Caustic Injuries of the Esophagus

A. J. W. Millar, A. Numanoglu, and S. Cox

Contents

Introduction ................................................................. 2
Historical Note ............................................................ 2
Prevention ................................................................. 2
Epidemiology ............................................................... 3
Types of Ingested Substances ........................................... 3
Pathophysiology .......................................................... 4
Clinical Features ........................................................ 6
Initial Management ...................................................... 6
Long-Term Treatment .................................................. 10
Surgical Interventions .................................................... 12
Conclusion and Future Directions ....................................... 13
Cross-References ......................................................... 14
References ................................................................. 14

Abstract

Caustic ingestion continues to be a significant problem worldwide, especially in developing countries and particularly in the under 6 years age group. The presence or absence of symptoms or oral lesions does not reliably predict the existence or severity of esophageal lesions. Upper endoscopy remains the mainstay diagnostic modality for evaluation to define the extent and severity of the injury. The best predictor of morbidity and mortality is the extent of injury as assessed during the initial evaluation. Early management strategies for caustic ingestion are well defined. Controversy still surrounds the use of steroids, antibiotics, antacid therapy in the acute phase, and the use of esophageal stents and the frequency, timing, and method of dilatation in the prevention and management of esophageal strictures.

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There is a pressing need for noninvasive diagnostic modalities and effective therapeutic options to evaluate and treat the complications associated with caustic ingestion. Indications for definitive surgery or bypass and the type of procedure to use are also subject to ongoing debate. Novel therapies, including tissue engineering, are still in the animal study phase and may not be clinically applicable for years to come.

**Keywords**

Caustic ingestion · Esophagitis · Corrosive injury · Esophageal stricture · Pediatric

**Introduction**

A caustic agent (also known as corrosive) is a chemical substance capable of inducing injury on tissue contact. Accidental ingestion of caustic agents continues to be a significant problem worldwide, especially in developing countries and particularly in the under 6 years age group (Contini et al. 2009). In this chapter, we will provide a thorough review of the pathogenesis, clinical features, treatment, and long-term complications of caustic ingestion.

**Historical Note**

Caustic exposure became a significant problem at the end of the nineteenth and the start of the twentieth century, when lye (sodium hydroxide) products were introduced to the market as household cleaners (Leape et al. 1971). As the industrial chemical industries developed in the latter part of the nineteenth century, so did home access to a variety of chemicals, which had domestic use. The most common of these was caustic soda, which was used predominantly as a cleaning agent, in the manufacture of soap in the home and as a drain cleaner in concentrated 20% and even 40% strengths. In industrialized countries in the first half of the twentieth century, this resulted in a massive increase in childhood poisonings. Children’s hospital wards were frequently occupied by a small cohort of children with irreversibly damaged esophagi, requiring an extensive and innovative array of surgical procedures to overcome this terrible injury. The tragic consequences of ingesting caustic substances and the evolution of treatment methods have been well summarized by Tucker many years ago (Tucker et al. 1974). Esophageal dilatation of the resulting stricture, initially using blind bougie dilatation through the mouth, has changed little in principle but greatly in practice as a result of technologic advances. Development of the distally lighted esophagoscope, appropriate early airway management, the introduction of string-guided retrograde dilatation via gastrostomy, and improvements in general medical and nutritional support have nearly eliminated early mortality. Based on experimental evidence, the use of steroids and antibiotics became widespread in the 1950s and 1960s, in an attempt to reduce the incidence of stricture by inhibiting inflammation, scar formation, and infection. There has been some recent progress in managing the fibrotic healing process, the use of Mitomycin C being an example, and the increasing use of stents. However, mortality still occasionally occurs from pharyngeal and laryngeal burns resulting in edema and airway obstruction, large volume liquid ingestion with esophageal perforation, and complications after stricture dilatation or surgical bypass of an irreversibly damaged esophagus.

**Prevention**

With the problem of caustic ingestion having been identified, it was pediatricians and pediatric surgeons who acted as advocates on behalf of their patients, to lobby government to put legislation in place controlling the availability, sale, distribution, transport, packaging, and labeling of hazardous chemical substances. This, together with increased public awareness of the problem of caustic ingestion and activists’ efforts, resulted in the Federal Caustic Act in the USA in 1920, which required basic labeling of toxic substances, and the Poison Prevention Packing Act in 1970,
which mandated toxic material to be packaged in childproof containers and imposed restrictions on the concentration (<10%) of the liquid products. The creation of poison centers in 1953 was a great achievement as a valuable central source of information regarding products’ toxicities and treatment options (Elshabrawi and A-Kader 2011).

