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Original Article

Caustic Ingestion in the Paediatric Age: Single Centre Experience

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Abstract ***Purpose:*** This study aimed to evaluate the clinical consequences of caustic ingestion and identify prognostic risk factors. ***Methods:*** All children under the age of 18 years who presented to our emergency department between January 2020 and January 2021 due to corrosive substance ingestion and underwent endoscopy were included in the study. ***Findings:*** The study population included 176 patients (male/female: 96/80). Alkaline agents were more commonly ingested than acidic agents (84.7%). None of the patients who had ingested acidic agents had esophagitis, while 20 (13.4%) patients who had ingested alkaline agents had oesophageal burns. Two asymptomatic patients had second-degree oesophageal burns. Oesophageal stenosis was detected in 2 of 8 patients with Grade ≥2A oesophageal lesions, and dilatation was performed. ***Conclusions:*** Our data suggests that endoscopic evaluation is mandatory in patients who ingest alkaline agents, although it may be avoided in patients who ingest acidic agents, especially in asymptomatic patients.

Key words *Acids; Alkalies; Caustics; Child; Endoscopy*

Introduction

Accidental ingestion of caustic agents is common in children[1,2] and a common cause of visits to the hospital. Notably, it is more prevalent among children from low-income families. Easy access to corrosive household products and indiscriminate use of them are determining factors for these injuries, especially in developing countries. However, this is an important public health issue with a serious risk of morbidity and mortality and could result in serious complications such as respiratory distress, oesophageal stricture and perforation.

Endoscopy plays a fundamental role both in the diagnosis and determination of prognosis, as well as subsequent management. Because of the discrepancy between clinical and endoscopic findings, clinicians often have difficulties in deciding between endoscopy and management.[3] The European Society for Paediatric Gastroenterology Hepatology and Nutrition (ESPGHAN) recommend performing endoscopy in symptomatic patients within 24 hours after corrosive ingestion (strong recommendation, high-quality evidence).[4] However, several studies have reported that clinical findings are not always good predictors of the degree of injury and possibility of stricture formation.[5-8]

This study aimed to analyse epidemiological data, endoscopic findings, and clinical consequences of caustic ingestion, as well as risk factors affecting prognosis.

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Methods

This single-centre retrospective study was conducted on paediatric (<18 years) patients diagnosed with acute caustic ingestion from January 2020 to January 2021. All children referred to our tertiary paediatric hospital post-caustic ingestion underwent routine blood analysis and upper endoscopy. Routine endoscopy was performed within 24-48 hours from the time of ingestion. Oesophageal injuries were graded according to Zargar's endoscopic classification (Table 1),[9] whereas gastric damage was classified as either mild (gastric hyperaemia) or severe (gastric ulcer).

Demographic data, type of the caustic agent (acidic or alkaline), concentration of the substance, cause of ingestion (accidental or suicidal), and symptoms and signs were collected from medical records. In addition to the clinical examination, the diagnosis was made based on the parents' account of history of exposure, including the brand. All patients presented with complaints such as fever, vomiting, drooling, dyspnoea, and dysphagia. They showed signs such as pharyngeal, lingual (mouth) or lip burns, and respiratory findings. It is noteworthy that induced vomiting was not considered a symptom.

Experienced paediatric gastroenterologists performed the upper endoscopies following deep sedation administered by an anaesthesiologist. The Zargar classification was used to estimate the severity of the lesions.[9] The patients with Grade 0 lesions were orally fed and discharged after 3-4 hours after endoscopy without therapy. The patients with Grade 1 oesophageal injury were orally fed and discharged within 8 hours after endoscopy and prescribed proton pump inhibitors (PPI). The patients with Zargar ≥2A were hospitalised and monitored daily for symptomatic convalescence. Grade 2A patients were fed liquid food products 24 hours after the endoscopy and treated with proton pump inhibitors, sucralfate, and intravenous antibiotics. The patients with Grade 2B and 3 lesions were given proton pump inhibitors, sucralfate, intravenous antibiotics, parenteral nutrition, and steroids. An esophagogram was performed 3-4 weeks after injury to assess oesophageal stricture in the patients with Grade ≥2A oesophageal lesions. Oesophageal dilations were performed with a balloon or Savary's dilator if needed.

Table 1 Zargar's grading classification of mucosal injury of esophagus caused by corrosive agents

Grade 0	Normal examination
Grade 1	Oedema and hyperaemia of mucosa
Grade 2A	Superficial ulcerations, haemorrhage, erosions
Grade 2B	Circumferential ulcerations
Grade 3A	Deep widespread ulcers and areas of necrosis in gray-black colour
Grade 3B	Perforation

The patients with an unconfirmed diagnosis and those discharged on request against medical advice were excluded from the study.

