

Caustic ingestion

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Corrosive ingestion is a rare but potentially devastating event and, despite the availability of effective preventive public health strategies, injuries continue to occur. Most clinicians have limited personal experience and rely on guidelines; however, **uncertainty persists about best clinical practice**. Ingestions range from mild cases with no injury to severe cases with full thickness necrosis of the oesophagus and stomach. **CT scan is superior to traditional endoscopy for stratification of patients to emergency resection or observation**. Oesophageal **stricture** is a common consequence of ingestion and **newer stents** show some promise; however, the place of endoscopic stenting for corrosive strictures is yet to be defined. We summarise the evidence to provide a plan for managing these potentially life-threatening injuries and discuss the areas where further research is required to improve outcomes.

Introduction

Corrosive ingestion is a rare but potentially devastating event that induces significant burdens on modern health systems worldwide.^{1,2} Management requires a multi-disciplinary approach involving a wide range of specialties including emergency care physicians, surgeons, anaesthesiologists, gastroenterologists, radiologists, otorhinolaryngologists, and psychiatrists.³ The low incidence of caustic injuries means that clinicians usually have limited personal experience and in the **absence of evidence-based guidelines**, uncertainty persists about best clinical practice. This uncertainty is mirrored by significant variations in patient management and reported outcomes across the world. The aim of this Review is to summarise current data and highlight existing controversies regarding digestive tract injuries resulting from caustic ingestion.

Worldwide epidemiological data are scarce mainly because of under-reporting of caustic ingestion.^{2,3} According to the 2013 annual report of the American Association of Poison Control Centers, there were nearly 60 000 cases of exposure to **corrosive agents** (48 000 to **bleach**, 7500 to **acids**, 4000 to **alkalis**), most of which occurred by ingestion; 30 fatalities could be undoubtedly related to corrosive ingestion.⁴ In the UK, 15 000 corrosive exposure incidents are recorded every year, but these figures also include other exposure routes (dermal, ocular).⁵ However, the true worldwide incidence of ingestion and prevalence of lesions such as strictures, including high-incidence countries and regions such as France, India, northern Africa, and eastern Europe, is unknown.^{3,5} It has been suggested that the incidence of

corrosive injuries is increasing, especially in low-income countries because of a lack of effective regulatory measures and public health prevention programmes.³ Two age groups are most at risk: first, children aged 2–6 years who unintentionally ingest household cleaning products and account for up to 80% of caustic ingestion cases but usually have mild injuries;^{6–8} and second, adults aged 30–40 years who have usually ingested strong corrosives with suicidal intent and present with severe, life-threatening injuries.^{9,10}

The corrosive agents involved include a wide range of chemicals that cause damage to and destruction of living tissue on contact. **Strong acids produce a coagulative necrosis that lessens tissue penetration and decreases damage**.^{2,3,11,12} Oesophageal eschar formation and prolonged gastric contact time due to pylorospasm explains the **preferential stomach involvement of acids**. **Alkalis produce liquefactive necrosis**, which results in immediately severe injuries at **all levels** of the gastrointestinal tract.^{2,3,11,12} Acid ingestion is more

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Key messages

- Accidental ingestion by **children accounts for 80%** of cases worldwide whereas in adults most ingestions are intentional resulting from underlying psychiatric illness
- Emergency management of caustic ingestion and the treatment of late sequelae require a multidisciplinary approach
- **CT examination** is reliable and reproducible in assessing **transmural digestive necrosis** and improves the **selection of patients for surgery**
- **Surgical resection** of organs subject to **transmural necrosis** is **life-saving** and should be done in first-level hospitals; age, the extent of initial damage, and the derangement of laboratory test results predict survival in these cases
- Treatment of **late sequelae** of caustic ingestion relies mainly on **endoscopy** (dilation, **stenting**) or **complex surgical** reconstructive procedures and should be done in expert referral centres
- Surgery (emergency or reconstructive) is seldom required in children; on such rare occasions, surgical decisions and procedures should be done in expert centres
- In low-resource settings, simple solutions such as gastrostomy placement are preferable and can be life-saving by addressing vital nutritional issues; complex endoscopic or surgical procedures in such conditions should be done cautiously
- Public health programmes to educate the public and establish effective measures limiting access to strong corrosive agents are paramount to decrease the incidence and severity of caustic ingestion

Search strategy and selection criteria

We searched MEDLINE and Embase for relevant papers published in English between Jan 1, 1990, and Feb 1, 2016, using the following terms: "caustic ingestion", "caustic lesions", "corrosive injuries", "oesophagus", "stomach", "oesophageal dilatation", "gastric outlet obstruction", "oesophageal reconstruction", and "coloplasty". Reports from within the past 5 years were selected preferentially together with commonly quoted older publications.

