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REVIEW ARTICLE

Caustic injury of the oesophagus

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Abstract Caustic ingestion continues to be a significant problem world-wide 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 oesophageal 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 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 oesophageal stents and the frequency, timing and method of dilatation in the prevention and management of oesophageal strictures. There is a pressing need for non-invasive 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.

Keywords Caustic ingestion · Esophagitis · Corrosive injury

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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 world-wide especially in developing countries and particularly in the under 6 years age group [1]. In this manuscript, 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 [2]. 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 % strength. In industrialised 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 oesophagi, 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 summarised by Tucker many years ago [3]. Oesophageal 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 oesophagoscope, 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 oedema and airway obstruction, large volume liquid ingestion with oesophageal perforation, and complications after stricture dilatation or surgical bypass of an irreversibly damaged oesophagus.

Prevention

The problem of caustic ingestion having been identified, it was paediatricians and paediatric 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 labelling of hazardous chemical substances. This, together with increased public awareness of the problem of caustic ingestion and activists' efforts resulted in the Federal Caustic Actin the USA in 1920 which required basic labelling of toxic substances and the Poison Prevention Packing Act in 1970, which mandated toxic material to be packaged in child-proof containers and imposed restrictions on the concentration (<10 %) of the liquid products. The creation of poison centres in 1953 was a great achievement as a valuable central source of information regarding products' toxicities and treatment options [4].

The United Kingdom 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 have been set up which document exposure incidents and investigate with a view to instigating preventive measures, and if necessary, prosecutions if regulations have been 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.

World-wide, similar agencies exist. Canada has the Canadian Hospitals Injury Reporting and Prevention Program (CHIRPP). Other examples include Australia (Kidsafe), Austria (Grosse Schuetzen Kleine), Europe (European Child Safety Alliance), USA (Safekids) and South Africa (Childsafe) 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 were able to provide data collection, such that 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 labelling 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 programmes aimed at both children and adults, preventive labelling 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 industry 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 results in some degree of oesophageal 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 [5].

Comprehensive statistics dating back to the 1970s indicate a decrease in the incidence of severely caustic ingestions; however, in developing countries, many reports of oesophageal replacement procedures bear witness to this serious world-wide public health problem [6]. In a recent review from the eastern mediterranean area, 71 studies were included from 12 countries. Burn injuries were found

to be one of the leading causes of injury morbidity and mortality. The reported incidence of burns ranged from 112 to 518 per 100,000 per year [7]. 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, 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 oesophagus due to increased contact time. Crystals or powders tend to affect mainly the supra-glottic 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 common 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 [4]. The most common causes of ingestions are common household cleaning products (Table 1).

Table 1 Common caustic substances ingested [15]

Caustic substance	Type	Commercially available form
Acids	Sulphuric	Batteries
		Industrial cleaning agents
		Metal plating
	Oxalic	Paint thinners, strippers
		Metal cleaners
	Hydrochloric	Solvents
		Metal cleaners
		Toilet & 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	
Detergents, bleach	Sodium hypochlorite	Household bleach
	Sodium polyphosphate	Household cleaners
Condy's crystals	Potassium permanganate	Disinfectants, hair dyes

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. 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 [8]. Acid ingestion leads to coagulation necrosis of the mucosa, hard eschar formation and usually limitation of acid penetration through the mucosa [9, 10]. 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. Ischaemia and thrombosis are dominant early processes. A friable discoloured eschar develops, under which tissue destruction continues until the alkali is neutralised. The oesophagus is damaged principally at the areas of holdup; the cricopharyngeal area, the mid-oesophagus where it is crossed by the aortic arch and left main bronchus, and immediately above the oesophago-gastric junction [11, 12]. Spasm of the oesophagus and disorganised motility occurs immediately after the caustic substance ingestion; these events may result in delayed emptying and even gastric regurgitation [13].

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 [14]. Haemorrhage, thrombosis and marked inflammation with oedema may be seen in the first 24 h after injury. Depending on the extent of burn, inflammation may extend through the muscle layer until perforation occurs. After 48 h, there is evidence of thrombosis of sub-mucosal 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, oedema

decreases and neovascularization begins. This early reparative or sub-acute 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 re-epithelialization then commences and is usually complete by the sixth week. It is during this period that adhesions may form, narrowing or obliterating the oesophageal lumen. The end result may be a fibrotic stricture and a shortened oesophagus [15].

