SciForschen
Open HUB for Scientific Research

ISSN 2381-8689

Journal of Gastric Disorders and Therapy

Review Article | Volume: 3.2 | Open Access

Caustic Ingestions in Pediatric Patients

Ayesh K[1] and Sultan MI[2]*

[1]*Al-Makassed Hospital, East Jerusalem, Palestine, Israel*
[2]*Department of Pediatrics, Al-Quds University, Mount of Olives, Palestine, Israel*

***Corresponding author:** Sultan MI, Department of Pediatrics, Al-Quds University, Mount of Olives, Palestine, Israel, **E-mail:** drmutazsultan@gmail.com

Received date: 17 Aug 2017; **Accepted date:** 16 Oct 2017; **Published date:** 20 Oct 2017.

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

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 submucosa 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).

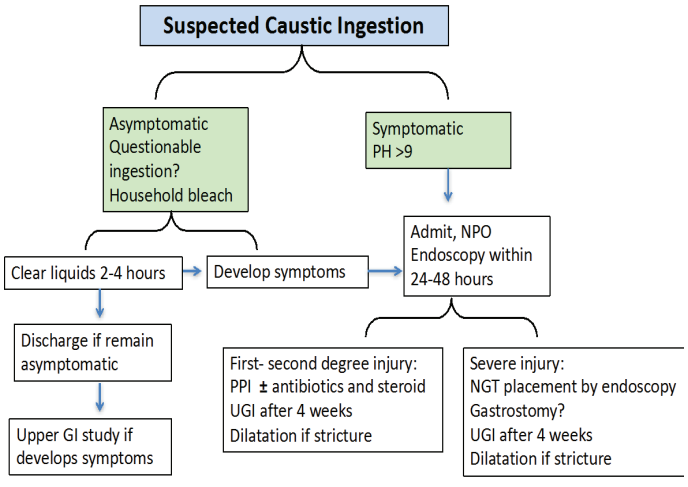

Figure 1: Management of suspected caustic ingestion.

Induction of emesis or neutralizing (weakly acidic or basic substances) after a caustic ingestion is strongly contraindicated because this may further expose the oesophagus to the offending agent and exaggerate the extent of thermal injury and chemical destruction of oesophageal 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 oesophagus might lead to perforation.

d) **Medications**

To minimize the reflux of gastric contents into the oesophagus, thereby minimizing oesophageal 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 scares 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.

Grade 0	No identifiable injury
Grade 1	Erythema and edema of mucosa
Grade 2a	Noncircumferential and superficial ulceration with white plaques or haemorrhage
Grade 2b	Circumferential injury or deep ulcerations with features of 2a
Grade 3a	Small or patchy necrosis
Grade 3b	Extensive or circumferential necrosis
Grade 4	Perforation before or during endoscopy

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 dilatation of caustic-induced esophageal strictures, however its use is still controversial [24]. Recently, mitomycin C has been used as an adjunct after dilatation 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].

e) **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 esophagealstricture [26]. The timing of management is crucial in achieving long term functional effects.

f) **Endoscopic dilatation**

First-line non-surgical treatment for strictures is dilation. Balloon ormechanical (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 dilatation force should be used initially to avoid perforation [28]. This may need to be repeated and advanced slowly to achieve effective and safe dilatation. The interval between dilatations varies from 1-3 weeks among different studies [29].

g) **Esophageal stents**

Though endoscopic dilatation 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.

h) **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 dilation 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

1. Turner A, Robinson P (2005) Respiratory and gastrointestinal complications of caustic ingestion in children. Emerg Med J 22: 359-361.
2. Elshabrawi M, A-Kader HH (2011) Caustic ingestion in children. Expert Rev Gastroenterol Hepatol 5: 637-645.
3. AAPCC (2016) Poison Center Data Snapshot – 2015. Annual Report of the American Association of Poison Control Centers, National Poison Data System, USA.
4. The University of Chicago (2016) Poison Center Data Snapshot - 2015. The University of Chicago, Chicago, USA.
5. Ekpe EE, Ette V (2012) Morbidity and Mortality of Caustic Ingestion in Rural Children: Experience in a New Cardiothoracic Surgery Unit in Nigeria. ISRN Pediatr 2012: 210632.
6. Contini S, Swarray-Deen A, Scarpignato C (2019) Oesophageal corrosive injuries in children: a forgotten social and health challenge in developing countries. Bull World Health Organ 87: 950-954.
7. Christesen HB (1994) Epidemiology and prevention of caustic ingestion in children. Acta Paediatr 83: 212-215.
8. Vezakis AI, Pantiora EV, Kontis EA, Sakellariou V, Theodorou D, et al. (2016) Clinical Spectrum and Management of Caustic Ingestion: A Case Series Presenting Three Opposing Outcomes. Am J Case Rep 17: 340-346.

9. Dogan Y, Erkan T, Cokugras FC, Kutlu T (2006) Caustic gastroesophageal lesions in childhood: an analysis of 473 cases. Clin Pediatr (Phila) 45: 435–438.