The UK developed regulations on packaging in the form of the Chemical Hazard Information and Packaging for Supply (CHIPS) regulations, an act of parliament governing sale and distribution of caustic substances. On the monitoring side, agencies were set up to document exposure incidents and investigate with a view to instigating preventive measures and, if necessary, prosecutions if regulations were transgressed. The Agency for Toxic Substances and Disease Registry (ATSDR), US Department of Health, is one such agency. Environmental assessment, substance specific intervention, links with primary care to educate families, and public health reporting, all operate with the aim of preventing a recurrence of such incidents.

Worldwide, similar agencies exist. Canada has the Canadian Hospitals Injury Reporting and Prevention Program (CHIRPP). Other examples include Kidsafe (Australia), Grosse schutzen Kleine (Austria), European Child Safety Alliance (Europe), Safe Kids (USA), and ChildSafe (South Africa), to name a few. The establishment of these child accident prevention agencies in the 1970s, usually linked to academic institutions and largely privately and charity funded, was able to provide data collection, such that the government could be approached to enact the appropriate supporting legislation. Successes in this area have been in the introduction of safety bottle tops for hazardous substances, as well as labeling and packaging and in many other areas of home safety.

These agencies have gone further with providing wide publicity in the media, reporting instances of ingestion and in developing educational packages for schools and workplaces. Safe houses have been built, which act as a “museum”-type resource for the community.

### Epidemiology

The ingestion of corrosive substances remains a major health hazard in children, despite the aggressive educational programs aimed at children and adults, preventive labeling and packaging, and legislation limiting the strength and availability of caustic substances mentioned above. In rural areas and in developing countries, caustic soda in both crystal and liquid form is used in home industries for soap making, fruit drying, and container cleaning on farms. In addition, the availability of innumerable over-the-counter caustic cleaning agents virtually ensures that children will continue to be at risk. Twenty to forty percent of ingestions of caustic substances result in some degree of esophageal injury.

The most distressing aspect is that the majority of ingestions occur in children younger than 3 years and are entirely preventable. Toxic ingestion in children older than 5 years is suspect, and ingestion in adolescents (where girls predominate) is usually intentional. In these cases, larger volumes and more potent corrosive and caustic materials tend to be used. Although mortality is rare, morbidity is often devastating and associated with lifelong consequences. Cases of alkali ingestion as a result of child abuse have also been reported (Dine and McGovern 1982). Comprehensive statistics, dating back to the 1970s, indicate a decrease in the incidence of severely caustic ingestions; however, in developing countries, the many reports of esophageal replacement procedures bear witness to this serious worldwide public health problem (Hamza et al. 2003). This high incidence is particularly evident in areas where corrosive substances are available in containers that are not childproof or where such substances have been decanted from larger containers for use in homes. There is still a great need for adult education and for legislation to be enforced.

### Types of Ingested Substances

Caustic agents can be broadly classified into strong bases and strong acids. Alkalis are bases that dissolve in water and include sodium,
Table 1 Common caustic substances ingested. (Originally published in Pediatric Surgery (editor A. G. Coran) 2012 © Elsevier)

| Caustic substance | Type | Commercially available form |
|-------------------|------|-----------------------------|
| Acids             | Sulfuric | Batteries, Industrial cleaning agents, Metal plating |
|                   | Oxalic | Paint thinners, strippers, Metal cleaners |
|                   | Hydrochloric | Solvents, Metal cleaners, Toilet and drain cleaners, Antirust compounds |
|                   | Phosphoric | Toilet cleaners |
| Alkali            | Sodium hydroxide | Drain cleaners |
|                   | Potassium hydroxide | Oven cleaners, Washing powders |
|                   | Sodium carbonate | Soap manufacturing, Fruit drying on farms |
| Ammonia           | Commercial ammonia | Household cleaners |
|                   | Ammonium hydroxide | |
| Ammonium hydroxide | Sodium hypochlorite | Household bleach |
| Detergents, bleach | Sodium polyphosphate | Household cleaners |
| Condy’s crystals   | Potassium permanganate | Disinfectants, hair dyes |

potassium, and ammonium hydroxide. Alkali burns are more frequent than acid burns due to the lower prevalence of strongly acidic products. Household bleaches, which produce weak acidic effects on tissue contact, may also result in erosion.

Powdered products are more likely to produce injury to the airway, pharynx, and upper esophagus due to increased contact time. Crystals or powders tend to affect mainly the supraglottic and oropharyngeal areas due to the limited quantities ingested. Liquid forms are associated with circumferential lesions as a result of complete contact with the surface of the lumen.

Non-phosphate compounds, such as dishwashing and laundry detergents, have become widely available recently due to concerns about the environment. Although these products have less titratable bases than sodium hydroxide and are therefore considered less dangerous, they contain silicate and carbonate that produce a high pH which can cause severe injury if ingested (Elshabrawi and A-Kader 2011). The most frequent causes of ingestions are common household cleaning products (Table 1). Some other unusual substances include:

(a) Hydrofluoric acid, which is produced by the reaction of calcium fluoride with sulfuric acid; initially a gas, it becomes liquid on cooling. The liquid is used as an industrial cleaning agent and is very corrosive. The effects of fluoride ions on cellular enzymes can also lead to systemic complications.