common in Asian countries such as India,¹³ Taiwan,¹⁰ and South Korea,¹⁴ whereas alkalis account for most severe caustic injuries in western Europe¹ and South America.¹⁵ It is unclear whether the nature of the ingested corrosive (ie, acid or alkali) affects patient outcomes in cases of massive ingestion.^{2,3,11,16} Oxidants (bleach) usually cause mild injuries by transformation of aminoacids into aldehydes and protein denaturation. Ammonia induces superficial haemorrhagic gastritis, which might progress 24–48 h after ingestion and requires specific surveillance.

The physical form of the ingested agent determines the pattern of damage to the gastrointestinal tract. Solid agents adhere to the mouth and pharynx producing maximum damage to these areas, whereas liquids transit rapidly and induce burns of the oesophagus and the stomach; concomitant vapour aspiration (of ammonia or formaldehyde) may cause airway burns. The ingested quantity is a major determinant of outcome (in adults, a normal sip is 30–50 mL, a large gulp is 60–90 mL) but this information is seldom available.^{2,3} Early contact of poison control centres is recommended¹⁷ because some corrosives can also cause severe systemic effects such as hypocalcaemia (phosphoric, hydrofluoric acids), hyponatraemia (strong acids or alkalis), hypokalaemia, and acidosis.

Significant tissue damage occurs within seconds of ingestion of strong corrosive agents.¹⁸ Haemorrhage, thrombosis, and inflammation with oedema are the dominant processes during the first 24 h following ingestion. Severe burns can progress to focal areas of necrosis with perforation as inflammation extends through muscle layers with submucosal thrombosis and bacterial invasion. On pathological examination, transmural necrosis shows specific criteria of coagulation necrosis (preservation of the general tissue architecture, preservation of the basic outline of the coagulated cells, presence of marked cytoplasmic eosinophilia) and nonspecific criteria of advanced necrosis (disruption of oesophageal wall architecture, karyolysis, presence of anucleate cells, necrotic debris, and leucocytic infiltrates).¹⁹ Fibroblast colonisation, mucosal sloughing, and granulation tissue appear at the end of the first week; oesophageal repair begins 10–15 days after ingestion and mucosal re-epithelisation is usually completed by the sixth week. Scar retraction, starting by the third week and evolving for several months, leads to stricture formation. Oesophageal dysmotility due to scarring can be associated with gastro-oesophageal reflux, which can in turn accelerate scarring.²⁰

If necrosis is transmural, immediate life-threatening complications can occur. Necrosis initially involves the oesophagus and the stomach but subsequent transpyloric passage of strong corrosives can result in duodenal or more distal small bowel and colonic necrosis.²¹ Occasionally, direct extension of gastric injuries to the transverse mesocolon causes colon necrosis. In the absence of appropriate management,

necrosis of intra-abdominal organs results in perforation, peritonitis, and death.^{22,23} Transoesophageal extension of necrosis to the mediastinum might involve adjacent structures with dramatic effects (eg, tracheobronchial necrosis,²⁴ aorto-oesophageal fistula²⁵). However, it is unclear whether isolated full-thickness oesophageal necrosis is uniformly fatal without surgery.^{23,26} Concomitant airway aspiration of the corrosive agent can result in progressive development of caustic pneumonia.²⁷ Along with local effects, caustic ingestion might induce systemic inflammatory response syndrome, sepsis, and a severe catabolic state, increasing systemic complications and mortality.¹⁷

Management

The main purpose of emergency management is patient survival and then all efforts should focus on treatment of early complications, prevention of delayed sequelae, preservation of nutritional autonomy, and quality of life.

Initial approach

Initial measures aim to avoid aggravation of injuries, control organ failure, and address potential systemic effects. During the pre-hospital phase, it is paramount to confirm ingestion and identify the corrosive agent, evaluate the context (accidental vs intentional) and the time from ingestion, detect co-ingestion of drugs including alcohol, and identify additional risk factors (pregnancy, extreme age, medical comorbidities).¹⁷ Manoeuvres liable to induce repeat oesophageal passage or risk aspiration of the corrosive agent (supine position, gastric lavage, ingestion of diluents) as well as attempts at pH neutralisation should be avoided because they are likely to exacerbate existing injuries.^{2,3,11,12,17,28} Support of vital functions (securing an airway, intravenous fluid replacement, pain medication) should be pursued during emergency department management alongside an evaluation of the extent of damage to the gastrointestinal tract. Laryngeal injuries are associated with severe oesophageal injuries in 40% of patients²⁹ and about 10% require intubation and mechanical ventilation on admission. Tracheostomy was eventually done in a third of patients with severe burns in one study.¹ The effectiveness of nasogastric tubes in preventing vomiting and stricture formation is controversial and routine insertion should be avoided.¹⁷ Systematic administration of antacid medication, corticosteroids, and broad spectrum antibiotics is not recommended, because of questionable efficacy.^{2,11}

Evaluation of gastrointestinal tract injuries

Specific evaluation of gastrointestinal damage aims to distinguish patients with severe life-threatening injuries who require emergency surgery from patients with mild injuries who are eligible for non-operative management.

