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Research Article

## Study of Some Gauges of Epidemiologic in Children with Caustic Ingestions That Admitted in Tehran Children Poison Center for Five Years (2015-2010)

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### Abstract

Caustic ingestions including Acids & Alkalians agents are the serious injuries for GI tract. Particularly esophagus and stomach. Those frequently in young children (below five years old). Severe caustic injuries have been reported from developing countries. Those common in males than females and often occurred accidental in children.

### Method

Our study was descriptive (retrospective) by extraction of some data of our patients (with caustic ingestion) from their files including (age-gender-acid or alkalian-severity of injury-out come). And compared them with other studies.

### Results

In this period (Dec 2010 to Dec 2015) we admitted 252 children with intoxication in our ward. 16 cases of them were caustic ingestions. (6.34%) (8 females & 8 males). Because of incomplete of two files of them, its out of our study. Then we had been 14 files (8 females and 6 males). The mean age of females was 4.25 & males 3.75 years. According to the PH of agents separated them to strong or weak acids and strong or weak alkalians. Ingestion of strong acids common than alkalians. According to the endoscopic findings, The 8 cases of them had esophagitis grade 2a, 2b or 3a. Because of we had not pediatric gastrologist, our patients were referred to another hospital. Then unfortunately we had not any information about there out come. The whole of our patients with oropharyngial. Signs and symptoms (including edema, erytoma, drooling, respiratory distress, erosion and ulcer) associated with severe findings in GI tract.

### Conclusion

Caustic ingestions are the serious injuries in GI tract particularly in children. Those often in young children (below five years old). According to the our study, Our patients unfortunately ingested strong acids, and for treatment and long time following of them must to be a pediatric gastrologist in this wards.

**Keywords:** Caustic Ingestion; Epidemiologic-Esophagitis; Poison Center

### Introduction

Caustic injury remains an important public health problem in the United States and the world despite various education and regulatory efforts to reduce its occurrence. In the United States, an estimated 5,000 to 15,000 caustic ingestions occur per year [1-3]. The age of occurrence of these ingestions

shows a bimodal pattern. 1 The first peak is seen in children aged 1 to 5 years, with most of these ingestions being accidental, though reports of child abuse have been reported in the literature [1,4,5]. The problem of caustic ingestion in children is spreading in developing countries, as these agents become more available, with much of the recent research coming from that part of the world. 6 The other peak age for

caustic ingestion is seen among adults aged 21 years and older. Most of the ingestions seen in this population are intentional suicide attempts [1,4,5].

Caustic ingestions may cause widespread injury to the lips, oral cavity, pharynx, and the upper airway. The effect that these agents have on the esophagus accounts for most of the serious injuries and long-term complications seen among both children and adults [3]. Short-term complications include perforation and death. Long-term complications include stricture and increased lifetime risk of esophageal carcinoma [7]. In children, 18% to 46% of all caustic ingestions are associated with esophageal burns. This number may be higher in adults who often consume larger amounts of the caustic substance as part of a suicide attempt [7,8]. Because of its importance, a large portion of the discussion will be concerned with esophageal injuries.

The nature of the injury caused by caustic ingestion is determined by a number of factors including the identity of the agent, the amount consumed, the concentration, and the length of time the agent is in contact with a given tissue [5,9]. Caustic materials cause tissue injury by chemical reaction. These materials are generally acidic or alkali. Usually, acids with pH less than 3 or bases with pH greater than 11 are of the greatest concern for caustic injury [10]. Alkalis contain bases dissolved in water.5 Examples are such agents as sodium hydroxide and potassium hydroxide (for complete list see Table 1). Lye is a general term for alkali found in cleaning products [7]. Caustic alkaline materials are also found in drain cleaners, various cleaning agents, hair relaxers, dishwasher detergents, and disk batteries [4,11].

**Table 1.** Summary founding of our patients.

**Sex:** Male(6)-Female(8)

**Mean of age:** Male(3.75)-Female((4.25)

**Caustic Agents:** strong Acid(10)-Weak Alkalian(3)-Strong Alkalian(1)

**Grade of Esophagitis:** 2a(8)-2b(4)-3a(2)

**Symptoms & Signs:** oral lesions(14)-drooling(12)-Respiratory distress(8)-Ulcer(3)

In liquid form, bases are tasteless and denser than water, resulting in pronounced distal injuries [1,9] Alkali ingestion causes liquefactive necrosis with diffusion into deeper layers of the injured mucosa. Saponification of fats, denaturation of proteins, and blood vessel thrombosis will occur during the injury process [1,4,5,11] This injury occurs quickly, with a 30% solution of sodium hydroxide being able to produce full thickness injury in 1 second [7] Even lower concentrations can produce extensive injury. A recent animal study from Brazil [12] showed that a low concentration solution (1.83%) was able to produce esophageal epithelial necrosis in 1 hour.

Like their liquid counterparts, solid strong alkali substances can produce deep injuries although they tend to adhere to the mucosa of the mouth and esophagus, thus sparing the stomach. Weaker alkalis that are powdered or granular, such as dishwasher detergents, tend to injure the upper airway and can cause laryngeal edema and airway compromise [1,8,13].