Complete blood count and venous blood gas analyses, including lactate, were performed. Chest radiographs were also included.

Statistical Analysis

The IBM SPSS Statistics for Windows Version 25 (IBM Corp., Armonk, NY, USA) was used for all the statistical analyses. Categorical variables were compared using a Chi-square test, and numerical variables were compared using the Mann-Whitney U test. We conducted a multivariate logistic regression analysis to identify predictive factors for oesophageal and gastric caustic injuries.

The study was approved by the local ethical committee.

Results

During the study period, a total of 343 children suspected of acute caustic ingestion were admitted to our emergency department. Of these, 313 patients confirmed that they had consumed a corrosive agent. A total of 137 of them were excluded from the study because they refused to undergo endoscopy. The study population included 176 patients (M/F: 96/80; age: 2-199 months; mean age ± standard deviation: 31.95 ± 31.83 months). The frequency of caustic agent ingestion was more common in boys (54.5%, 95% CI: 1.47-1.62); however, this difference was not statistically significant.

A total of 91 of 176 (51.7%) were infants (<2 years old), 77 (43.8%) were children (2-12 years old), and 8 (4.5%) were adolescents (13-18 years). The vast majority of patients (160 patients; 90.9%) were younger than 5 years of age. About 91% of patients were consulted as outpatients; the rest were hospitalised. Regarding the percentage distribution of the nature of ingested substances, 149 out of 176 (84.7%) patients had ingested

alkaline agents, whereas the remaining 27 (15.3%) had ingested acidic agents. The most commonly ingested caustic agent was 5% sodium hypochlorite (household bleach), which was involved in 69 of 176 patients (39.2%).

At admission, 161 of 176 (91.5%) patients were asymptomatic. The most frequently reported symptom was vomiting (9 patients). Family members induced vomiting in 30 children (17%) before admission to the hospital, whereas 2 children were fed with yoghurt and one child was given milk. Seven (4%) patients had obvious signs of oral cavity lesions (tongue and/or lip burns, ulcerations), 4 (2.3%) had drooling, and 1 patient had respiratory distress.

Upper endoscopy was performed in all patients at a mean time of 32 hours (range: 13-74 hours) after ingestion. A total of 116 (66%) patients had no caustic injury of the oesophagus or the stomach. Of all, 12 patients (6.8%) had oesophageal erythema (Grade 1), 5 patients (2.8%) had non-circumferential superficial ulcerations and erosions (Grade 2a), and 3 (1.7%) had deep, discrete or circumferential ulcerations (Grade 2b). None of the patients had Grade 3 injuries. Gastric hyperaemia was reported in 40 patients, but gastric ulcer and duodenal injury were not found. Of note, when we evaluated oesophagus lesions according to the chemical feature of the ingested agent, we found that all patients with oesophageal burns were patients who ingested alkaline agents. The classification of esophagogastric lesions in the patients according to the ingested agents is shown in Table 2.

Eight patients had Grade 2 oesophageal injuries, and four of them had no complaints at admission to our emergency department. Moreover, 2 of them had no signs on physical examination.

Table 2 Distribution of corrosive agents according to grades of esophagitis

	Grade 1 Esophagitis	Grade 2a Esophagitis	Grade 2b Esophagitis
	n	n	n
Household bleach	4	1	0
Fat solvent	3	2	2
Drain opener	0	1	1
Dishwashing detergent	3	0	0
Blondine	0	1	0
Warts medicine	0	0	1

The results of the laboratory tests were not well correlated with the degree of the injury. Leucocytosis was detected only in 3 patients with Grade ≥2A oesophageal lesions. In only one of these patients with Grade 2b oesophageal injury, the leucocyte count was found to be over 20.000 cells/mm^3.

Eleven (6.25%) patients were hospitalised, but none of them had to be admitted to the paediatric intensive care unit.

At follow-up, 8 patients who had Grade ≥2A oesophageal lesions underwent an esophagogram 3-4 weeks after injury to assess oesophageal stricture regardless of symptoms. Among these, 2 (25%) were found to have oesophageal stricture and underwent dilation. These 2 patients had Grade 2b oesophageal injuries.

According to the multivariate logistic regression analysis, leucocytosis, anaemia, and thrombocytosis at first admission were not associated with oesophageal injury. However, high lactate was not found to be a predictor of oesophageal damage. Nevertheless, alkaline rather than acid agents were associated with a worse endoscopic score (r: 0.49).