(b) Button batteries which contain high concentrations (up to 45%) of either sodium or potassium hydroxides. If the grommet seal is eroded, which typically occurs when lodged in an acid environment and contents leak out, a local injury may occur. They can also cause a localized electrical burn if charged.

(c) Glacial acetic acid, which is the concentrated form of acetic acid. It is commonly used in the preparation of Hindustan-Suriname food. Glacial acetic acid is also known to cause severe systemic complications such as hepatic and renal insufficiency, hemolysis, and disseminated intravascular coagulation (DIC) (Davids et al. 2001).

Pathophysiology

Much of what is known about the pathology of caustic injury in children has been derived from adult experience with self-inflicted injury and experimental studies in animals (Osman et al. 2008). Injury to mucosal surfaces occurs within seconds after contact with a strong acid or alkali. The nature of the injury caused by acidic and
alkaline substances differs considerably (Moore 1986). Acid ingestion leads to coagulation necrosis of the mucosa, hard eschar formation, and usually limitation of acid penetration through the mucosa (Ozcan et al. 2004). In contrast, alkali ingestion leads to tissue penetration with liquefactive necrosis, followed by destruction of the epithelium and submucosa and frequently extension into and sometimes through all muscle layers. Ischemia and thrombosis are dominant early processes. A friable discolored eschar develops, under which tissue destruction continues until the alkali is neutralized. The esophagus is damaged principally at the areas of holdup: the cricopharyngeal area, the mid-esophagus where it is crossed by the aortic arch and left main bronchus, and immediately above the esophagogastric junction (Haller et al. 1971). Spasm of the esophagus and disorganized motility occurs immediately after the caustic substance ingestion; these events may result in delayed emptying and even gastric regurgitation (Guelrud and Arocha 1980).

The strength (concentration), physical form of the ingested agent (solid, powder/crystal, liquid, or gel) impact on the degree of injury and site most severely affected. Crystals and powder form predominantly affect the cricopharyngeal area and upper esophagus. Also, solid form ingestion tends to have a longer local contact time which also adds to the degree of damage inflicted. The effects of duration of contact and concentration of the caustic substance on generation of injuries were reported in animal studies which have shown significant tissue damage occurring within seconds of ingestion of strong alkalis or acids (Mattos et al. 2006). The term “strength” refers to the ability of the alkali to dissociate in aqueous solution and is expressed by the equilibrium constant pKa, which is the pH at which the alkali is 50% dissociated to its conjugate acid. Strong alkalis are compounds that have a pKa value of greater than 14 or are capable of complete dissociation in water e.g. 20% sodium hydroxide has a pH of 13.8 but its pKa value when viewed as an acid is around 35 (Sivilotti and Ford 2000).

Hemorrhage, thrombosis, and marked inflammation with edema may be seen in the first 24 h after injury. Depending on the extent of chemical burn, inflammation may extend through the muscle layer until perforation occurs. After 48 h, there is evidence of thrombosis of submucosal vessels, which gives rise to local necrosis and gangrene. Bacterial contamination leads to the development of small intramural abscesses, which may extend to the mediastinum with full-thickness injury. After several days, necrotic tissue sloughs, edema decreases, and neovascularization begins. This early reparative or subacute phase is evident from the end of the first week through the second week after injury. Scar formation begins in the third week, when fibroblast proliferation replaces the submucosa and muscularis and stricture formation commences. Mucosal reepithelialization then commences and is usually complete by the sixth week. It is during this period that adhesions may form, narrowing or obliterating the esophageal lumen. The end result may be a fibrotic stricture and a shortened esophagus (Millar and Numanoglu 2012).

If the injury is transmural, necrosis may extend to the surrounding mediastinum leading to mediastinitis or if in an anterior direction may result in tracheaesophageal or even aorto-esophageal fistulas.

Steroids have been used to modify the inflammatory response both at the site of the burn and in the deeper tissues, with the ultimate goal of less extensive scarring. However, the extent of the initial injury largely determines the outcome of the healed injury. This can range from mucosal reepithelialization, with loss of esophageal glands and some submucosal fibrosis but preservation of the muscularis, to complete replacement of the esophageal wall by fibrous tissue.

Once the muscle of the esophagus has been destroyed, it cannot regenerate; at this point, maturation of the fibrous replacement with epithelialization of the luminal surface is the only “positive” outcome. Reduction of scar tissue formation by induced inhibition of intermolecular covalent bonding of collagen with lathyrogens and other anti-fibrotic and anti-inflammatory agents has been demonstrated experimentally, and recently Mitomycin C has been reported to be efficient when applied locally immediately.
after dilatation of the established stricture (Berger et al. 2012; El-Asmar et al. 2013; Ley et al. 2019; Ghobrial and Eskander 2018).