Alkaline disk batteries contain a 45% solution of potassium hydroxide or sodium hydroxide. Their ingestion will result in damage and leakage within 1 hour and in perforation in 8 to 12 hours if they lodge in the esophagus; this event mandates the immediate removal [4,5] If they are ingested and become lodged in the esophagus, an emergency situation arises.

Hair straighteners and relaxers containing calcium or lithium hydroxide are highly alkaline. Despite this, they rarely cause severe injury or squeal; thus, treatment is largely conservative with overnight observation and management [4]

Acids, in contrast to alkali substances, have a poor taste and are irritating, which may lead to a child's choking and gagging. This may predispose the patient to aspirate the caustic material, with subsequent airway compromise [1,4,9]. Examples of acids in commercial products include hydrochloric acid, sulfuric acid, and silver nitrate [4,11]. They are found in toilet bowl cleaners, swimming pool cleaners, and rust removers [4].

Acids are conventionally thought to cause coagulation necrosis that forms an Escher, preventing deep tissue penetration [1,4,11]. This Escher is thought to decrease the rate of esophageal burn, though pooling of these agents in the stomach is thought to predispose patients to gastric perforation and stricture [1,9]. One recent caustic ingestion series from Israel [3] questions this protective Escher theory, showing increased rates of esophageal perforation in patients who ingested acid.

Bleaches such as chloride bleach, peroxide, and mildew remover are also considered caustic esophageal irritants, though pH is typically neutral in commercial preparations [1,5,9] Because of the low concentrations and the neutral pH, bleaches do not cause extensive esophageal damage, though they may cause laryngeal edema and airway compromise in the short-term setting [1].

As mentioned above, the esophagus is the site of most long-term squeal from caustic ingestion. The esophagus is a 10 to 12 cm tube at birth, which increases to from 25 to 30 cm in the adult. It has 3 primary layers. The innermost is the mucosa, consisting of squamous epithelium, lamina propriety, and muscularis mucosa. A deeper layer is the submucosa, which consists of fibrous connective tissue with blood vessels, nerves, and numerous mucous glands. Finally, there is a muscular layer divided into inner and outer muscle layers [11]. The anatomic layers of the esophagus are of particular importance because most staging systems for injury are based

on findings seen on endoscopy that correlate with the depth of injury. Multiple staging systems for injuries are described in the literature, with older systems characterizing injury severity according to different degrees, similar to that used for burns of the skin. The system correlates findings seen on endoscopy. Eighty percent of patients with grade 3 burns develop stricture, while only one third of those with grade 2 burns will eventually develop stenosis [7].

Injury to the esophagus is rapid, as described above, for both acids and alkalis, but this acute tissue disintegration and deep tissue penetration may continue for hours. Injury progresses within the first week after ingestion, with inflammation and vascular thrombosis. A developing ulcer with fibrin crust will be seen in a few days. Granulation tissue develops between 2 to 4 days and is revealed under shed necrotic tissue by days 15 to 20. This is clinically relevant to healing because collagen deposition may not begin until the second week after injury; thus, the healing tissue will be less strong. This weakness may increase risk during endoscopy performed between 5 to 15 days after injury and may predispose the esophagus to spontaneous rupture during this period [4,14].

### Presentation and Treatment

After caustic ingestion, patients may present with a combination of many symptoms or none at all depending on the nature of the agent, the specifics of the ingestion (quantity, intent, timing), and what tissues were affected. Patients may have obvious burns to the lips, mouth, and oropharynx. The presence or absence of these lesions does not correlate with the presence of injury to the esophagus or stomach [1,5]. Patients with significant laryngeal or epiglottic edema may present with stridor, aphonia, hoarseness, or dyspnea [1]. More non-specific presenting symptoms include nausea, recurrent emesis, hematemesis, dysphagia, odynophagia, and drooling. The presence of abdominal pain or rigidity as well as sub sternal/chest or back pain may be a sign of severe burn or perforation [1,5,11]. Some authors [10,14] claim that presentation with 2 or more symptoms may suggest esophageal injury, but there is no 100% accurate sign or group of symptoms that indicates esophageal injury.

The adequacy of the patient's airway must first be addressed. Fiber optic laryngoscopy can be useful in this regard. If the airway is unstable, intubation under direct visualization is required. "Blind" intubation can lead to bleeding and additional injury and make additional attempts more difficult. If it is impossible to secure the airway through intubation, a surgical airway may be required [1,4,5] When the airway is secured, a thorough physical examination should be completed and a thorough history taken, with particular attention to the timing of the ingestion, identity of the agent, and amount ingested [1,4,5,9] Chest and abdominal radiographs should be obtained

to detect free air in the mediastinum (esophageal perforation) or under the diaphragm (gastric perforation) as well as to provide a baseline if aspiration pneumonia develops [5] Furthermore, oral food and fluids should be withheld from the patient, who should, however, receive aggressive hydration. Laboratory results and vital signs should be assessed for signs of acidosis and shock. Induction of emesis should be avoided to prevent further injury as the agent is vomited [11]. Neutralization of the caustic material should be avoided because of the potential for causing an exothermic injury, which may worsen an existing injury [1]. When the patient is stable, the pH of any unknown liquid or agent should be obtained, and the poison control center should be contacted [4,5,9].

