

Caustic ingestion in children

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Accidental ingestion of caustic agents may cause devastating injury in children. Strong alkalis are present in a wide range of household and industrial cleaners and beauty products, often attractively packaged and easily accessible to children, both at home and on low shelves in shops. Even in small quantities, ingestion of such substances can result in profoundly disabling morbidity and even mortality. Rapid, comprehensive management of the acute injury is required to minimize these complications. Anaesthetists may encounter such patients both at first presentation and subsequently for management of sequelae. Alkali ingestion is increasingly rare in the UK and is usually accidental in children. In teenagers and adults, alkali poisoning is more likely to occur as a form of deliberate self-harm and is associated with a higher mortality. In developing countries, cheap and widely available corrosive substances are frequently used for suicidal purposes.

Friedman investigated risk factors for alkali ingestion in children.¹ Household stress (such as marital conflict, mental or physical illness or loss of a family member) was identified as the leading risk factor. Children with accidental caustic ingestions were no more likely to live in a 'unsafe home' with regard to accessibility of toxic agents to children, storage, or the degree of parental supervision, than those in control homes.

Aetiology and pathology

The most common agents implicated in caustic ingestion are cleaning and dishwasher products and industrial paint strippers (Table 1). Alkalis

are also contained in hair straighteners and relaxers, which may be freely accessible to children in the bathroom environment.

Injury patterns for alkali burns differ from those of acid burns. Acids cause coagulative necrosis, which results in a self-limiting burn pattern, while alkalis induce liquefactive necrosis with saponification of fats and solubilization of proteins. These are hygroscopic and absorb water from tissues, resulting in deeper tissue penetration and more extensive burns. Because of increased tissue adherence, alkali also causes more damage to the oesophagus, while acid ingestion tends to result in more severe gastric injury. However, deliberate ingestion of large quantities of alkali may injure the stomach and even the small intestine. Gastric injury is more likely to follow ingestion of a liquid than solid alkali.

The severity of injuries depends on multiple factors, such as the concentration of the agent, volume ingested, duration of contact with mucosal surfaces and pH of the solution (damage is greatest when the pH is >12). Solid preparations and viscous liquids produce more severe injury owing to a longer contact time with the oral and oesophageal mucosa.

Types of injuries

Injuries may be divided into three main types: (i) airway and facial burns; (ii) oesophageal/gastrointestinal burns; and (iii) splash injuries.

Airway and facial burns

After alkali ingestion, immediate burning pain is experienced in mouth, oesophagus

Key points

Alkali ingestion may result in a severely disabling condition affecting otherwise healthy children.

Injury develops over hours to days after ingestion, and often results in progressively more difficult airway management.

Early assessment of the extent of gastrointestinal damage is essential.

Splash burns to skin and eyes need to be excluded in patients and prevented in attending medical staff.

The local Poisons Centre should always be contacted for advice.

Table 1 Alkaline substances commonly implicated in injuries

Substance	Products	Comments
Sodium hydroxide (caustic soda)	Drain cleaners, bleaches, oven cleaners, some non-phosphate detergents,	Term refers to dissolved and crystalline products
Potassium hydroxide (caustic potash, lye)	industrial paint strippers	
Sodium hypochlorite	Some household bleaches	Tends to be less toxic in children
Calcium hydroxide		Often contained in hair straighteners and relaxers
Sodium metasilicate	Mainly in liquid dishwasher products	Now gradually replaced by disilicates, which are less alkaline and less water soluble. This should reduce the amount of mucosal damage on ingestion.

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and stomach. Swelling of the lips may occur as well as oesophageal and pharyngeal oedema, leading to varying degrees of respiratory compromise. Dyspnoea and stridor may be seen early and require urgent intervention. Resuscitation is carried out after general resuscitation principles. The appearance of oral structures as seen on laryngoscopy should be documented, i.e. colour of mucous membranes and presence of oedema. Appearances may change as the burn develops, and the initial presentation may look deceptively harmless.

Patients with severe burns may need admission to intensive care for observation and management of respiratory distress. As with any other airway burn, this needs to be managed in an anticipatory attitude as oedema and damage progresses. Early intubation is often required, either for management of respiratory compromise or to facilitate diagnostic procedures such as gastroscopy and bronchoscopy. A chest radiograph should be obtained to assess possible aspiration or a perforating injury. If the latter is observed, involvement of the surgical team is essential. Oral, retrosternal and chest pain may be intense and require i.v. opioid analgesia.

Oesophageal and gastrointestinal burns

Gastrointestinal signs and symptoms such as vomiting, haematemesis, increased salivation, ulcerative mucosal burns or dysphagia may occur. There may be cardiovascular compromise requiring administration of fluids and inotropes, especially if perforation of the gastrointestinal tract has occurred. Arterial blood gas monitoring and assessment of acid–base balance may help to guide supportive management.

Stomach washout or lavage and induction of vomiting are contraindicated to avoid further damage to the oesophagus. Any regurgitated alkali would further worsen oesophageal injuries. Neutralizing chemicals should never be used. Heat production during the chemical reaction will exacerbate any tissue injury.

