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DISEASES OF THE ESOPHAGUS

Original article

Pediatric caustic ingestion: 50 consecutive cases and a review of the literature

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SUMMARY. **Caustic injury to the aerodigestive tract remains a significant medical and social concern despite various efforts to minimize hazards of caustic household products. Agents with a pH less than two or greater than 12 are extremely corrosive, causing damage that can range from mild to extensive, including esophageal perforation leading to mediastinitis and death at the extreme scale. Methods include retrospective case note review of all admissions to the otolaryngology unit with caustic injury that underwent esophagoscopy to the Children's Hospital Westmead between 1990 and 2007. A protocol-based management system with antibiotics and steroids together with esophagoscopy at 48 hours was implemented. A total of 50 admissions were identified with an average follow-up of 5 years. There were a total of 28 males and 22 females with a median age of 22 months. Forty-nine cases (98%) were accidental. Thirty-eight cases (76%) occurred within the interiors of the family home with the kitchen being the common location. Another seven (14%) occurred within the external environment of the home, usually in the garage or pool shed. The causative agents were varied with 37 (74%) being alkali, three cases (6%) being acidic, and other agents, such as chlorine bleach, being the remainder. The most frequently ingested alkalis were dishwashing powder and disinfectants closely followed by degreasers. Twenty-five children (50%) drank directly from a container with the remainder ingesting granules or powder directly. At esophagoscopy, 17 cases (34%) had grade 1 injury and 10 (20%) had grade 2 injury. Fifty percent of patients of grade 2 injury subsequently developed strictures requiring multiple dilatations. Importantly, six cases (12%) had evidence of esophageal injury without oral injury. Caustic injuries continue to be a significant morbidity in the pediatric patient group. Most cases are still happening as a result of accidental ingestion from unmarked containers within reach of children at home. Oral injury is not always a useful marker of more significant distal injury. A protocol-based management can identify children at risk for long-term stricture earlier.**

KEY WORDS: **caustic, esophagoscopy, esophagus, pediatric, stricture.**

INTRODUCTION

Caustic ingestion continues to be a major health hazard in developed and developing countries despite continuing educational programs and legislation limiting the strength and availability of corrosive substances. At the end of the 19th century and beginning of the 20th century, lye products became commercially available for domestic use primarily as drain cleaners. Commonly available household products such as dishwashing liquids, window cleaning agents, and drain cleaners are highly corrosive agents if accidentally ingested. This increased availability is associated with an increasing number of accidental caustic ingestions in the pediatric population.[1] Noting that no warning labels were being used on these products, Chevalier Jackson began a public campaign against some opposition to institute proper labeling on these containers and the Federal Caustic Act of 1927 was enacted.

In Australia, there was a Sale and Use of Poisons Act in Tasmania and Queensland in 1891, but the regulations concerning proper labeling of potentially hazardous substances did not appear until 1964 with the introduction of the Poisons Act, subsequently followed by the Poisons and Therapeutic goods Act of 1966. Further, recent reforms in the New South Wales Poisons and Therapeutics Goods Act (1966) in 2002 has meant that regulations have become stricter, with the latest changes requiring proper labeling, antidote instructions, concentration restrictions, and child-resistant packaging.

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In urban societies, caustic ingestion occurs largely within a domestic environment, a fact corroborated by the available literature.[2] However, in rural areas, caustic soda in both crystal and liquid form is widespread for cleaning agricultural containers, fruit drying, and soap making.

The clinical spectrum of pediatric caustic ingestion can vary from no apparent injury to potentially fatal sequelae. Severe complications, such as esophageal perforations as well as strictures requiring multiple dilatations or esophageal replacement, have been described.[3] Despite the relative frequency of caustic ingestions, there are no clear guidelines regarding their management. This fact is further complicated by their admission under different specialty groups of otolaryngology, gastroenterology, and pediatric surgery.

In this report, we aim to summarize our experience of 50 consecutive cases of the management of caustic ingestion in a pediatric tertiary teaching hospital in Sydney, Australia.