Signs of digestive perforation (eg, rebound tenderness, subcutaneous emphysema) and haemodynamic instability are rare and should prompt immediate surgery

after minimal preoperative assessment.^{1,30} Otherwise, symptoms do not correlate reliably with the extent of damage; the absence of pain and oral lesions does not rule out significant gastrointestinal injury.^{2,3,6,8,11,31} Specific symptoms might suggest severe involvement of the larynx (hoarseness, stridor), the oesophagus (dysphagia, odynophagia, drooling), or the stomach (epigastric pain, haematemesis).^{3,5} Investigations might be unnecessary in asymptomatic patients following accidental ingestion of a weak corrosive.^{2,5}

After massive ingestion, emergency laboratory tests should be done, including white blood cell count, haemoglobin, platelet count, pH and serum lactate, serum concentrations of sodium, potassium, chlorine, magnesium, calcium, urea, creatinine, aspartate aminotransferase, alanine aminotransferase, bilirubin, alcohol levels, and measurement of β -HCG in young women.¹⁷ Although initially normal laboratory test results do not exclude transmural necrosis, leucocytosis, high serum C-reactive protein concentration,¹ severe acidosis (low pH, high blood lactate concentration),³² renal failure,²⁶ deranged liver function tests,³² and thrombocytopenia³³ can predict transmural necrosis and poor outcome. The pattern of changes in laboratory data is useful for monitoring patients and in guiding subsequent management.^{19,26}

Oesophagogastroduodenoscopy has been the mainstay of emergency management algorithms worldwide for decades.^{2,3,5,12,28} Early (3–48 h) flexible endoscopy assesses the extent and severity of caustic injuries from the luminal perspective.^{9,13} In expert hands, endoscopy can be safely repeated up to 3 weeks after ingestion without increasing risk of perforation.^{2,3,12} Several endoscopic classifications of upper digestive corrosive injuries have been proposed.^{2,13,22,23} The Zargar classification¹³ has gained wide acceptance and is used in most centres: grade 0 is normal; grade 1 is oedema and hyperaemia of the mucosa; grade 2a is superficial localised ulcerations, friability, and blisters; grade 2b is circumferential and deep ulcerations; grade 3a is multiple and deep ulcerations and small scattered areas of necrosis; and grade 3b is extensive necrosis.

Endoscopic grading predicts systemic complications, respiratory failure, emergency mortality, nutritional autonomy, and long term survival.^{9,34,35} Initial endoscopy is reliable in predicting future stricture formation, with low grade injuries (grades 1–2a) rarely causing strictures but stricture can occur in as many as 80% of patients with severe burns (grade 3b).⁹

The major drawback of endoscopy is its inability to predict accurately the depth of necrosis, which could lead to inappropriate non-operative management jeopardising survival and unnecessary resective surgery with deleterious effects on long-term survival, function, and management costs.^{1,22,23} Moreover, endoscopy is observer dependent and if it is delayed beyond 24–48 h there is the potential disadvantage of misinterpretation due to submucosal haemorrhages and oedema.

Kamijo³⁶ proposed an endoscopic ultrasound scoring system suggesting that the destruction of oesophageal muscular layers predicted stricture formation and response to dilation. However endoscopic ultrasound failed to improve the accuracy of conventional endoscopy in predicting early or late complications in another study.³⁷ Fibre-optic bronchoscopy is reliable in detecting tracheobronchial involvement and is mandatory for patients being considered for emergency surgery.²⁴ Transthoracic oesophagectomy should be done instead of transhiatal stripping oesophagectomy if transmural tracheobronchial involvement is suspected on bronchoscopy.

