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Caustic Ingestion in Children

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Abstract

Caustic ingestion (CI) is an unfortunate event that occurs in families with a poor prevention culture. Its prevalence is unknown in developing countries; it occurs mainly in children <5 years and is more common in boys. The chemical caustic agents are alkaline (85%) or acid products stored in food or beverage containers without warning labels and safety caps. The immediate symptoms include salivation, oropharyngeal burns, vomiting and oropharyngeal/retrosternal pain. Upper endoscopy is the first-line tool to identify the type and extension of oesophageal and gastric damage. A barium swallow performed 2–3 weeks after the CI may identify oesophageal stricture. Dysphagia occurs in about one-third of cases. Regarding the nutritional status, children with dysphagia and/or oesophageal strictures may have lower fat reserves or muscle mass than the cases without these complications, meaning impaired nutritional status. All patients should be hospitalized for evaluation and treatment. Hemodynamic stabilization and adequacy of the patient's airway are priorities; vomiting induction and gastric lavage are contraindicated. Methylprednisolone in II-b oesophageal burns for 3 days diminishes the risk of stricture. Selected cases will require oesophageal dilatations, gastrostomy or oesophageal replacement by colon or stomach. There are other promising agents in the management of caustic oesophageal strictures.

Keywords: caustics, oesophageal burns, oesophageal stricture, dysphagia, child malnutrition

1. Introduction

CI in children and adolescents is an unfortunate event that may lead to significant upper gastrointestinal tract damage manifested in the short term as oesophageal burns and in the long term as oesophageal stricture or acquired motility disorders. A high proportion of

these patients may evolve to permanent disability associated to feeding difficulties as well as growth and nutritional impairment. The aim of the chapter is to discuss the experience with our CI paediatric patients treated in a paediatric referral hospital in the context of the published data from other authors.

2. Epidemiology

Although is not a public health problem, CI is a significant issue worldwide, particularly in developing countries in which an unsafe environment is a substantial risk factor for child injury [1]. In 1985, Wasserman reported that approximately 5000 children younger than 5 years ingested lye each year in the United States [2]; in 2008, the USA National Poison Data reported over 200,000 exposures to caustic substances [3]. Unfortunately, the true prevalence regarding this health problem is not known in most developing countries and cannot be extrapolated from publications of series of children with CI treated in paediatric hospitals; however, these publications describe demographic, clinical and treatment characteristics of children from low, lower-middle and high-income countries information is scarce specially in lower-middle income countries [1].

Overall, the paediatric population is the most affected; the highest risk age groups are infants and pre-schoolers [4–13]. This may be attributed to the fact that children younger than 6 years are not able to make reasonable decisions about what they should or should not drink in the context of their developmental stage of motor and sensory skills [8]. Regarding gender, the higher frequency trend of CI in males could be attributed to both a gender-related biological and learned gender-role behaviour of boys different from girls in terms of expressing health and disease and their interaction with risk factors [4–7, 13–15]. A recent publication reveals that children with attention deficit/hyperactivity disorder had a higher risk to suffer CI than children without this condition [16].

Reports related to the social, economic, and educational characteristics that may play a role in families of children with CI are scarce [17]. In this context, the authors performed a case-control study in which the aim was to evaluate the association of socio-demographic factors in a group of 94 children and adolescents with CI; the controls were a random sample of children hospitalized or seen as outpatients in the same paediatric referral hospital and period. The sociodemographic variables were studied using a validated questionnaire. CI occurred at home in 63.8% and at a relative's home in 23.4%. Alkaline products were the most frequently ingested (85.1%); containers had no warning labels in 72.3% and no childproof safety caps in 92.6% of the cases. The sociodemographic variables associated with CI included higher family income, mother's lower education, higher proportion of fathers working as independent professionals, extended family, mother's age <30 years, and mother's working outside home. The fact that both parents work outside the home and that the mother has a low educational level may reflect a lack of awareness and knowledge of the hazards of corrosive substances kept in the house. Family organization classified as extended implies crowded living conditions and enables both parents to work outside home while other family members take care of their children [18]. Other authors

have reported that unsafe homes where safety rules regarding caustic substances are lacking have been identified as a substantial risk factor for CI in children [1, 9, 13, 17, 21]. Sarioglu-Buke *et al.* found that children from families where both parents with low educational level and low socioeconomic status had a higher risk for a CI event. Urganci *et al.* found in a series of 1709 cases that 30% of the mothers were illiterate [13]. Contini *et al.* described most CI particularly in developing countries as being due to parent's lack of knowledge of the hazards of corrosive substances kept in the house, especially among illiterate individuals [1]. Risk factors are summarized in **Table 1**.