METHODS

We conducted a retrospective case note review of the 50 consecutive cases presenting to the otolaryngology unit of the Children's Hospital Westmead between 1990 and 2007. Parameters analyzed were patients age at presentation, gender, nature of the caustic substance ingested, amount of substance, circumstances of injury whether accidental or intentional, anatomic location and distribution of injury, early and late complications, as well as the requirement for ventilatory support.

A protocol-based management, with the usage of steroids (dexamethasone 0.5 mg/kg/day), anti-reflux medications (weight-based omeprazole or pantoprazole daily), and antibiotics (third generation cephalosporins) as well as laryngoscopy and esophagoscopy at 48 hours after ingestion, was carried out. A classification system similar to accepted Estreta system was used to grade the severity of injury (Table 1).[4]

Table 1 Endoscopic grading of esophageal injury

Grade 0	No detectable mucosal change
Grade 1	Erythema of mucosa
Grade 2	Erythema, sloughing, ulceration and non-circumferential exudates
Grade 3	Deep mucosal ulceration and circumferential mucosal sloughing
Grade 4	Eschar, full thickness changes and perforation

RESULTS

A total of 50 admissions were analyzed with an average follow-up of 5 years. There were 28 boys and 22 girls with a median age of 20 months at presentation. All but one case was attributable to accidental ingestion. There was no mortality in the series. A total of 38 (76%) incidents happened within the home, eight (16%) occurred in the garage or pool shed, one at a grandparent's home, one at the hairdresser's salon, one was at a hospital clinic, and one more where the location was unclear.

The causative agents were varied and 37 (74%) of the agents were alkali, 3 (6%) cases of acid ingestion were recorded, and 10 (20%) cases involving other substances such as chlorine bleach comprised the remainder. The most frequently ingested alkalis were dishwashing powder, disinfectants and caustic soda used for cleaning ovens, and degreasers. Half of the children consumed liquids straight from a container, cup or bottle, while another 24 (48%) consumed caustic substances in powdered or granulated forms. There were six cases (12%) where cups or drink bottles were used for keeping decanted caustic solutions with the child innocently picking it up for consumption (Table 2).

At presentation to hospital, the salient clinical features noted were drooling (56%), vomiting (48%), as well as a refusal of further oral intake (76%). There was accompanying clinical signs of lip swelling (40%) and tongue erythema (20%), leukoplakia or oral ulceration (10%).

As soon as the diagnosis was established, the treatment protocol was commenced with the appropriate dosage of dexamethasone, a proton pump inhibitor, and a third generation cephalosporin. The patient then proceeded to esophagoscopy after 48 hours had elapsed since the ingestion. It was completed before 72 hours in all patients.

During endoscopy, 17 (34%) cases had mild mucosal changes involving the esophagus classified as grade 1 injury and 10 (20%) cases suffered more significant grade 2 esophageal injury. Of this latter group, five patients developed permanent damage requiring repeated dilatation. The presence of dysphagia at the follow-up clinic reviews was required for

Table 2 Patient characteristics, caustic agents, and mechanism of consumption

Total patients	50 (28 males; 22 females)
Median age (range)	22 months (6–48 months)
Ingested substance (%)	
Alkali	74
Acid	6
Chlorinated bleach	20
Form of agent (%)	
Granules	48
Liquid	50
Intention of ingestion (%)	
Accidental	98
Deliberate/inflicted harm	2

Table 3 Clinical outcomes of caustic ingestion

Mortality (%)	0
Esophageal injury (%)	
Grade 0	46
Grade 1	34
Grade 2	20
Grade 3	0
Grade 4	0
Airway injury (%)	18

the child to undergo repeat endoscopy. A decision was made not to perform a routine barium swallow unless endoscopy facilities were unavailable. All the patients who underwent stricture treatment were symptomatic by their 6-month review. An average of three dilatation procedures was required to achieve a satisfactory lumen. There was no injection of intralesional steroids or application of topical mitomycin C. Furthermore, six (12%) children had evidence of esophageal changes without clinically detected oropharyngeal signs. There were no long-term laryngeal sequelae in the case series. Nine cases (18%) had laryngeal trauma and required temporary intubation in the intensive care unit (Table 3).

There were no consistent social predicaments noted in the parents of the children with more significant injury.