If the child is able to swallow, water may be given unless there is evidence of severe injury. Early insertion of nasogastric tubes is used in some centres to remove any remaining alkali from the stomach. However, blind placement of a nasogastric tube is controversial as it may create a false passage and further tissue damage. If a child is received at a hospital with a nasogastric tube already present, aspiration of stomach contents may be performed. This is less likely to be useful in alkali than in acid ingestion, as alkali damage occurs predominantly in the oesophagus, but may be beneficial if large amounts have been ingested.

Splash injuries

Splash injuries may occur to the eyes of the patient and require immediate copious irrigation to avert blindness. Ocular burns rapidly penetrate the cornea and anterior chamber. Tissue softening occurs because of disruption of cells and stromal mucopolysaccharides. An ophthalmology opinion should be sought as soon as possible.

Liquid alkali in particular permeates outer clothing and may cause skin burns, which are often missed. Children wearing nappies may have alkali seeping into the nappy, leading to severe perineal burns. Skin burns may be initially painless, resulting in a delay in treatment. Injury can progress over several hours and discolour the skin brown or black, which can make assessment of burn depth difficult. Complete exposure and prompt irrigation of affected skin areas is essential. Recurring skin breakdown and poor healing are frequently observed and increase the risk of secondary infection for a long time after the initial injury.

Emergency management

Key aspects of emergency management for caustic injuries are summarized in Table 2. When treating a patient with caustic injuries, caustic substances may transfer to the skin of attending medical staff, causing irritation and chemical burns. There is also a risk of splash injury to the eyes. Gloves, gowns and goggles should be worn especially for laryngoscopy and endoscopic procedures. Detailed information regarding the time of ingestion, and the amount and type of substance must be elicited. The original bottle from which the substance was ingested should be retained and the National Poisons Unit should be contacted for further details.

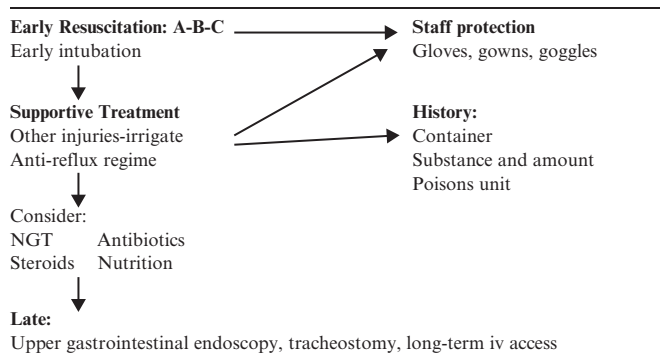
Subsequent supportive management

Owing to the evolving nature of the injury, laryngoscopy is likely to become increasingly difficult over the following days and weeks. Difficult intubation must be anticipated, even if previous intubations have been uneventful. In the presence of significant airway burns, early elective tracheostomy formation is advisable (Fig. 1).

In the long-term, the connection between trachea and hypopharynx may be lost owing to progressive severe scarring. Such patients require permanent tracheostomies. When children present for reconstructive surgery, a gas induction is usually preferred.



Fig. 1 Management of caustic injuries (Figure reproduced with the parents' permission).

Table 2 Management Summary

The tracheostomy tube is usually changed to a cuffed, reinforced tube for surgery to allow controlled ventilation without excessive leak, while providing good surgical access. These patients often have excessive secretions that are difficult to clear, and meticulous suctioning before and after completion of surgery is essential.

The use of broad-spectrum antibiotics is now generally accepted as part of prophylactic management of severe chemical burns. Stress ulcer prophylaxis with antacids, H₂ blockers or proton pump inhibitors should also be commenced.

Nutrition strategies need to be considered for patients with severe oesophageal burns. After endoscopic assessment, some patients with mainly upper airway and oesophageal damage will have a gastrostomy placed for feeding. Some centres advocate early management with parenteral feeding, while the extent of the injury is determined. If this is the plan, thought should be given to provision of long-term venous access such as tunnelled central venous catheters rather than the use of short-term central lines.

Areas of controversy

Endoscopy

Numerous studies have been conducted to investigate the relationship between clinical signs and symptoms and endoscopic findings after alkali ingestion. While some authors^{2,3} concluded that endoscopy is not merited in asymptomatic patients, others found no correlation between clinical presentation and extent of oesophageal injury on endoscopy.⁴ The National Poisons Unit concurs with the latter view and recommends that upper gastrointestinal endoscopy should be performed within the first 24 h to assess the extent of injury even in asymptomatic or mildly symptomatic patients, as absence of oral burns does not preclude oesophageal injury. This has been observed particularly in alkali ingestion with suicidal intent, when liquid alkali was quickly and purposefully swallowed. Endoscopic findings are classified broadly as shown in Table 3.

Repeated endoscopies are often required to monitor changes and for therapeutic purposes. Oesophageal stricture and or pyloric stenosis may develop, starting usually 14–21 days after ingestion. Most strictures are manifest within 2 months. These

Table 3 Endoscopic classification of burn severity

Grade 0: Normal oesophagoscopy
Grade 1: Mucosal injury with reddening and oedema
Grade 2: Submucosal injury, with deep ulceration and whitish pseudomembranes
Grade 3: Transmural injury, black-brown necrosis
The extent of the burn is described as circumferential or non-circumferential.

may present with persistent drooling and weight loss owing to inadequate nutritional intake.