2.1. Physiopathology

The physical form, concentration, and pH of the corrosive agent play a significant role in the location and type of resultant injuries [2]. Acid substances lead to coagulation necrosis that usually limits acid penetration and results in damage to the epithelium and submucosal layer. Their low viscosity and specific gravity result in rapid transit to the stomach, and gastric injury is more common than oesophageal injury. Their bitter taste and development of pain with ingestion may result in lower volumes of ingestion. The gastric injury may result in gastric outlet obstruction or perforation [19]. The ingestion of alkalis causes liquefaction necrosis with damage of the epithelium and the submucosa layer with deep penetration and may result in perforation [2]. Injury most typically involves the oesophagus, but the gastric injury may also occur.

Haemorrhage, thrombosis and a marked inflammatory response with significant oedema are seen within the first 24 h of injury [19]. The tissue repair phase extends from the end of the first week through the second week after injury and if the insult has been relatively minor, oesophageal function returns gradually. The healing phase begins around the third week when fibroblast proliferation replaces the submucosa and *muscularis mucosae*; at this time, stricture formation begins [2, 19]. Additionally, lower oesophageal sphincter pressure becomes impaired, leading to increased gastroesophageal reflux, which in turn accelerates stricture formation [20].

Age	Below 6 years old
Gender	Boys
Alkali or acid caustic substances at home	Improper storage Food or drinks storage containers Lack of warning labels Lack of security caps
Extended families	Both parents working away from home, children cared for other family members and mother with a low education level

Table 1. Risk factors for caustic ingestion in paediatric patients [18, 21].

In most series, the most commonly reported corrosive agents are alkaline products as caustic soda, sodium hypochlorite and household chemicals [13]. It is essential to highlight that most of these products ingested by children were kept in containers that had no warning labels and no childproof safety caps. These key risk factors should be taken care by worldwide governments by means of health education and implementation of legislative and preventive strategies on labeling, formulation, and packaging of corrosive substances [1, 21].

2.2. Clinical manifestations

The ingestion of acid or alkali substances frequently leads to damage to the gastrointestinal tract. The spectrum damage ranges from mild to severe and may affect the mucosa as well as the muscular layer. In a study performed by the authors in a paediatric referral hospital, the aim was to evaluate the association of clinical data with the oesophageal damage of 94 children who suffered CI. The mean age when the CI occurred was 38.4 months (SD 28.7). Thirty-five cases (37.2%) were females. The higher frequency was in infants (40.4%) and pre-schoolers (45.7%). The symptoms recorded were salivation (77.7%), oropharyngeal burns (76.6%), and vomiting (74.5%), oropharyngeal pain (45.7%), and epigastric pain (18.1%). Symptoms of airway involvement included shortness of breath (14.9%) and retrosternal pain (10.6%) [19]; these signs and symptoms are similar to those reported by other authors in paediatric series [3–7, 9, 12, 13, 21].

2.3. Diagnosis

In a number of cases, CI patients may be symptom-free on admission; in spite of this, endoscopy is mandatory as an effective tool to identify both oesophageal and gastric damage [22]. Even more, the presence or absence of symptoms does not predict the injury severity to the gastrointestinal tract, supporting the endoscopic procedure [23, 24]. The degree of the mucosal injury should be determined by a standardized classification; most colleagues use the Zargar's classification: grade 0 = normal; grade I = oedema and erythema; grade II = linear ulcerations and necrotic tissue with whitish plaques; grade II-a = linear ulcerations and necrotic tissue with whitish plaques and deeper and more circumferential; grade III = circumferential injury which may be transmural with mucosal sloughing, multiple deep ulcerations and areas of necrosis (areas of brown-black or greyish discoloration of mucosa were taken as evidence for necrosis); grade III-a = focal necrosis; grade III-b = extensive necrosis; and grade IV = perforation [25]. A study performed in 206 children reported no complications when the endoscope was advanced beyond the first severe burn; this approach could identify additional oesophageal and gastric lesions that could be missed if the endoscopy is discontinued when finding the first circumferential oesophageal burn, as is usually recommended because of perforation risk [23].

