharyngeal burns were not seen in 61% of patients with esophageal lesions.

Management

a) General measures

The cornerstone for the management of all caustic ingestions is airway and hemodynamic stabilization, and the patency of the patient’s airway must be addressed first [14]. Fiberoptic laryngoscopy can be useful in this regard. If the airway is unstable, intubation under direct visualization is required [14,15]. A surgical airway may be required in case it’s difficult to secure the airway though intubation [16]. After the airway is secured, a thorough physical examination should be completed and a thorough history taken with specific attention to the timing of the ingestion, identity of the agent, and amount ingested [14]. Chest and abdominal radiographs should be obtained to detect pneumomediastinum (esophageal perforation) or air under the diaphragm (gastric perforation) [17].

Nevertheless, for patients with a clear history of accidental ingestion of a low-volume, low-concentration caustic substance and with no signs and symptoms of oropharyngeal injury, endoscopy may be deferred. These patients may then be discharged after a 48-h observation period [18]. For any patient who is symptomatic, has oropharyngeal burns, or has significant history of ingestion, an upper endoscopy is recommended (Figure 1).

Induction of emesis or neutralizing (weakly acidic or basic substances) after a caustic ingestion is strongly contraindicated because this may further expose the esophagus to the offending agent and exaggerate the extent of thermal injury and chemical destruction of esophageal tissue, respectively.

b) Endoscopy

Esophagogastroduodenoscopy is an important and highly recommended diagnostic tool in the evaluation of caustic injury especially during the first 12 to 48 hours of caustic ingestion, though several reports indicate that it can be safely performed up to 96 hours after ingestion. Timing of endoscopy is important; an endoscopy done too early may not be able to show the extent of the burns and an endoscopy done after 48 hours increases the risk of perforation if necrosis has occurred.

Endoscopy is important not only in the diagnosis of caustic ingestion but also in determining subsequent management. According to Zargar grading (Table 1), Hao-Tsai Chenget al. [19] suggests that patients with mucosal damage exceeding grade 2a are at a higher risk of developing serious complications, while patients with mild mucosal damage have a significantly reduced mortality and morbidity. In the patients with grade 2b and 3a injuries, ICU observation and nutritional support may be mandatory if there are any signs of bleeding and the patient experiences abdominal pain, and antibiotics are cautiously recommended in those with lung involvement. Patients with grade 3a lesions may not require immediate surgery [15,20,21].

Due to the risk of high volume of intake and increased rate of gastric injury, intentional ingestions should be evaluated by endoscopy which may spare the oropharynx from injury, or in the case of acid ingestions [22].

c) Nasogastric tubes

Nasogastric tubes may be used as a stent in case of circumferential burns when there may be increased risk of stricture. However, care should be taken and nasogastric tube should not be inserted without endoscopic guidance because manipulation of the necrotic esophagus might lead to perforation.

d) Medications

To minimize the reflux of gastric contents into the esophagus, thereby minimizing esophageal injury, initiation of proton pump inhibitors and H2 blockers is recommended after caustic ingestion [23].

Data in the literature concerning the use of antibiotics are scarce and the role of antibiotics in stricture prevention is less clear. In theory; antibiotics reduce the bacterial count in the wound, thereby decreasing inflammation and reducing scar tissue formation. Currently there is no sufficient human data to support the global use of antibiotics in patients with caustic ingestion.

### Table 1: Endoscopic grade of esophageal caustic injury.

<table>
<thead>
<tr>
<th>Grade</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>No identifiable injury</td>
</tr>
<tr>
<td>1</td>
<td>Erythema and edema of mucosa</td>
</tr>
<tr>
<td>2a</td>
<td>Noncircumferential and superficial ulceration with white plaques or haemorrhage</td>
</tr>
<tr>
<td>2b</td>
<td>Circumferential injury or deep ulcerations with features of 2a</td>
</tr>
<tr>
<td>3a</td>
<td>Small or patchy necrosis</td>
</tr>
<tr>
<td>3b</td>
<td>Extensive or circumferential necrosis</td>
</tr>
<tr>
<td>4</td>
<td>Perforation before or during endoscopy</td>
</tr>
</tbody>
</table>

Citation: Ayesh K, Sultan MI (2017) Caustic Ingestions in Pediatric Patients. J Gastric Disord Ther 3(2): doi http://dx.doi.org/10.16966/2381-8689.133
Corticosteroids have been proposed as a treatment to reduce stricture formation after caustic ingestion. The rationale for corticosteroid use in this setting is due to their ability to attenuate inflammation, granulation, and fibrous tissue formation. However, studies comparing the benefits after corticosteroid administration are of conflicting results. Hence based on the current evidence it seems prudent to avoid systemic corticosteroids in caustic ingestion until further research confirms its efficacy.