Whenever possible, endoscopy should be avoided in the subacute phase (5–15 days after injury) owing to greater risk of perforation. At this time, the tissues are most friable, softening with sloughing but scar formation has not yet occurred. Traditionally, endoscopists stopped at the first deep, penetrating or circumferential burn. Use of flexible fibreoptic endoscopes appears to be associated with a lower risk of perforation allowing full assessment of injuries on panendoscopy. Other relative contraindications to endoscopy include third degree burns of the hypopharynx, burns involving the larynx, respiratory distress and suspected perforation. In these cases, radiographic studies with water-soluble contrast may be undertaken instead.

Both initial and follow-up endoscopic procedures invariably require general anaesthesia. After acute ingestion, presentation may range from a comparatively well child with minimal oropharyngeal changes to the cardiovascularly compromised patient with gross mucosal oedema, distorted anatomy and obvious oropharyngeal necrosis. Appearances often change over time, and a high index of suspicion with regard to developing airway problems is required by the anaesthetist.

Steroids

Steroids are a potentially valuable adjunct in the management of alkali ingestion because of their anti-inflammatory effects and reduction of collagen deposition, resulting in less severe scarring. Animal data suggest that strictures formed after steroid treatment are less well structured, with fewer inflammatory changes and less fibrin deposition. However, the clinical evidence for the efficacy of steroids is less clear. A review of 14 studies by Oakes⁵ showed no measurable benefit of the use of systemic steroids compared with patients not receiving steroids.

Steroids are generally thought to be of no value in first degree burns as these heal well and stricture formation is virtually unknown. Some authors recommend steroids in grade 2 burns as long as treatment is commenced early (i.e. within 48 h).^{6,7} Steroid use in third degree burns may be contraindicated because of a higher risk of developing perforations, although this risk has not been well quantified. Beneficial effects of other methods of steroid administration have been published in the form of case reports, such as interlesional injection of triamcinolone and the use of oral steroid solution.

In summary, the role of steroids is debatable, but there is some evidence that they may be useful in second to third degree burns if

given early. The risks of steroid administration must be carefully weighed in third degree burns.

Late management

Endoscopic dilatation may be required as primary treatment for oesophageal strictures. Frequency and timing is individualized and based on symptomatology and previous experience. Complications such as oesophageo-aortic fistulae, aortic rupture, tracheo-oesophageal fistula or broncho-oesophageal fistulas are rare, but can occur secondary to therapeutic dilatation. Recent work suggests that early stenting may reduce stricture formation and that strictures occurring despite the stent tend to be easier to dilate. Stenting is also suggested for strictures that are particularly resistant to repeated dilatation.

Severe oesophageal burns may result in destruction of the oesophagus such that oesophagectomy is required. At a later date, colonic or gastric interposition may be performed. These are extensive surgical procedures and children require assisted ventilation and intensive care support for at least one week after surgery. Extensive scarring after reconstructive surgery often remains a problem, and strictures seem to most commonly occur at the cervical anastomosis.

After caustic injury, there is an increased risk of oesophageal cancer, which may be up to 1000 times higher than in the general population. Malignancy usually occurs at the level of a stricture, most often in the mid-thorax, and screening with regular endoscopies is required.

Mitomycin C is an antifibrotic agent, which acts by interfering with RNA synthesis, thus inhibiting fibroblast proliferation and decreasing scarring. It may be injected or applied topically in patients with caustic injury and is mainly used to reduce scarring in the oral cavity. The ideal dosage is unclear and there is an increased risk of malignancy associated with the use of mitomycin C. A recent case report of a child with caustic injury who had undergone 16 previous endoscopic oesophageal dilatations, describes topical oesophageal application of mitomycin C.⁸ This was performed twice (one week apart) and the child required only one subsequent dilatation.

Psychosocial implications

At the time of injury, the medical team caring for the injured child will also need to support the child's parents and other caregivers. There is often a sense of guilt, blame and anxiety over the child's future health. This may be expressed in tearfulness or aggression

and frequent requests for reassurance. Long-term profound psychosocial impact of disfiguring corrosive injuries has been documented. One study showed that 50% of children develop behavioural or educational problems, such as delinquency and school avoidance. Antisocial behaviour is also more common. The whole family unit is affected, and there is a significant incidence of family break-ups, abandonment and job losses. Financial implications for the families are hard to measure but are probably substantial.⁹ The affected child as well as the family may benefit from the involvement of a clinical psychologist for counselling and development of coping strategies.

Schools may also find it difficult to cope with such a child in class, especially during long periods of reconstructive surgery. Loss of speech has profound effects on schooling and social development. Sign language (British Sign Language, Makaton) and other aids such as picture charts may be used. Even if speech is preserved, there is a need for close liaison with a speech and language therapist as inability to swallow and drooling may occur.

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See multiple choice questions 24–26.