All the patients were followed up at 3, 6, and 12 months in the otolaryngology unit and then once a year after that for up to 5 years. Close liaison was maintained with the general practitioners (GP) and provisions were in place for earlier review if clinically significant features were noted by the GP. This was, however, not required.

DISCUSSION

The biggest risk group for accidental caustic ingestion is children younger than 5 years and most groups agree that the incidence peaks at around 2 years when children develop skills of localization, but are poor discriminators between the harmless and harmful substances.[5] Despite all the new laws and precautions, it is still estimated that in the USA, 5000 accidental lye ingestions occur yearly in children under 5 years of age.[6] A variety of substances are responsible for caustic injuries ranging from alkalotic bases with pH up to 12, to acidic substances with pH as low as 2, as well as bleaching substances where the pH is around 7. High concentrations of alkali are found in lye-based (NaOH, KOH) drain cleaners, ammonia, as well as dishwashing soap. Detergent powders and hair-straightening formulas are especially hazardous because childproof packaging is not standard on these substances.

The overall management depends on accurate diagnosis. A careful history will detail the brand name, type, and amount of substance ingestion. As these ingestions are often unwitnessed, this portion of the workup can be difficult. Once the agent is known, the local poisons center can be contacted for assistance. It is also important to know whether vomiting occurred, as this can increase the length of time of esophageal exposure.

The symptoms of hoarseness, stridor, and dyspnea are noted for their possible harbinger of airway injury.[7] Odynophagia, drooling, and refusal of food suggest a more severe injury usually greater than grade 1.[8] Substernal chest pain, abdominal pain, and rigidity suggest profound injury and perforation of the esophagus or stomach. Although such clinical indicators may be useful, it has to be borne in mind that the patient's signs and symptoms are not always reliable predictors of the patient's ultimate injury.[9]

The factors other than pH of the offending agent that is responsible for the degree of damage to the pharyngoesophageal passage are the amount of substance ingested, the physical state of the agent, as well as the duration of exposure.[10] This is most manifest when crystalline drain cleaners adhere to natural points of constrictions of the esophagus and cause deep injury.[7] These anatomical constrictions, namely the cricopharyngeus, area of compression by the aorta and left main bronchus, and the lower esophageal sphincter, are at highest risk from caustic injuries and their subsequent complications.

The underlying pathophysiology of alkali and acid ingestion differ.[11] Alkalis cause mucosal liquefactive necrosis and the subsequent submucosal destruction allows for deeper penetration which may even penetrate through the muscularis propria layer. Because they are soluble once they form soaps with fat, there is an edematous loosening of the tissue with deep diffusion of the alkali into the tissues. Only neutralization of the substance by the tissue itself will cease the reaction.[6,12] This potential injury is often made worse by the innocuous taste of alkali substances which allow for ingestion of larger quantities. Acidic substances, which are rather sour in taste, tend to cause a coagulative necrosis of the mucosa and the resultant eschar formation tends to limit penetration and the subsequent injury. The degraded tissue is susceptible to bacterial superinfection.[13] After 48 hours, fibroblast proliferation and collagen synthesis begins with the weakness of the esophageal wall peaking between 1 and 3 weeks. The healing phase results in fibrosis and stricturing at around 4–6 weeks.[14]

Lateral neck and chest X-rays are performed within the initial assessment to corroborate any underlying suspicion of perforation. Barium swallows are of little use in the acute phase because it delays endoscopy and will not reveal first- or second-degree mucosal injuries.[6]

Esophagoscopy was carried out in every patient who was suspected of caustic ingestion. Prior to endotracheal intubation for a rigid esophagoscopy, an assessment of laryngotracheal injury was performed with a Hopkins rod telescope. In our series, we did not find any major airway injury necessitating prolonged intubation or a tracheotomy. Nonetheless, major airway injury requiring a tracheotomy has been described by other groups.[15]

We used esophagoscopy to grade the severity of the injury 48 hours after the inciting event. During this time period, the effects of the injury will have demarcated itself so that an appropriate grading of severity can be reliably predicted. Prior to this time period, one can underestimate the severity of injury as only erythema may be seen in the early phases of even grade 2 and 3 injuries. Endoscopy after 72 hours increases the chance of iatrogenic perforation as there will be potential structural weaknesses in the esophageal wall. A grading system similar to one described by Estreta was utilized to stage and, hence, prognosticate on the outcome. In summary, grade 1 injuries are superficial, grade 2 transmucosal, and grade 3 refers to transmural injuries.[4,11]