Esophageal dysmotility may persist for several weeks or may be permanent if muscle is replaced by fibrous tissue (Cadranel et al. 1990). As inflammation resolves, the contractile stage of healing begins; this may last for a few months and often results in stricture formation.

**Clinical Features**

Patients with caustic ingestion may be asymptomatic or may present with an array of systemic and local symptoms and signs including agitation and tachycardia, dyspnea, dysphagia, oral pain, drooling, odynophagia, and chest or abdominal pain. Drooling and inability to swallow indicate severe posterior pharyngeal or upper esophageal injury. Alkaline agents usually cause yellow-brownish lesions, while acidic agents may result in white-grayish ulcers.

The presence of hoarseness, stridor, and nostril flaring or rib retraction on inhalation suggests airway involvement (Goussard et al. 2019). Acute obstruction of the upper airway may result from posterior pharyngeal and laryngeal edema caused by spillage of the caustic agent into the upper airway. Concentrated ammonia fumes may be inhaled, causing nasopharyngeal edema and leading to respiratory injury (Einhorn et al. 1989). Airway symptoms may be seen immediately, or there may be a few hours delay especially with powdered agents. The airway may be sufficiently compromised to require emergency cricothyroidotomy or tracheostomy. The presence of fever, chest pain, peritonitis, or hypotension may indicate visceral perforation. Several studies have tried to correlate symptoms and clinical findings with the severity of esophageal injury with contradicting results (Gaudreault et al. 1983). The presence of drooling and dysphagia usually predicts the presence of aerodigestive tract injury. The presence of more than three symptoms or signs is associated with increased likelihood of esophageal injury. Other studies failed to establish a good correlation between symptomatology and the severity of the lesions. In a study which included 156 children with caustic ingestion, 96 (61.6%) showed no visible signs of contact with the caustic substance; however, in 36/96 (37.5%) of the patients, endoscopy revealed burns in one or more visceral sites (Previtera et al. 1990). Thus, in the absence of oral lesions, esophageal injury cannot be excluded with confidence.

Dysphagia is the most common symptom following caustic ingestion. However patients can present with dysphagia even in the absence of severe esophageal lesions. During the acute stage, dysphagia results from decreased motility and increased transit time and can persist for several weeks in patients following severe injury. After the acute stage, dysphagia develops secondary to fibrosis in the deep tissue which decreases the amplitude of esophageal peristalsis with or without stricture formation.

There is scarcity of data in the literature regarding gastric injury following caustic ingestion (Tekant et al. 2001). This may be explained by the termination of the endoscopic examination when encountering a significant esophageal lesion. Serious gastric lesions may occur following caustic alkali as well as acid ingestion and may result in perforation, hemorrhage, and death although gastric injury is usually more severe with acid ingestion such as sulfuric acid which may cause pyloric stenosis and gastric outlet obstruction (Maull et al. 1979).

There is no clear correlation between the results of full blood count and blood gas investigations and the outcome of caustic ingestion. A leucocyte count of >20,000, arterial pH less 7.22, and a base excess greater than −12 are indicative of severe esophageal injury and the need for emergency surgical intervention. Little correlation was found between leucocyte count or C-reactive protein and the severity of esophageal injury.

**Initial Management**

The initial assessment of patients with caustic ingestion should include a detailed history ascertaining the timing of the exposure and the amount, type, and brand of the ingested substance.
Asking the caregiver to bring the container can be helpful to provide this kind of information. Suicide and non-accidental injury need to be ruled out (Dine and McGovern 1982). The possibility of toxic effects of ingestion in addition to caustic injury should also be considered.

A careful physical examination focusing on assessment of the airway and hemodynamic stabilization should be performed. In some patients endotracheal intubation or surgical airway management may be urgently needed.

Vomiting should not be induced to avoid re-exposing the esophagus, pharynx, mouth, and larynx to the caustic material. In contrast to intoxicant ingestion, charcoal administration is not recommended as it does not adsorb to caustic agents, and in addition it may interfere with the endoscopic evaluation. Although animal studies have shown that neutralizing agents can be safely used to decrease the esophageal damage following caustic agents, this practice has not been proved safe in humans and is best avoided for fear of inducing a compounding injury secondary to an exothermic reaction.

Dilution therapy with water or milk has also been suggested, but there is no evidence to support this kind of therapy in humans, and it is best avoided as it may induce vomiting or perforation and similar to charcoal administration may interfere with the endoscopic evaluation.

Management of children suspected of having ingested caustic agents depends on several factors including the presence of symptoms and oral lesions in addition to the nature of the caustic material. Patients with a vague history and without symptoms or oral lesions should be observed for few hours and offered clear liquids and discharged home after instructing the family to seek medical advice if the child experiences any symptoms such as dysphagia.