If the injury is trans-mural, necrosis may extend to the surrounding mediastinum leading to mediastinitis, or if in an anterior direction may result in trachea-oesophageal or even aorto-oesophageal 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 re-epithelialization, with loss of oesophageal glands and some sub-mucosal fibrosis, but preservation of the muscularis, to complete replacement of the oesophageal wall by fibrous tissue.

Once the muscle of the oesophagus 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 [16, 17] has been demonstrated experimentally and recently Mitomycin-C has been reported to be efficient when applied locally immediately after dilatation of the established stricture [18, 19].

Oesophageal dysmotility may persist for several weeks or may be permanent if muscle is replaced by fibrous tissue [20]. 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 symptoms including agitation and tachycardia, dyspnoea, dysphagia, oral pain, drooling, odynophagia and chest or abdominal pain. Drooling and inability to swallow indicate severe posterior pharyngeal or upper oesophageal injury. Alkaline agents usually cause yellow-brownish lesions, whilst acidic agents may result in white-greyish ulcers.

The presence of hoarseness, stridor, nasal flaring or rib retraction on inhalation suggests airway involvement. Acute obstruction of the upper airway may result from posterior pharyngeal and laryngeal oedema caused by spillage of the caustic agent into the upper airway. Concentrated ammonia fumes may be inhaled, causing nasopharyngeal oedema and leading to respiratory injury [21]. 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 oesophageal injury with contradicting results [22, 23]. The presence of drooling and dysphagia usually predicts the presence of oesophageal injury. The presence of more than three symptoms or signs is associated with increased likelihood of oesophageal 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 %), patients endoscopy revealed burns in one or more visceral sites [24]. Thus, in the absence of oral lesions oesophageal 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 oesophageal lesions. During the acute stage, dysphagia results from decreased motility and increased transit time and can persist for few weeks in patients following severe injury. After the acute stage, dysphagia develops secondary to fibrosis in the deep tissue which decreases the amplitude of oesophageal peristalsis with or without stricture formation.

There is scarcity of data in the literature regarding gastric injury following caustic ingestion [25]. This may be explained by the termination of the endoscopic examination when encountering a significant oesophageal lesion. Serious gastric lesions may occur following caustic alkali as well as acid ingestion and may result in perforation, haemorrhage and death although gastric injury is usually more severe with acid ingestion such as sulphuric acid which may cause pyloric stenosis and gastric outlet obstruction.

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 is indicative of severe oesophageal injury and the need for emergency surgical intervention. Little correlation was found between leucocyte count or C-reactive protein and the severity of oesophageal injury [26].

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 [5]. 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 stabilisation 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 oesophagus, 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 neutralising agents can be safely used to decrease the oesophageal 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 exothermic reaction [27].

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-labelled sucralfate scan is a useful and cost-effective screening method to exclude significant injury in these circumstances [28]. The sucralfate adheres to inflamed mucosa which is recorded on a scan (Fig. 1). Patients without any significant adherence do not have a significant injury and can be discharged without follow-up [28]. 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 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 oesophagography 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 [29, 30]. On the other hand, in a retrospective study, patients who unintentionally swallowed a corrosive and had no symptoms were unlikely to develop significant injury. It is also in this context that a technetium-labelled sucralfate scan is a useful and accurate screening investigation to exclude serious injury [15, 28, 31].

Fibre optic endoscopy should definitely be done in the presence of any symptoms or oral lesions, when a technetium-labelled sucralfate scan shows evidence of oesophageal 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 having swallowed household bleach since it rarely causes significant injury; however, personal experience dictates otherwise [24, 30–32].

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 stabilisation [33].

The flexible endoscope carries a low risk of perforation and can be safely performed up to 96 h following ingestion [33]. Endoscopy allows for inspection of the whole oesophagus, 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 oesophageal injury provides important information that helps to determine the best therapeutic approach (Table 2) [33]. 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 whilst clear liquids are given orally to