Intralesional steroid injection such as triamcinolone (40-100mg/session) has long been known to augment the dilation of caustic-induced esophageal strictures, however its use is still controversial [24]. Recently, mitomycin-C has been used as an adjunct after dilation of caustic strictures in humans (including those with long strictures) due to its antifibroblastic properties by applying mitomycin-C topically at a dose of 0.4 mg/mL [25].

c) Late complications and management

Esophageal stricture is one of the most common sequelae of caustic injury. Up to 70% of patients with grade 2B and more than 90% of patients with grade 3 injury are likely to develop esophageal stricturing [26]. The timing of management is crucial in achieving long term functional effects.

d) Endoscopic dilatation

First-line non-surgical treatment for strictures is dilation. Balloon or mechanical (bougienage) dilators can both carry out dilation. The most common type of mechanical dilators are Savary-Gilliard dilators, which are passed over a guide wire and deliver both radial and longitudinal force to the stricture.

The method of choice depends on operator experience and comfort with the equipment because there is no clear difference in effectiveness and safety between balloon and mechanical dilation [27].

However, using balloon dilators, a lower dilation force should be used initially to avoid perforation [28]. This may need to be repeated and advanced slowly to achieve effective and safe dilation. The interval between dilatations varies from 1-3 weeks among different studies [29].

e) Esophageal stents

Though endoscopic dilation with balloon has been the standard of treatment for benign esophageal strictures, the recurrence rate still reaches 30%-40%. Approximately 10% of these patients fail to achieve clinical improvement and remain refractory to repeated dilatations. In such patients a good option is stent insertion [18].

The use of self-expanding plastic stents (SEPS) and fully covered self-expanding metal stents (FCSEMS) has been reported to provide an alternative or adjunctive means of preventing stricture formation by providing continuous dilation of the esophagus for prolonged periods. Broto et al. [30] showed a 50% success rate with SEPS and Zhang et al. [31] reported a 75% success rate using FCSEMS. Both studies are limited by their small sample size and retrospective design.

f) Surgery

Surgery may be necessary in cases where dilation fails to produce adequate lumen size to permit only minimal dysphagia symptoms. Surgical procedures performed include partial esophagectomy, local resection of the stricture, and esophageal replacement. Types of esophageal replacement include gastric pull-up, gastric tube, colon interposition, and jejunal interposition. The most common operations are gastric pull-up and colon interposition [32].

Other late complications are dysmotility of the esophagus and stomach, which occurs when the lower third of the esophagus is involved in the burn. Depending on the depth of the scar, the myenteric plexus may be damaged and the normal syncytium of smooth muscle cells may be interrupted [33].

There’s also an increased rate of esophageal cancer, both adenocarcinoma and squamous cell carcinoma which have been reported at a rate of 1000 to 3000 times higher than the normal population [34,35].

Gastric with subsequent gastric outlet obstruction can occur after both alkali and acid ingestion [33]. Early surgery has been reported to decrease mortality and morbidity [34]. Endoscopic gastric dilatation has been considered an alternative to surgery, but dilations have a less than 50% success rate in preventing surgery [35]. In the setting of severe gastric adhesions and significant duodenal injury, gastrojejunostomy should be considered as an alternative to gastric resection [36].

i) Prevention

In some places, where prevention campaigns are not as widespread, children continue to have high rates of caustic ingestion and may come to medical attention later with more severe injuries. Accordingly, the role of prevention has to be executed when there are many opportunities to prevent caustic ingestions.

Taking two steps to prevention is a way to minimize the prevalence of accidental ingestion; identifying at-risk patients (young children and suicidal patients) and ensuring that patients and parents know the proper storage of household agents - keeping substances in their original container.

Conclusions

Accidental caustic ingestion in the pediatric age group lead to a high number of emergency department visits, and it continues to be a major concern for pediatric emergency department clinicians. Increased prevention of ingestion through community awareness and watchful childcare are needed. There is a pressing need for non-invasive diagnostic modalities and effective therapeutic options to evaluate and treat the complications associated with caustic ingestion.

References