A technetium labeled sucralfate scan is a useful and cost-effective screening method to exclude significant injury in these circumstances (Millar et al. 2001; Nondela et al. 2018). The sucralfate mixed with technetium-99m adheres to inflamed mucosa. The technetium labeled adherence of sucralfate is then recorded as radioactivity on a scan (Fig. 1). Patients without adherence do not have a significant injury and can be discharged without follow-up (Millar et al. 2001). Patients with symptoms or oral lesions should be admitted and started on intravenous fluids and be kept nil by mouth. Chest and abdominal radiographs are needed in order 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. Contrast esophagography is usually not needed during the initial assessment, unless perforation is suspected, as it is not a sensitive technique to detect partial thickness lesions. If a contrast study is done, a water-soluble contrast should be used because of the risk of perforation. Patients who swallowed the caustic material intentionally as a suicide attempt need special attention as they are more likely to have ingested large amounts compared to patients who ingested the caustic material accidentally.

Since it has been shown that the absence of symptoms and clinical findings does not exclude a serious injury, some authors have recommended that all patients undergo endoscopy (Lamireau et al. 2001). On the other hand, in a retrospective study, patients who unintentionally swallowed a corrosive substance and had no symptoms were unlikely to develop significant injury. It is also in this context that a technetium labeled sucralfate scan is a useful and accurate screening investigation to exclude serious injury (Betalli et al. 2008; Millar and Numanoglu 2012; Millar et al. 2001).

Fiber-optic endoscopy should definitely be done in the presence of any symptoms or oral lesions, when a technetium labeled sucralfate scan shows evidence of esophageal adherence and in those who ingested very strong material (an acidic material with pH <3 or alkaline material with pH >11). These patients need endoscopy even in the absence of any symptoms or signs. It has been stated that endoscopy is not needed in patients who have swallowed household bleach since it rarely causes significant injury; however, personal experience and several published studies dictate otherwise (Betalli et al. 2008; Lamireau et al. 2001; Previtera et al. 1990).

There are no controlled studies comparing the effectiveness and accuracy of early versus late endoscopy; however, most authors have advocated
performing endoscopy within the first 24–48 h following ingestion, after clinical stabilization (Zargar et al. 1991).

The flexible endoscope carries a low risk of perforation and can be safely performed up to 96 h following ingestion (Zargar et al. 1991). Endoscopy allows for inspection of the whole esophagus, stomach, and the duodenum. The only contraindication to endoscopy is clinical or radiological suspicion of perforation. Most physicians will stop the examination once a circumferential lesion is encountered, but distal lesions can be missed. Endoscopic examination should be performed exercising great caution, using gentle insufflation, and avoiding advancing the scope blindly or against resistance. The placement of a nasogastric tube under vision for feeding purposes, to maintain luminal patency and as a guide for later dilatations should be considered at the time of the endoscopy.

Grading the esophageal injury provides important information that helps determine the best therapeutic approach (Table 2) (Zargar et al. 1991). Patients with grade 0 or I are unlikely to have a complicated course or develop complications. The patients are usually observed for 12–24 h advancing oral intake from clear liquids to an age-appropriate diet before discharge. Patients with grade II injuries should be observed while clear liquids are given orally to an age-appropriate diet. A contrast swallow should be done 3 weeks after ingestion to rule out stricture formation.

The management of patients with higher-grade lesions represents a challenge to the treating physicians (Fig. 2). These patients may require intravenous antibiotics for 10–14 days. Stricture formation is a very likely complication, and therefore enteral nutrition is provided through a nasogastric tube inserted under vision during endoscopy (some patients may require gastrostomy or jejunostomy tubes).

A grading system based on ultrasonic images (Kamijo et al. 2004) concluded that endoscopic ultrasonographic (EUS) images consistent with
destruction of muscular layers, as opposed to only edema (grade I), may be associated with a higher incidence of stricture formation, although this has not been validated by other studies. Technetium-99m sucralfate swallowing during the first 24 h after caustic ingestion is able to detect esophageal injury and provides useful information regarding healing in repeated studies (Millar et al. 2001). Computer tomography or magnetic resonance imaging is sometimes needed where perforation or erosion into the adjacent mediastinal structures is suspected.

In most cases, oral feeding commences as soon as the patient is able to swallow saliva. If dysphagia occurs, an esophagogram, usually done about 2 weeks after the ingestion, can identify the extent of involvement. Concomitant use of antifungal agents, antacids, and acid-secreting inhibitors (H2-receptor blockers or proton-pump inhibitors) is widespread, but their efficacy has not been proved.