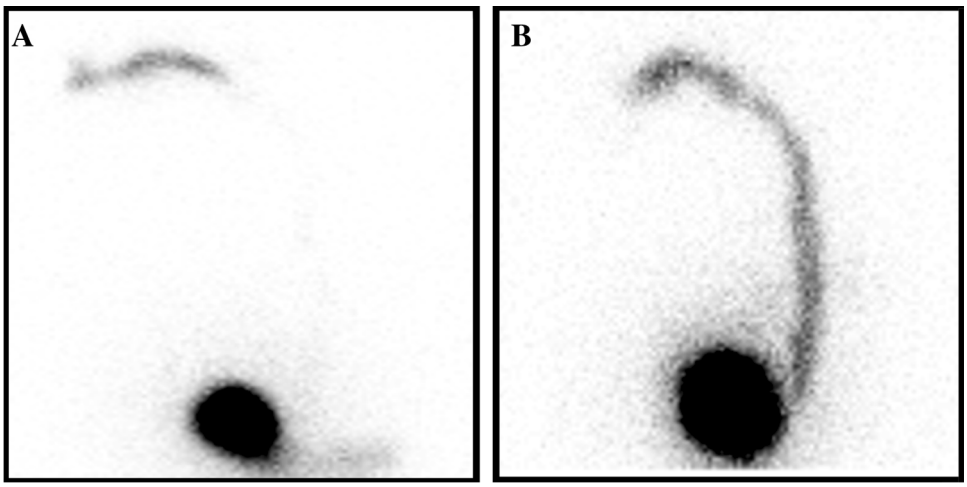

Fig. 1 Technetium-99 m-labelled isotope scan. **a** Normal scan showing oral and gastric activity. **b** Abnormal adhesion of Sucralfate to the entire oesophagus. Residual buccal and gastric activity is noted. The findings are in keeping with a caustic injury to the entire oesophagus

Table 2 Endoscopic grading of injury severity

Grade	Characteristics
Grade 0	Normal oesophagus
Grade 1	Mucosal oedema and hyperemia
Grade II a	Friability, erosions, haemorrhage, blisters, exudates, whitish membrane and shallow ulcers
Grade II b	Grade II lesions in addition to deep or circumferential lesions
Grade III a	Small or scattered areas of necrosis
Grade III b	Extensive necrosis

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 the nasogastric tube inserted under vision during endoscopy (some patients may require gastrostomy or jejunostomy tubes).

A grading system based on ultrasonic images [34] concluded that endoscopic ultrasonographic (EUS) images consistent with destruction of muscular layers, as opposed to only oedema (grade I), may be associated with a higher incidence of stricture formation, although this has not been validated by other studies [35]. Technetium 99 m sucralfate swallowing during the first 24 h after caustic ingestion is able to detect oesophageal injury and provides useful information regarding healing in repeated studies [28]. 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 oesophagogram, 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.

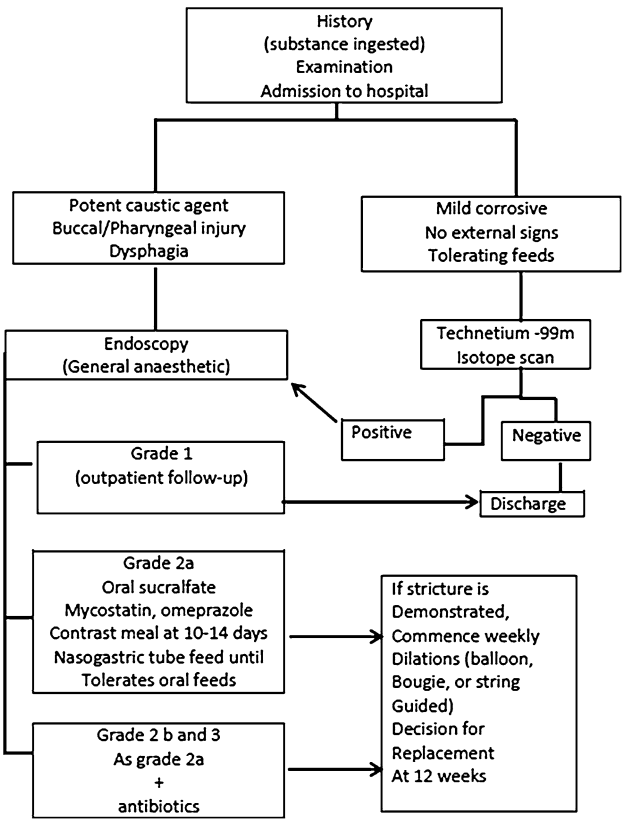

Fig. 2 Management protocol for caustic strictures of the oesophagus [15]

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 to prevent stricture formation. Studies have shown that the use of high dose, short-duration methylprednisolone in patients with grade II b burns can decrease oesophageal injury and the need for dilatation of the oesophagus without significant steroid-related morbidity [36, 37]. Other studies have suggested that the use of steroids can be harmful [38]. 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 [39–41].

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 micro-abscesses in the oesophageal 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 oesophageal dilatation [42]. 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 [43]. Oesophageal stenting with nasogastric or medical grade silastic stents has been tried over many years [44]. With improved technology, the use of retrievable self-expanding covered metal or plastic stents is becoming more widely practised (Fig. 3). Although controlled studies are not available, a prospective trial is due to start soon [45]. The concept of using a stent is to prevent contact of opposing sides 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.