In our series, all cases underwent upper endoscopy within 48 h of CI; in 66 of 78 children (84.6%), we identified moderate or severe oesophageal burns, 76.9% had grade II and 7.7% grade III. An endoscopy plus a barium swallow were repeated after 2 or 3 weeks of the CI to assess mucosal healing and to look for strictures; mucosal healing occurred in all cases but 64.5% had oesophageal stricture [21]. Clinical, endoscopic and image data of our cases are summarized in **Table 2**.

	Variable	<i>n</i>	%
Symptoms and signs	Salivation	73	77.7
	Oropharyngeal burns	72	74.5
	Vomiting	70	70.3
	Oropharyngeal pain	42	45.7
	Epigastric pain	17	18.1
	Shortness of breath	14	14.9
	Retrosternal pain	10	10.6
	Septic shock ^a	4	4.2
	Dysphagia ^b	23	24.5
Endoscopy	II or III degree esophageal burns ^c	79	84
	Esophageal stricture ^d	46	48.9
Barium swallow	Esophageal stricture	46	48.9

^aOesophageal perforation.
^bWeeks/months after the caustic ingestion.
^cAdmission endoscopy.
^dEndoscopy performed 2–3 weeks after admission.

Table 2. Clinical data in 94 children with caustic ingestion [21, 38].

In 2012, Uygun *et al.* developed a tool for predicting risk of developing an esophageal stricture without endoscopy called the DROOL score based on the severity and duration of initial signs and symptoms (drooling, reluctance to eat, presence of burns in oropharynx, fever, hematemesis, abdominal tenderness, dyspnea, chest pain and leukocytosis). A DROOL score ≤ 4 was a significant predictor of esophageal stricture with 100% sensitivity and 96% specificity; this score needs further investigation and validation [26]. It has been recently published that a CT scan may offer a more detailed evaluation than early endoscopy in assessing the transmural damage of esophageal and gastric walls and the extent of necrosis; a CT grading system has also been proposed to predict esophageal stricture [27]. These important findings deserve to be validated because this could mean a significant change in the diagnostic approach of these patients.

2.4. Complications

Acute complications such as mediastinitis and oesophageal perforation may occur and they may represent life-threatening complications; in selected cases, chest and abdominal radiographs are needed to rule out the presence of free air in the mediastinum or peritoneum. Lateral neck radiographs should be obtained in patients with stridor or hoarseness [28].

Long-term complications frequently include oesophageal stricture and dysphagia [4–9, 11, 13, 18]. In our series, oesophageal stricture occurred in 48.9%; II and III-degree burns were

associated with oesophageal stricture ($p = 0.004$) and ingestion of alkaline products ($p = 0.027$). The observation that one-half of patients with stricture did not complain of dysphagia and that some patients with dysphagia had no oesophageal strictures, points to the underlying complexity of the functional and anatomical oesophageal damage induced by the chemical agent as well as to the individual variation in visceral sensitivity [29].

Several studies have reported oesophageal dysmotility after a CI; oesophageal manometry has revealed hypoperistalsis, with normal upper and lower oesophageal sphincter [30, 31]. Rana *et al.* showed that patients with corrosive injury have prolonged oro-cecal transit time using the lactulose hydrogen breath test, even in the absence of gastric symptoms; this transit time was maximally prolonged in patients with lower third oesophageal scars [32]. Gastric emptying time assessed by radionucleotide scintigraphy after a CI was significantly prolonged in patients with oesophageal stricture, even in the absence of gastric symptoms [33]. Another study reported that oesophageal transit time assessed by scintigraphy was prolonged in one-third of patients with corrosive induced oesophageal strictures, despite having achieved adequate oesophageal dilatation; these authors found that the prolongation of oesophageal transit time correlated with the length of the stricture and that the severity of the dysphagia correlated with the prolongation of total oesophageal transit time [34].

CI with stricture has been associated with increased risk of oesophageal carcinoma with an incidence of 1000 times the expected in normal population; this important finding points the need for endoscopic surveillance 15–20 years after the caustic injury [28, 35].