Corticosteroids can decrease inflammation, granulation tissue, and fibrous tissue formation. Therefore the use of corticosteroids has been suggested in the management of patients with caustic ingestion in order to prevent stricture formation. Studies have shown that the use of high-dose, short duration methylprednisolone in

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**Fig. 2** Stents currently used for benign esophageal strictures. (a) Niti-S covered stent (Taewoong Medical). (b) Polyflex stent (Boston Scientific Medical). (c) ELLA-BD stent (ELLA-CS) (Ham and Kim 2014)
patients with grade IIb burns can decrease esophageal injury and the need for dilatation of the esophagus without significant steroid-related morbidity (Usta et al. 2014; Cadranet et al. 1993). Other studies have suggested that the use of steroids can be harmful. Meta-analysis of several studies done over 15 years has concluded that steroid use does not decrease the incidence of stricture formation following caustic ingestion, and therefore the use of steroids was not advised (Anderson et al. 1990; Ulman and Mutaf 1998).

The routine use of antibiotics in patients with caustic ingestion is also controversial. Theoretically, antibiotics are indicated in grade III injuries on the basis of known pathophysiology of the development of microabscesses in the esophageal wall. Antibiotics are indicated to manage associated respiratory sepsis or if perforation is suspected. Prophylactic antibiotics may be indicated during dilatation procedures as cerebral abscesses have been reported following repeated esophageal dilatation (Angel et al. 1991). There is a good case for the use of an oral antifungal agent such as Mycostatin as prophylaxis against fungal infection of the eschar.

Early prophylactic dilatation can be effective for reducing time for stricture resolution (Tiryaki et al. 2005). Esophageal stenting with nasogastric or medical grade Silastic stents has been tried over many years (Atabek et al. 2007). With improved technology, the use of retrievable self-expanding covered metal or plastic stents is becoming more widely practiced (Fig. 3). Although controlled studies are not available, a prospective trial is due to start soon (Zhang et al. 2005). The concept of using a stent is to prevent contact of opposing sides in order to decrease adherence and subsequent stricture formation. Despite the fact that this approach has been shown to decrease the rate of stricture formation, it has yet to be accepted as routine clinical practice.

Esophageal rest in the initial phase has also been advocated. The principle behind esophageal rest is to allow reepithelialization and avoid irritation to healing tissue by food. Nutrition is provided by total parenteral nutrition or tube feeding (nasogastric, gastrostomy, or jejunostomy). However, in the absence of controlled trials, there is no evidence that this approach is beneficial, and most physicians recommend liquid diet as soon as the patient is able swallow. A compromise strategy has been proposed of esophageal rest for 10 days followed by introduction of oral feeds (Kikendall 1991).

**Long-Term Treatment**

During recovery, it is essential to provide adequate nutrition; in most cases, the gastrointestinal tract can be used, with access through a nasogastric tube or by placement of a feeding gastrostomy or jejunostomy tube. An upper gastrointestinal radiograph with oral contrast should be done if the patient develops dysphagia after few weeks. Dismotility of the esophagus may persist even without evidence of a stricture (Cadranet et al. 1990; Dantas and Mamede 1996).

If a stricture is demonstrated on contrast radiography, a program of dilatation is commenced. Various methods can be used, ranging from mercury filled bougies, flexible-graded bougie dilatation, guide-wire directed metal olives (Eder-Puestow system), to various balloon dilators. Balloon dilatation has some advantages (Alshammari et al. 2011; Uygun et al. 2013); this technique is considered safe and efficacious as it avoids the
shearing longitudinal force exercised by other
dilatation techniques but is less effective in
established fibrotic strictures. The gastrostomy
also provides an opportunity to ensure mainte-
nance of the nutritional status of the child during
the period of healing and stricture dilatation.

Dilatation should always be attempted with
great care. Initial passing of bougies for prograde
dilatation should never be done blindly. If there
are several strictures and visualization is difficult,
it is much safer to place a transesophageal string,
which is then used to guide the dilators either
retrograde through the gastrostomy or preferably
antergrade through the mouth to avoid dilatation
of the gastrostomy orifice. If the esophageal stricture
cannot be negotiated via the proximal esophagus,
passing a soft-tipped, flexible guide-wire into the
distal esophagus via a gastrostomy is usually pos-
sible (Millar et al. 1993). Easy access to the gas-
troesophageal orifice is gained by introducing a
polyvinyl chloride endotracheal tube into the
stomach via the gastrostomy and passing it up
the lesser curve of the stomach into the distal
esophagus or bypassing a guide-wire under vision
using a trans-gastric fiber-optic endoscope. For
satisfactory dilatation of a stricture, a general
anesthetic is required in the early stages to protect
the airway.

To be effective, dilatations should be done at
least once a week, commencing with catheters that
are one or two French sizes smaller than the esti-
mated diameter of the stricture. It is generally
prudent not to dilate more than two to three sizes
larger than the size of the first dilator meeting
resistance. Initially, dilatation should be continued
as long as esophageal healing and a progressive
increase in esophageal caliber are noted, along
with re-establishment of normal feeding.