CT was the logical choice to alleviate the shortcomings of endoscopy because of its widespread use in the assessment of gastro-oesophageal diseases.^{38,39} Ryu and colleagues¹⁴ were the first to propose a CT-based classification of oesophageal corrosive injuries and showed that it was better than endoscopy in predicting long-term complications. Lurie⁴⁰ proposed an emergency CT grading system for corrosive injuries but suggested that endoscopy was better than CT in guiding emergency decisions. The discriminating power of this study was limited by the small cohort size and the strict criteria used to define necrosis (the highest grade category included only patients with radiological signs of perforation). A review⁵ based on these data concluded that CT cannot replace early endoscopy for the emergency assessment of gastrointestinal injuries after caustic ingestion.

These conclusions have been challenged by two studies from a high volume centre.^{19,33} A first analysis of 72 patients with grade 3b oesophageal endoscopic necrosis showed that use of CT to select patients for emergency oesophagectomy improved patient survival and functional outcomes and decreased management costs.¹⁹ Subsequent analysis of 120 consecutive cases of caustic ingestion showed that CT outperformed endoscopy in selecting patients for surgery or non-operative management.³³ Moreover, the high inter-observer agreement between general and gastrointestinal radiologists when assessing transmural gastro-oesophageal necrosis suggested that CT assessment of caustic injuries was reproducible outside specialised centres. In these studies, criteria of transmural gastro-oesophageal necrosis were derived from radiological reports on bowel ischaemia³⁹ and mainly relied on the persistence of anatomical structures and the degree of wall enhancement after contrast administration. In 2015, the World Society of Emergency Surgery consensus conference supported the introduction of emergency CT in the management of corrosive ingestion.¹⁷

Based on retrospective analysis of more than 300 CT scans, we propose a simplified radiological classification of corrosive injuries (figure 1). Grade 1 is normal appearing organs. When present, injuries usually correspond to low grade (0–2a) endoscopic burns; grade 2

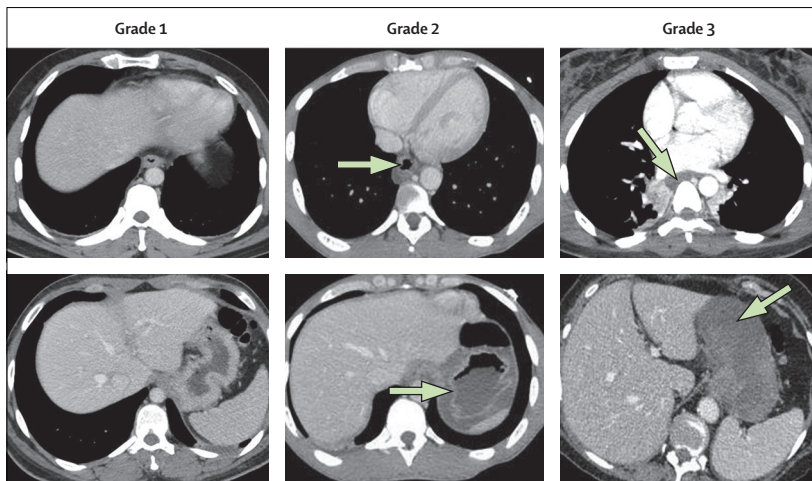


Figure 1: CT grading of corrosive injuries of the oesophagus and the stomach
Grade 1=normal appearance; grade 2=wall and soft tissue oedema, increased wall enhancement (arrow); grade 3=transmural necrosis with absent wall enhancement (arrow).

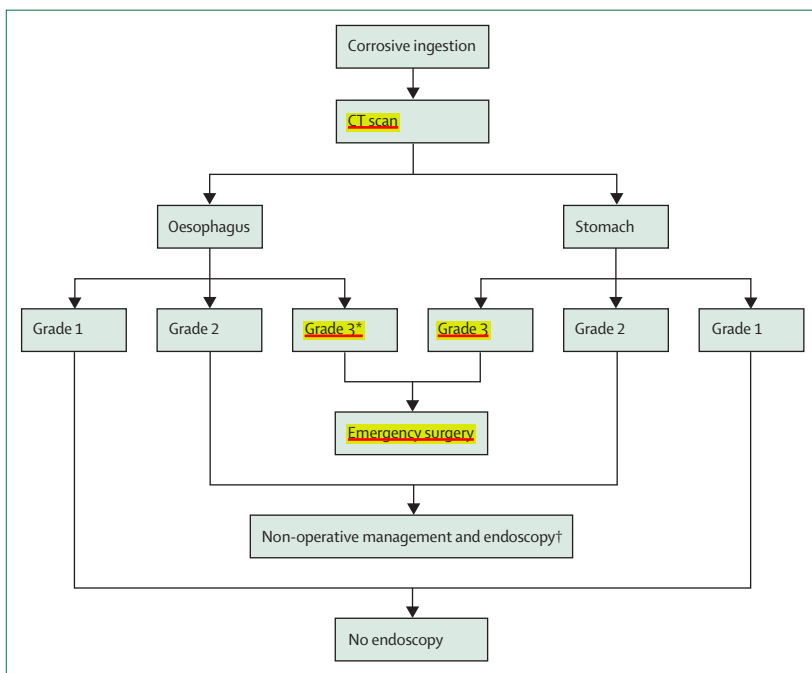


Figure 2: Management algorithm for caustic ingestion in adults

*Perform endoscopy before surgery in case of difficulties with CT interpretation. †Endoscopy done before discharge helps predict stricture risk.