Discussion

Caustic ingestion is an important public health problem, especially in developing countries, among individuals with a poor socioeconomic status.[10] It could lead to harmful consequences and impose an economic burden for diagnosis, treatment and follow-up. Although accidental caustic ingestion is relatively common among paediatric patients, the probability of death or serious injury is lower than in adults.[11]

In our study, caustic ingestion was more common among boys; however, this difference was not statistically significant. This finding is in line with the findings of a meta-analysis from 2016,[11] which reported a slight tendency towards the male gender, but it was not considered a risk factor.[11] In accordance with previously published data, 91% of our patients were younger than 5 years of age (mean age = 31.95 months).[10-12] The factors favouring these accidents could be a typical curiosity of this age and sex, caregivers' neglect, and parents' common habit of keeping caustic agents in beverage bottles.[6,13] In our study, the majority of corrosive agents ingested were alkaline, among which sodium hypochlorite was the most commonly ingested agent, accounting for 39.2% of the cases. A review published in 2016 found that most

accidental ingestion by children involved alkaline substances such as bleach products, oven cleaners, and soda.[11] The dominance of corrosive substances can differ depending on the country or the region,[14,15] probably because household bleach is widely used in house cleaning in our country. The initial clinical symptoms and signs exhibited by patients, such as vomiting, oral and/or oropharyngeal lesions, and drooling, were not predictors of more severe injuries, according to our results. This may be because solid agents get stuck in the mouth and pharynx due to their quick attachment to those sites, while fluid agents pass through them, causing significant damage to the oesophagus and stomach. In a study by Balderas in Mexico, the clinical symptoms were not predictors of more severe injuries, similar to our results.[6]

However, we did not observe serious oesophageal damage in most of the patients. This could be attributed to the fact that most of the children in our study were under 5 years old and may have swallowed only small amounts of corrosive agents accidentally. A total of 8 out of 176 (4.5%) patients had Grade 2 oesophageal damage. The oesophageal stricture was observed in two of them.

Interestingly, all the patients with oesophageal damage had ingested alkaline agents. None of the patients who had ingested acidic agents had esophagitis or oesophageal stricture. A total of 7 of 27 (26%) patients had gastric hyperaemia, probably related to the different degrees of tissue damage induced by acids and alkalis. Ingestion of acids usually causes superficial coagulation necrosis.[15] Acidic substances have less surface tension and, therefore, rapidly reach the stomach because of their low viscosity and specific gravity, resulting in more gastric than oesophageal injuries. Upon swallowing, acids cause severe oropharyngeal pain; thus, they are usually consumed in small volumes because of the pungent smell and a disagreeable taste compared with alkaline substances.[1] On the contrary, alkaline agents are usually colourless and relatively tasteless. Alkaline agents cause liquefactive necrosis and saponification, resulting in deep penetration of tissue, mainly limited to the oesophagus.[16] This kind of necrosis leads to the destruction of oesophageal mucosa within a few minutes.[15] In addition, alkaline fluids have stronger surface tension and stay in the tissue for a longer period, thereby worsening the injury. Within the stomach, injuries are limited because gastric acid can partially neutralise the alkaline agents.[1] In a study published in 2020 in Italy, alkaline agents rather than acid agents were associated with a worse endoscopic score and a higher probability of oesophageal stricture, as found in our study.[17]

The grade of lesions was determined based on Zargar's endoscopic classification. It was performed during the first endoscopic examination, as it is an important predictor for strictures.[15] In our study, severe strictures were observed in half of the patients with Grade 2B injuries during the second control endoscopy, which shows that Grade 2B injury is likely to result in strictures. Temiz et al reported oesophageal stricture rates of 14.3%, 32.1%, and 100% in patients with Grade 2a, Grade 2b, and Grade 3 oesophageal injuries, respectively.[18]

The extent of injury is also related to other factors like amount, concentration, and duration of tissue contact with caustic substances.[19] A limitation of this study is that the amount and concentration of corrosive agents could not be analysed because it was retrospective. Moreover, induced-vomiting after ingestion of corrosive substances by family members was not found to be associated with severe oesophageal injury in our study.

Although some studies suggest that WBC count >20,000 cells/mm^3 and an increase in the C-reactive protein concentration and amylase level are predictive factors correlated with severe oesophageal injury, the correlation between the results of the laboratory tests and the oesophageal injury or stricture is not clear.[19-21] Notably, our lab results at first admission were not found to be associated with oesophageal injury.

The most important step in the management of caustic ingestion in paediatric patients is prevention. The families and community must be educated about using labelled, controlled household cleaners containing corrosives and storing them in safe places out of reach of children.[13]

Conclusion

Our data suggests that endoscopic evaluation is mandatory in patients who ingest alkaline agents, whereas it could be avoided in patients who ingest acidic agents, particularly the asymptomatic ones.

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Conflicts of Interests

The authors have no relevant financial or non-financial interests to disclose.

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