Attempts at more frequent dilatations leaving a
balloon dilator in situ with daily inflation have
also been reported. Factors indicating a poor prog-
nosis in achieving a stable stricture are delay in
presentation, extensive grade III injury, ongoing
esophageal ulceration, a densely fibrotic stricture
that cracks on dilatation, a stricture longer than
5 cm, and inadequate lumen patency despite
repeated dilatations over a 9- to 12-month period
(Alshammari et al. 2011; Uygun et al. 2013).

Unfortunately up to 10% of patients will not
experience any meaningful improvement to
repeated endoscopic dilatations (Fig. 4). If dilata-
tion fails and a dense stricture develops, it requires
treatment. As with other benign esophageal stric-
tures, the incidence and severity of gastroesopha-
geal reflux must be investigated and excluded as a
contributing cause of the persisting stricture
(Mutaf et al. 1996). Gastroesophageal reflux
should be managed surgically by fundoplication,
if necessary, before definitive procedures to resect
a stricture or replace the esophagus are attempted.
Localized strictures may be resected with an end-
to-end anastomosis. However, the whole esopha-
gus must first be carefully assessed endoscopically
to confirm that the stricture is indeed
localized, because histologic evidence of fibrotic
injury may be much more extensive than is evi-
dent on radiography. A healthy color of the esopha-
geal mucosa and distensibility with air
insufflation at esophagoscopy are useful signs
when assessing the esophagus.

Fig. 4 Contrast esophagogram of a 23-month-old child, 6 months after extensive caustic injury due to battery acid ingestion. Initial grade 2b burns progressed to extensive stricturing despite weekly dilatations.
Local injection of steroids (1% triamcinolone acetate) into short strictures has had some success when combined with dilatation but has not been assessed prospectively (Berenson et al. 1994). Likewise, application of Mitomycin C (an inhibitor of fibroblast proliferation) has also been used with reported success (Berger et al. 2012). Intraluminal steroid or Mitomycin C may decrease the dilatations required for severe strictures, although long-term effects are unknown (Arnold and Numanoglu 2017; Ley et al. 2019).

A number of other substances have been effective in reducing the fibrotic response in experimental animals but have yet to reach established clinical practice. These include the use of antioxidants, halofuginone, and phosphatidylcholine, which inhibit collagen production, cytoprotective agents such as iloprost, anti-inflammatory hormones like glucagon-like polypeptide 2 (GLP-2), and sucralfate which may inhibit collagen deposition (Osman et al. 2008).

Some investigators advocate the use of esophageal stenting. The lumen is maintained, adhesion of de-epithelialized areas of the esophagus is prevented, and simultaneously tube feedings can be given. Over the years, various types of stents have been used (silicone, polytetrafluoroethylene, metal expanding, and biodegradable) (Best et al. 2009; Karakan et al. 2013; Vandenplas et al. 2009). If used, stents should remain in place for at least 6 weeks, at which time epithelial healing should be complete and fibrosis will have begun to mature. However, in many cases, these tubes are not well tolerated; they may promote gastroesophageal reflux, and if an extensive inflammatory response through the muscle occurs, the stent may need to be in place for much longer to be effective. Stents may cause erosion into the trachea or bronchus but have also been used in the management of esophageal fistulas resulting from caustic injury or dilatation therapy, mainly as a temporizing measure before surgical repair or esophageal bypass. Stents have the advantages of avoiding repeated sessions of dilatation, which are closely linked to increased physical and emotional burden to patients and caregivers. Other advantages claimed from the use of stents are longer-lasting dilatation effects, the ability to maintain luminal patency, and simultaneous stretching of the strictures in comparison with dilatation procedures.

Currently, three types of esophageal stents are in use: the self-expanding metal stent (SEMS), self-expanding plastic stent (SEPS), and a biodegradable (BD) stent. Stents can be manufactured to the specific size and length required. Problems with stents of all types have been tolerability; displacement; tissue hyperplasia, particularly at each end; and the need to remove the stent without complications. Covered stents show the most promise. Also drug-eluting stents have shown efficacy in animal models but have yet to be used in children in this clinical setting (Ham and Kim 2014).

### Surgical Interventions

Unfortunately, in some patients surgical intervention is needed to maintain an airway, where extensive necrosis is noted on endoscopy and in patients with evidence of perforation. Exploratory thoracotomy and/or laparotomy may be needed if there is strong suspicion of full-thickness injury with widespread necrosis. Esophagectomy, esophagogastroctomy, or gastrectomy may be indicated if necrosis is confirmed.

Caustic injury may heal without stricture or may respond to the various prophylactic and therapeutic measures outlined. However, residual motility dysfunction can be expected, and an achalasia-like picture has been described.

Surgical intervention is also warranted in patients with tight strictures which are associated with failed dilatation or stenting. Indicators of likely failure of dilatation therapy are delay in presentation, severe laryngeal and airway compromise at first presentation, a stricture greater than 5 cm in length, and prolonged dilatation without progress (Panieri et al. 1998).