The severity of the oesophageal damage that occurs frequently in children with CI may limit the normal feeding process leading to malnutrition and growth impairment [18, 22, 36]; it is surprising that there is very little published information regarding the nutritional status. In France, Ganga-Zandzou *et al.* found that the nutritional status in 34 children with oesophageal stricture was not affected [37]; whereas, in Egypt, Hamza *et al.* recorded 15% of malnutrition [15]. In another study performed by the authors, the aim was to evaluate the nutritional status and its association with dysphagia and oesophageal stricture in 62 children with CI. Although the proportion of cases with z-scores of height for age and weight for height below $-2SD$ was low, all anthropometric arm indicators (fat and muscle) of the children with dysphagia and oesophageal strictures were located in the negative area of the z-score curve. In all cases, adiposity z-score values were significantly lower in children with dysphagia or strictures when compared with the cases without these complications. Arm muscle area z-scores showed the same trend. We concluded that children with dysphagia and/or oesophageal strictures associated to CI had lower fat stores and muscle mass than the cases without these oesophageal complications, meaning impaired nutritional status [38]. In this context, both growth and nutritional status surveillance as an effective nutritional intervention protocol should be part of the mid- and long-term monitoring of these patients.

2.5. Treatment

All patients who suffered a CI should be hospitalized and evaluated comprehensively. The clinical history must emphasize the type and amount of caustic and if possible get the product

package. Hemodynamic stabilization and adequacy of the patient's airway are priorities. The induction of vomiting or gastric lavage is contraindicated following CI because a re-exposure of the oesophageal mucosa to the caustic agent with further injury may occur [35]. An endoscopic evaluation with an adequate paediatric endoscope performed within the first 24–48 h is essential to evaluate the type and extent of injury to the oesophagus and stomach.

The information about the efficacy of proton-pump inhibitors and H₂ blockers to minimize the oesophageal injury in subjects whom ingested caustic is scarce. Cakal *et al.* performed an open clinical trial on 13 adult patients who were admitted due to CI and received omeprazole 80 mg in bolus IV, followed by continuous infusion of 8 mg/hour for 72 h. A control endoscopy was performed 72 h after admission, identifying a significant difference regarding endoscopic healing between the before and after omeprazole infusion ($p = 0.004$); however, this study lacks a control group and is not possible to draw definitive conclusions [39].

The use of corticosteroids is controversial; two meta-analyses did not demonstrate benefits of steroid administration in terms of stricture prevention [40, 41]. In a recent study on 83 children with II-b oesophageal burns, one group received methylprednisolone (1 g/1,73 m/day/3 days), ranitidine, ceftriaxone, and total parenteral nutrition; the control group received the same regimen except methylprednisolone. The outcome variable was oesophageal stricture and it was more frequent in the control group ($p = 0.038$). The duration of total parenteral nutrition was shorter in the steroid intervention group compared with the control group ($p = 0.001$). The authors concluded that high doses of methylprednisolone used for the management of grade II-b oesophageal caustic burns may reduce the risk of stricture [40–42].

The use of antibiotics is controversial and should be considered in each particular case according to the clinical situation, laboratory inflammation values, the extent and type of mucosal damage and the concomitant use of steroids. The indications, dosages, duration and type of antibiotics are usually included in the protocols of each hospital serving this type of patients [19].

During the course of treatment, some patients may require oesophageal dilations and gastrostomy either for retrograde oesophageal dilations or trans-gastrostomy feeding. Topical mitomycin C application has been used recently to improve the results of endoscopic dilatation for short oesophageal strictures and is a promising agent in the management of the long-segment caustic oesophageal strictures, although long-term follow-up is needed to prove its efficacy and to evaluate potential long-term side effects [43]. The use of oesophageal stents and balloon dilators are known options for treating strictures [5, 28, 35].

A small number of cases with strictures refractory to dilation may require oesophageal replacement by a colonic graft or stomach's plasty to set up a gastric tube. Ezemba *et al.* reported a retrospective study of 21 patients who underwent substernal isoperistaltic colonic interposition graft for the management of corrosive oesophageal stricture. The long segment strictures and multiple strictures were the main indications for the procedure; the complications included cervical fistulae (19%), reflux neo-esophagitis (14.3%), graft infarction (9.5%); lethality occurred in 9.5%. In the mid term, dysphagia was completely relieved in 84% of the cases, no regurgitation or nocturnal aspiration was reported [44].

Special attention should be placed on growth, nutritional status and the emotional situation of the affected children and their families. A limited oral intake, decrease in growth velocity and low-fat reserves or muscle mass may require special dietary regimens with adequate energy and micro-nutrient intake by means of blended or polymeric diets. A CI is a traumatic event that may shake families emotionally, and in some cases, they may require emotional support by family therapists.

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