Oesophageal rest in the initial phase has also has been advocated. The principle behind oesophageal rest is to allow re-epithelialization 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 to swallow. A compromise strategy has been proposed of oesophageal rest for 10 days followed by introduction of oral feeds [46].

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. Dysmotility of the oesophagus may persist even without evidence of a stricture [20, 47, 48].

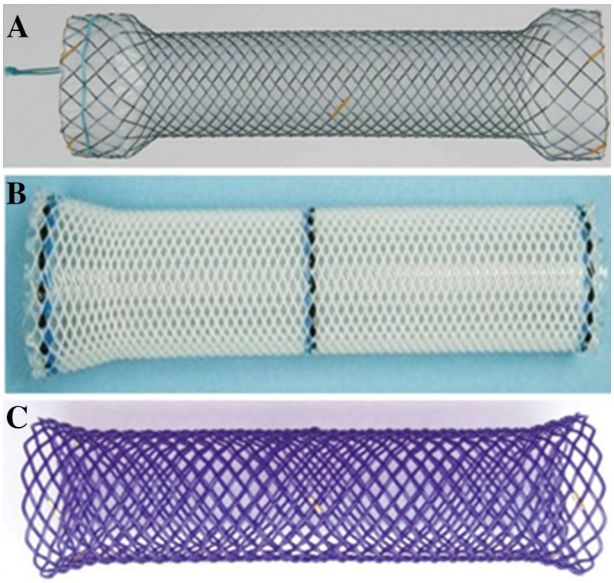

Fig. 3 Stents currently used for benign oesophageal strictures. **a** Niti-S covered stent (Taewoong Medical). **b** Polyflex stent (Boston Scientific). **c** ELLA-BD stent (ELLA-CS) [61]

If a stricture is demonstrated on contrast radiography, a programme of dilatation is commenced. Various methods can be used, ranging from mercury-filled bougies, flexible-graded bougie dilatation, guidewire-directed metal olives (Eder–Puestow system), or various balloon dilators. Balloon dilatation has some advantages [49–52], 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.

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 visualisation is difficult, it is much safer to place a trans-oesophageal string, which is then used to guide the dilators either retrograde through the gastrostomy or preferably ante-grade through the mouth to avoid dilatation of the gastrostomy orifice. If the oesophageal stricture cannot be negotiated via the proximal oesophagus, passing a soft-tipped, flexible guidewire into the distal oesophagus via a gastrostomy is usually possible [53, 54]. Easy access to the gastro-oesophageal 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 oesophagus or bypassing a

guidewire under vision using a trans-gastric fibre optic endoscope. For satisfactory dilatation of a stricture, a general anaesthetic 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 estimated 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 oesophageal healing and a progressive increase in oesophageal calibre 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 prognosis in achieving a stable stricture are delay in presentation, extensive grade III injury, ongoing oesophageal 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 [50–52].

Unfortunately up to 10 % of patients will not experience any meaningful improvement to repeated endoscopic dilatations (Fig. 4). If dilatation fails and a dense stricture develops, it requires treatment. As with other benign oesophageal strictures, the incidence and severity of gastro-oesophageal reflux must be investigated and excluded as a contributing cause of the persisting stricture [55]. Gastro-oesophageal reflux should be managed surgically by fundoplication, if necessary, before definitive procedures to resect a stricture or replace the oesophagus are attempted. Localised strictures may be resected with an end-to-end anastomosis. However, the whole oesophagus must first be carefully assessed endoscopically to confirm that the stricture is indeed localised, because histologic evidence of fibrotic injury may be much more extensive than is evident on radiography. A healthy colour of the oesophageal mucosa and distensibility with air insufflation at oesophagoscopy are useful signs when assessing the oesophagus.

Local injection of steroids (1 % triamcinolone acetate) into short strictures has had some success when combined with dilatation, but has not been assessed prospectively [56]. Likewise, application of Mitomycin-C (an inhibitor of fibroblast proliferation) has also been used with reported success.

Some investigators advocate the use of oesophageal stenting [57]. The lumen is maintained, adhesion of de-epithelialised areas of the oesophagus 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) [57–60]. 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 gastro-oesophageal reflux, and if an extensive inflammatory response through the muscle occurs, the stent must 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 oesophageal fistulas resulting from caustic injury or dilatation therapy, mainly as a temporising measure before surgical repair or oesophageal bypass. Stents have the advantages of avoiding repeated sessions of dilatation, which are closely linked to increased physical and emotional burden to patients and care givers. Advantages claimed from the use of stents are longer lasting dilatation effects, ability to maintain luminal patency, and simultaneous stretching of the strictures in comparison with dilatation procedures. Currently, three types of oesophageal stents are in use: the self-expanding metal stent (SEMS), self-expanding plastic stent (SEPS), and biodegradable (BD) stent. Stents can be manufactured to 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 [61–63].