10. Haller JA, Andrews HG, White JJ, Tamer MA, Cleveland WW, et al. (1971) Pathophysiology and management of acute corrosive burns of the esophagus: results of treatment in 285 children. J Pediatr Surg 6: 578–584.

11. Havanond C (2002) Is there a difference between the management of grade 2b and 3 corrosive gastric injuries? J Med Assoc Thai 85: 340–344.

12. Kay M, Wyllie R (2009) Caustic ingestions in children. Curr Opin Pediatr 21: 651–654.

13. Gaudreault P, Parent M, McGuigan MA, Chicoine L, Lovejoy FH (1983) Predictability of esophageal injury from signs and symptoms: a study of caustic ingestion in 378 children. Pediatrics 71: 767–770.

14. SalzmanM, O'Malley RN (2007) Updates on the evaluation and management of caustic exposures. Emerg Med Clin North Am 25: 459–476.

15. Tiryaki T, Livanelioğlu Z, Atayurt H (2005) Early bougienage for relief of stricture formation following caustic esophageal burns. Pediatr Surg Int 21: 78–80.

16. Mutaf O, Genç A, Herek O, Demircan M, Ozcan C, et al. (1996) Gastroesophageal reflux: a determinant in the outcome of caustic esophageal burns. J Pediatr Surg 31: 1494–1495.

17. Han SY, Mc Elvein RB, Aldrete JS, Tishler JM (1985) Perforation of the esophagus: correlation of site and cause with plain film findings. AJR Am J Roentgenol 145: 537–540.

18. De Lusong MAA, Timbol ABG, Tuazon DJS (2017) Management of esophageal caustic injury. World J Gastrointest Pharmacol Ther 8: 90-98.

19. Cheng HT, Cheng CL, Lin CH, Tang JH, Chu YY, et al. (2008) Caustic ingestion in adults: The role of endoscopic classification inpredicting outcome. BMC Gastroenterol 8: 31.

20. Mamede RC, De Mello Filho FV (2002) Treatment of caustic ingestion: an analysis of 239 cases. Dis Esophagus 15: 210-213.

21. Keh SM, Onyekwelu N, Mc Manus K, Mc Guigan J (2006) Corrosive injury to upper gastrointenstinal tract: still a major surgical dilemma. World J Gastroenterol 12: 5223-5228.

22. Ertekin C, Alimoglu O, Akyildiz H, Guloglu R, Taviloglu K (2004) The results of caustic ingestions. Hepatogastroenterology 51: 1397-1400.

23. Katzka DA (2001) Caustic injury to the esophagus. Current Treatment Options in Gastroenterology 4: 59-66.

24. Kochhar R, Poornachandra KS (2010) Intralesional steroid injection therapy in the management of resistant gastrointestinal strictures. World J Gastrointest Endosc 2: 61-68

25. El-Asmar KM, Hassan MA, Abdelkader HM, Hamza AF (2015) Topical mitomycin C can effectively alleviate dysphagia in children with long-segment caustic esophageal strictures. Dis Esophagus 28: 422-427.

26. Katz A, Kluger Y (2015) Caustic material ingestion injuries-paradigm shift in diagnosis and treatment. Health Care Current Reviews 3: 1-4.

27. Siersema PD, de Wijkerslooth LR (2009) Dilation of refractory benign esophageal strictures. Gastrointest Endosc 70: 1000–1012.

28. Nishikawa Y, Higuchi H, Kikuchi O, Ezoe Y, Aoyama I, et al. (2006) Factors affecting dilation force in balloon dilation of severe esophageal strictures: an experiment using an artificial stricture model. SurgEndosc 30: 4315-4320.

29. Rathnaswami A, Ashwin R (2016) Corrosive Injury of the upper gastrointestinal tract: a review. Arch Clin Gastroenterol 2: 56-62.

30. Broto J, Asensio M, Vernet JM (2003) Results of a new technique in the treatment of severe esophageal stenosis in children: poliflex stents. J Pediatr Gastroenterol Nutr 37: 203-206.

31. Zhang C, Yu JM, Fan GP, Shi CR, Yu SY, et al. (2005) The use of a retrievable self-expanding stent in treating childhood benign esophageal strictures. J Pediatr Surg 40: 501–504.

32. Spitz L, Kiely E, Pierro A (2004) Gastric transposition in children: a 21-year experience. J Pediatr Surg 39: 276–281.

33. Tekant G, Eroğlu E, Erdoğan E, Yeşildağ E, Emir H, et al. (2001) Corrosive injury-induced gastric outlet obstruction: a changing spectrum of agents and treatment. J Pediatr Surg 36: 1004–1007.

34. Tseng YL, Wu MH, Lin MY, Lai WW (2002) Early surgical correction for isolated gastric stricture following acid corrosion injury. Dig Surg 19: 276–280.

35. Temiz A, Oguzkurt P, Ezer SS, Ince E, Gezer HO, et al. (2012) Management of pyloric stricture in children: endoscopic balloon dilatation and surgery. Surg Endosc 26: 1903–1908.

36. Ozcan C, Ergun O, Sen T, Mutaf O (2004) Gastric outlet obstruction secondary to acid ingestion in children. J Pediatr Surg 39: 1651–1653.