The usage of routine endoscopy allowed us to conclude that the patient's symptoms and clinical signs are not always a reliable predictor of the ultimate injury and hence the long-term outcome. As noted in our results, approximately 12% of patients without oral injury have concurrent esophageal injury, and up to 70% of oral injury can be without esophageal injury. Although routine endoscopy for assessment of injury after caustic ingestion is controversial and there are no clear guidelines, within the setting of a tertiary care institution such as ours, the availability of staff and resources meant that we had a complete adherence to the protocol of endoscopy after 48 hours but before 72 hours. The use of an esophagoscope also allowed us to insert a nasogastric tube under vision when we stopped at the point of the first sighted injury. It has also been recommended by other groups that esophagoscopy be terminated at the level of the most proximal circumferential burn. The nasogastric tube allows the continuation of enteral feeding during the period when the patient is kept without oral feeding and it also keeps the esophageal lumen patent so that adherence and obliteration of the lumen does not occur.[16] The nasogastric tube was removed at 1 week and normal oral feeding was commenced as tolerated by the child. The first follow-up after discharge was at 3 months.

Bleach ingestion is an exception to the rule of esophagoscopy for virtually all caustic ingestions. Bleaches are approximately 5–6% sodium hypochlorite and produce ulceration that usually does not result in stricture or permanent sequelae at these concentrations.[17] If no oropharyngeal burn is present, a barium swallow is obtained in 3–6 weeks. If oropharyngeal burns are present, then esophagoscopy should be done and management proceeds similarly to the other caustic agents.

The use of antibiotics, steroids, and anti-reflux therapy were the other arms of management. There is evidence suggesting a lower rate of stricture formation with the use of antibiotics. Proponents state that by decreasing bacterial counts in the necrotic tissue, granulation tissue superinfection is reduced which in turn will lessen the chance of stricture formation. Others argue that antibiotics promote the influx of gram negative organisms into the tissue without decreasing stricture formation rates and may even mask the signs of a more serious infection.[6,18] We routinely used third generation cephalosporin antibiotics for 48 hours and if oral intake was tolerated, changed over to clindamycin for 1 week to ensure a broad spectrum cover. No deleterious effects were noted in our series.

Induced emesis and gastric lavage are contraindicated to avoid further exposure of the esophagus. Neutralization agents such as vinegar are also contraindicated as it is thought that an exothermic reaction will occur, further injuring the tissue.[19]

The additive role of corticosteroids remains controversial. We know from multiple studies that steroids are able to modify the inflammatory response at the site of injury and within the deeper penetrated tissue with the ultimate theoretical goal of producing less extensive scarring. Nonetheless, multiple trials and reviews have shown little or no measurable benefit from varying doses of steroids in their ability to reduce the rate of stricture formation.[20–22] At high doses, one also has to be wary of significant, and often idiosyncratic, undesirable side effects of corticosteroids such as peptic ulceration, mycotic infection of the esophagus, and osteoporosis. Grade 2 injuries are where steroids are felt to be most beneficial in preventing stricture formation.[11] It seems that steroids may also be justified when there is a concern regarding upper airway edema and laryngeal injury.[23] In our series, dexamethasone at a dose of 0.5 mg/kg was utilized for 48 hours prior to the esophagoscopy and oral equivalents continued for 1 week afterwards. We did not note any adverse side effects from this.

The routine usage of anti-reflux therapy is recommended in order to prevent secondary reflux-associated esophageal injury which may act synergistically to further damage an already damaged area.[24,25] We utilized available proton pump inhibitors such as omeprazole, pantoprazole, and esomeprazole depending on availability within the hospital formulary for a total 6 weeks.

Despite these measures, stricture formation still occurred in 10% of the total esophageal caustic burns. This rate is slightly lower than other published groups where stricture rates have been as high as 50%. As

noted, half of the grade 2 esophageal burns (50%) had long-term strictures.