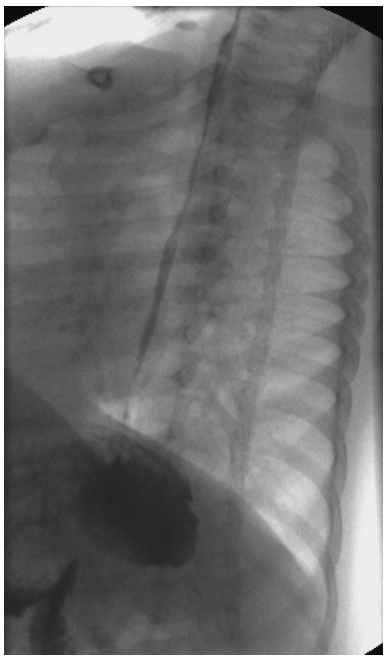

Fig. 4 Contrast oesophagogram of a 23-month-old child, 6 months after extensive caustic injury due to battery acid ingestion. Initial grade II b burns progressed to extensive stricturing despite weekly dilatations

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. Oesophagectomy, oesophago-gastrectomy 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 failure are delay in presentation, severe laryngeal and airway compromise at first presentation and prolonged dilatation without progress [64].

Both adenocarcinoma and squamous cell carcinoma, with an incidence of 2–8 % of the previously injured oesophagus are a real risk, but the disease usually has a latency period of 15–40 years [65–68]. However, a lethal squamous cell carcinoma of the oesophagus has been reported just 1 year after injury. The incidence is 1,000 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) [69]. 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 [69]. Barrett's oesophagus has been observed following lye-induced injury due to constant acid gastro-oesophageal reflux. Thus, long-term surveillance with oesophagoscopy is advocated. In this regard, two prudent questions arise: To what extent should the clinician try to preserve the damaged oesophagus? When should attempts at dilatation be abandoned? Currently, there is a trend toward earlier oesophageal bypass in a severely injured oesophagus, with the addition of resection of the damaged oesophagus [6]. Although complications such as abscess or cyst formation in the bypassed but retained oesophagus, where a retrosternal oesophageal graft has been done, occasionally occurs, this is usually because attempts at dilatation have been abandoned early with active granulation still present in the retained oesophagus. In this situation, removal of the damaged oesophagus and posterior mediastinal placement of the oesophageal replacement graft (colon or stomach) is preferred.

Esophageal perforation, as evidenced by pain, fever and tachycardia, is a life-threatening iatrogenic complication of oesophageal dilatation. With immediate recognition by endoscopy or contrast swallow, many patients with a perforated oesophagus 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 oesophageal and gastric diversion are reserved for patients with delayed recognition or extensive disruption [70].

If dilatation has failed or if the oesophagus cannot be salvaged, oesophageal bypass or substitution is indicated. Operations currently used are colonic interposition, gastric tube oesophagoplasty, jejunal interposition colonic patch oesophagoplasty and gastric advancement [6, 71–74]. 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 oesophagus is influenced by local practice and the morbidity and mortality from oesophageal resection. Clearly, the risks associated with resection of the oesophagus must be less than the risk associated with the described complications of the retained, but bypassed oesophagus [75]. Thoracoscopic and laparoscopic techniques have been described [76].

Pyloric stenosis and gastric outlet obstruction may follow both alkalis and acid ingestions [25]. Although balloon dilatation has been used successfully in a child with caustic ingestion and pyloric stenosis surgical bypass may be necessary [77]. Y–V advancement antropyloroplasty has been described as a corrective surgery for corrosive antral strictures [78].

Conclusion

The continued unacceptably high incidence of caustic ingestion highlights the need for prevention and adult education programmes. Caustic ingestion injury to mouth, pharynx, larynx, oesophagus and stomach may be severe and frequently results in permanent scan formation and stricturing. Despite advances in the initial management of the acute case, there has been little impact in preventing the inevitable caustic stricture, which occurs in 10–20 % of patients. The high rate of long-term complications following oesophageal replacement stresses the need for multidisciplinary lifelong follow-up, and research into future more satisfactory management alternatives.

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