Multiple modalities exist to assess the extent of injury to the esophagus, including barium esophagram, technetium-labeled sucralfate, and endoscopy [1]. Barium esophagram assesses mild to moderate esophageal burns with a 30% to 60% false-negative rate and is therefore of little use in the short-term setting [1]. It is, however, quite useful for following the development of late complications and strictures. Technetium-labeled sucralfate swallow has high sensitivity and specificity but limited ability to show extent of injury. Endoscopy, specifically flexible esophagoscopy, is the most effective method for visualizing the extent of esophageal injury [4]. Rigid esophagoscopy may be used but should not be extended beyond the site of caustic burn because of an increased risk of perforation. The flexible endoscope may be advanced past a site of mild injury but should only be stopped at the site of circumferential grade 2 or 3 burns [1,7]. Additionally, the flexible esophagoscope provides important information about the stomach and duodenum. Rigid endoscopy is recommended for nasogastric tube placement and airway management [1].

The timing of endoscopy and the circumstances for its use, as recommended in the literature, are controversial. In the past, there was a tendency to wait at least 24 hours to allow time for the injury to mature [5]. Most authors [4,7] are recommending earlier endoscopy and suggesting a wait of only 12 hours and a total wait of no more than 24 hours after ingestion for early assessment and treatment. Endoscopy past 48 hours is discouraged because of progressive wall weakening and increased risk of perforation [1]. Most agree that strong alkali ingestion mandates endoscopy, while asymptomatic or questionable ingestions may be observed, according to some sources [1,4].

Treatment schemes for caustic injuries vary and no consensus exists. Multiple protocols have been formulated on the basis of documented success in preventing impending complications, though a paucity of randomized trials have assessed any of these interventions. Nasogastric tube placement under endoscopic guidance has been suggested for grade 2b and 3 injuries to stent the injured area and to provide nutritional support [2]. Stent placement has shown success in some series [6] though

timing and type vary. Stents should be left in place for 14 to 21 days to allow for epithelization [4]. Early dilation has been proposed, but has been found to increase rate of perforation [8]. Regardless of the intervention, pain control is essential. Antibiotics are often used and have been shown to increase epithelization in animal models though they do not change stricture formation rate or infection rate in numerous series [1,4]. Antacids decrease pepsin and acid exposure, which may delay healing of the esophagus, though again, rigorous trials are lacking. Larythrogonic drugs, such as N-acetylcysteine and penicillamine, and mitomycin, an antibiotic and antineoplastic agent, decrease rate of collagen crosslinking and inhibit protein synthesis, respectively, and decrease scarring and stricture formation in animal models [1,5]. Data from human trials are lacking [4].

Steroids have been a controversial treatment since they were first shown to decrease granulation and stricture formation in animal models [4]. Some prospective and retrospective reports have shown that steroids decrease stricture formation in grade 2 injuries in humans [1,9,10]. Recent meta-analyses have revealed conflicting results. One recent analysis in 2005 included a total of 10 studies [15] in which 572 patients showed no decreased incidence of stricture, with steroids used for second and third-degree (grade 2 and grade 3) burns. In contrast, a meta-analysis of 362 children showed a 40% rate of stricture in the no-steroid group versus a 19% rate of stricture in the steroid-treated group [14].

The landmark randomized trial by Anderson et al [2] found no difference in the incidence of stricture formation with the use of steroids, though the number of patients in the study was relatively low. A definitive study on the value of steroids is lacking but, if used, most resources suggest the concurrent use of antibiotics [2,4,7]. Dosing is controversial and recommendations vary widely [1,8]. Side effects of steroids, especially vulnerability to infection, should be considered [2].

## Method

Our study was descriptive (retrospective) by extraction of some data of our patients (with caustic ingestion) from their files including (age-gender-acid or alkalian-severity of injury-out come). And compared them with other studies

## Result

In this period (Dec 2010 to Dec 2015) we admitted 252 children with intoxication in our ward. 16 cases of them were caustic ingestions. (6.34% 8 females & 8 males). Because of incomplete of two files of them, its out of our study. Then we had been 14 files (8 females and 6 males). The mean age of females was 4.25 & males 3.75 years.

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acids and strong or weak alkaliens. Ingestion of strong acids common than alkaliens. According to the endoscopic findings, The 8 cases of them had esophagitis grade 2a, 2b or 3a. Because of we had not pediatric gastrologist, our patients were referred to another hospital. Then unfortunately signs and symptoms (edema, erytoma, drooling, respiratory distress, erosion and ulcer) associated with severe findings in GI tract.

## Conclusion

Caustic ingestions are the serious injuries in GI tract particularly in children. Those often in young children (below five years old). According to our study, Our patients unfortunately ingested strong acids, and for treatment and long time following of them must to be a pediatric gastrologist in this wards. The whole of our patients with oropharyngial signs and symptoms (including: edema, erytoma, drooling, respiratory distress, erosion and ulcer) had been severe injuries in their GI tract.

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