The management of esophageal strictures in children is endoluminal first and should that fail, then esophageal replacement surgery is necessary. Prograde dilation with Savary Gillard or Jackson dilators has been used for the greatest length of time. These are repeated every few weeks until a satisfactory caliber is achieved and the patient is swallowing appropriately. The optimal frequency of dilation is not well established in the literature and our practice was to use a symptom-based approach. Our data does corroborate with the available evidence that caustic strictures often needed more dilations to achieve a clinically satisfactory outcome compared with benign strictures from other etiologies.[26]

Retrograde dilation is felt to be safer by some and was originally described by Tucker.[27] In this method, a continuous loop of string is kept in the esophageal lumen and brought out of the nose superiorly, and a gastrostomy inferiorly. A Tucker dilator is tied to the lower end of the string and pushed and pulled out of the patient's mouth using progressively larger dilators. The presence of a string will allow dilation to be performed even without the need for an experienced endoscopist. The added advantage is the ability to use the gastrostomy for feeding. This can be done daily and in a fully conscious patient until it is safe to proceed with prograde dilation.[28]

Dilation can also be done with the use of a balloon dilation catheter under radiographic control. Similar to angioplasty, the balloon is passed to the area of stricture and then inflated. The advantage lies in that a radial direction of stricture dilation is performed with this method, which is thought to be less likely to result in a tear of the esophagus rather than the other methods which work through a longitudinal direction of dilation.[29] This was our preferred approach in the last 5 years of the study period. The available literature suggests that balloon dilation is not as successful as bougienage in caustic strictures but this has not been our experience. Our low complication rate, compared with perforation rates as high as 32% reported in the literature, could be attributed to a high degree of experience of the staff in using balloon catheter for other vascular, urological, or airway stenosis.[30]

When these methods fail, esophageal replacement is necessary. Colonic interpositions, jejunal interpositions, and gastric pull-ups are options.[31] The final consideration relates to the increased risk of esophageal carcinoma in patients with histories of caustic ingestion.[32,33] Several historical reports of lye ingestion-associated esophageal carcinoma note that the interval between the caustic injury and development of carcinoma may be as high as 45 years.[34] Squamous cell carcinoma of the esophagus, rather than adenocarcinoma, seems to be the eventual malignant transformation and the response to treatment whether surgical or radiotherapy is reportedly better than historical results obtained from the treatment of sporadic esophageal carcinoma.[35] It would seem appropriate that patients who undergo endoscopies as children for caustic ingestion be followed up in adulthood for surveillance for possibility of development of carcinoma. We are unable to find any valid evidence-based recommendations for surveillance, but based on the aforementioned reports, ten yearly surveillances would seem appropriate based on the natural history.

The lack of mortality and the concentration of grade 1 and 2 esophageal injuries in our series could be attributed to the cumulative effects of legislative changes reducing toxic concentrations of common household substances, as well as the prevalence of tamper proof packaging. It is also noteworthy that on the whole, increased public awareness and the aforementioned legislative changes have meant that in developed countries units may only treat a small number of cases each year.[36] An analysis of our results show only four to five cases per year presenting to our institution requiring hospital admission. This adds further weight to the argument of centralizing the management and follow-up these children within pediatric institutions. Some would argue that the cumulative morbidity from this is still too high. The actual number is likely to rise higher, given the fact that admissions often happen under different specialties and limitations of a retrospective study.

CONCLUSION

Accidental caustic ingestions continue to be a significant occurrence among our pediatric population. Most of the cases happen in the toddler age group between the ages of 12 months and 2 years. The most significant injuries seem to occur as a result of drinking opened containers within reach of our children. There are still too many cases which are related to storing caustic solutions in unlabelled containers or more seriously, routine drinking bottles which other adults unknowingly give to their children. The community needs to be reminded of these potential hazards.

The management of these cases should be standardized. All suspected ingestions should have a complete evaluation of the upper aerodigestive tract 48 hours following the presumed injury. This will allow sensible management procedures to be performed as necessary. It would be our recommendation that broad spectrum antibiotic coverage, steroids, and proton pump inhibitors be administered as adjunct treatments.

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