Both adenocarcinoma and squamous cell carcinoma with an incidence of 2–8% of the previously injured esophagus are a real risk, but the disease usually has a latency period of 15–40 years (Appelqvist and Salmo 1980; Arnold and Numanoglu 2017). However a lethal
squamous cell carcinoma of the esophagus has been reported just 1 year after injury. The incidence is 1000 times the expected in normal population of similar ages. Carcinomas usually develop at the strictured site or the area of bifurcation of trachea and are often resectable. However they usually carry a poor prognosis (40% survival rate at 1 year and 13% at 5 years) (Isolauri and Markkula 1989). There is controversy regarding the need for periodic surveillance for the development of dysplasia and carcinoma following caustic ingestion, and periodic endoscopy should be considered in patients who are 20 years or more after the initial ingestion (Isolauri and Markkula 1989). Barrett’s esophagus has been observed following lye-induced injury due to constant acid gastroesophageal reflux. Thus, long-term surveillance with esophagoscopy is advocated. In this regard, two prudent questions arise: To what extent should the clinician try to preserve the damaged esophagus? When should attempts at dilatation be abandoned? Currently, there is a trend toward earlier esophageal bypass in a severely injured esophagus, with the addition of resection of the damaged esophagus (Hamza et al. 2003). Although complications such as abscess or cyst formation in the bypassed but retained esophagus where a retrosternal esophageal graft has been done do occasionally occur, this is usually because attempts at dilatation have been abandoned early with active granulation still present in the retained esophagus. In this situation removal of the damaged esophagus and posterior mediastinal siting of the esophageal replacement graft (colon or stomach) is preferred.

Esophageal perforation occurring following dilatation, as evidenced by pain, fever, and tachycardia, is a life-threatening iatrogenic complication. With immediate recognition by endoscopy or contrast swallow, many patients with a perforated esophagus can be treated conservatively with systemic antibiotics and parenteral nutrition. Established methods of management with either thoracostomy drainage or primary repair with proximal and distal esophageal and gastric diversion are reserved for patients with delayed recognition, extensive disruption, or with the development of a pleural effusion (van der Zee et al. 1988).

If dilatation has failed or if the esophagus cannot be salvaged, esophageal bypass or substitution is indicated. Operations currently used are colonic interposition, gastric tube esophagoplasty, jejunal interposition, colonic patch esophagoplasty, and gastric advancement (Bax and van der Zee 2007; Hamza et al. 2003; Othersen et al. 1997; Spitz and Lakhou 1993; Bradshaw et al. 2018). These procedures have also been used for less extensive but persistent strictures. Deciding which procedure to use and whether to bypass or resect the injured esophagus is influenced by local practice and the morbidity and mortality from esophageal resection. Clearly, the risks associated with resection of the esophagus must be less than the risk associated with the described complications of the retained but bypassed esophagus (Imre and Kopp 1972). Thoracoscopic and laparoscopic techniques have also been described (Nwomeh et al. 2004). Where the injury extends into the hypopharynx, there are advantages to using the colon as sufficient length and good vascularity of the graft is usually secure and there is no acid secretion from the graft. Jejunal grafts have also rarely been used in children but with a high risk of complications (Cauchi et al. 2007).

Pyloric stenosis and gastric outlet obstruction may follow both alkalis and acid ingestions (Tekant et al. 2001). Although balloon dilatation has been used successfully in a child with caustic ingestion and pyloric stenosis, surgical bypass may be necessary (Treem et al. 1987). Y-V advancement antpyloroplasty has been described as a corrective surgery for corrosive antral strictures (Brown et al. 2002).

Conclusion and Future Directions

The continued unacceptably high incidence of caustic ingestion highlights the need for prevention and adult education programs. Caustic ingestion injury to the mouth, pharynx, larynx, esophagus, and stomach may be severe and frequently results in permanent scar formation and stricturing. Despite advances in the initial management of the acute case, there has been little impact in preventing the inevitable caustic
striction, which develops in 10–20% of patients. The high rate of long-term complications following esophageal replacement stresses the need for multidisciplinary lifelong follow-up.

Research into future more satisfactory management alternatives continues. Studies on the use of antioxidants, agents which inhibit collagen production, cytoprotective agents, and anti-inflammatory hormones may progress from animal to human studies, and in the future these may show some promise as effective treatments preventing stricture formation. Tissue engineering techniques may progress to the development of implantable scaffolds for esophageal replacement, but currently the development of a functional muscle component remains a challenge (Maghsoudlou et al. 2014). In time, a bioengineered neo-esophagus may represent an effective treatment. Until such treatments are shown to be effective, the various management strategies discussed in this text will continue to be practiced.

Cross-References

> Esophageal Replacement
> Gastroesophageal Reflux and Hiatus Hernia
> Tissue Engineering and Stem Cell Research

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