is wall oedema, with surrounding soft tissue inflammatory change and increased post-contrast wall enhancement, which corresponds to more severe endoscopic burns (2b–3b) without transmural necrosis; and grade 3 is transmural necrosis as shown by the absence of post-contrast wall enhancement and in this situation, endoscopy uniformly shows grade 3b necrosis.

Management algorithm

Evidence of perforation after caustic ingestion is rare (0.5%) but should prompt emergency surgery.¹ Mortality

of caustic peritonitis is high and early identification of patients with full-thickness digestive necrosis is paramount as resection of the affected organs can improve survival by preventing intraperitoneal spillage of corrosive agents.^{1,22,23,26,30} Endoscopy-based algorithms aiming for no deaths among patients under watchful waiting recommend surgery for grade 3b injuries.^{22,23,34} Laparotomy²³ or laparoscopy⁴¹ can assess accurately the need for intra-abdominal organ resection and can correct endoscopic misvaluation. Because surgical exploration of the thoracic oesophagus is not feasible, futile oesophagectomy remains the major flaw of such algorithms.^{1,26} An algorithm based on combined CT and endoscopy can be used (figure 2).^{19,33}

Difficulties in interpreting CT scans and the ability to predict accurately the risks of stricture formation^{9,35} justify early endoscopy in patients with severe radiological injuries. Renal failure and allergy to contrast agents might contraindicate CT. There are no data on CT assessment of caustic injuries in children and therefore systematic use of CT assessment cannot be recommended in this group because of the scarcity of severe injuries and lifetime risks of radiation exposure.⁴² CT might be helpful in children with severe clinical, biological, and endoscopic criteria who are being considered for surgery.

Emergency surgery

The decision to perform an emergency operation after corrosive ingestion can be a heavy burden for the surgeon and is a life-changing event for the patient.¹ Emergency surgery is required whenever the initial assessment suggests transmural necrosis of the gastrointestinal tract. In one study,¹ the standard mortality ratio for patients operated for corrosive injuries was 21.5 compared with the general French population. In a population aged 40 years, half of patients died within 10 years of surgery and only half of patients eventually regained nutritional and respiratory autonomy.¹ Factors associated with negative effects on long term survival and functional outcomes included advanced age, tracheobronchial injuries, emergency oesophageal resection, need for extended resections, and severe derangement of laboratory test results. Finally, the overall cost of management of patients who underwent emergency surgery was high (€140 000 per patient).¹

Laparotomy is the standard emergency surgery but successful laparoscopic exploration has been reported.^{41,43} All obvious transmural necrotic injuries should be resected during the initial procedure but reoperation should be done whenever ongoing necrosis is suspected.²¹ Construction of a feeding jejunostomy at the end of surgery (irrespective of the procedure done) enables early enteral nutrition in patients with compromised digestive function.¹⁷

Surgical procedures

Oesophagogastrectomy, done through a combined abdominal and cervical approach using an oesophageal

	Year	Whole cohort (n) N	Emergency resection (n, %)	Oesophago-gastrectomy (n)	Oesophagectomy (n)	Gastrectomy (n)	Pancreato-duodenectomy (n)	Operative mortality (n, %)	Operative morbidity (n, %)	Reoperation (n, %)
Wu et al ⁴⁴	1993	..	28	22	0	0	6	5 (18%)	10 (36%)	..
Andreoni et al ⁷²	1995	57	11 (19%)	6	0	5	1	3 (27%)
Landen et al ⁴⁵	2000	..	14	12	0	2	14	7 (50%)	12 (86%)	..
Rigo et al ⁴⁶	2002	210	11 (5%)	6	0	5	0	9 (82%)
Ertekin et al ⁴⁷	2004	53	7 (13%)	2	0	3	2	3 (43%)
Tohda et al ⁴⁸	2008	92	12 (13%)	0	6	6	0	0
Chou et al ³²	2010	537	71 (13%)	71	0	0	0	29 (41%)
Zerbib et al ²⁶	2011	70	24 (34%)	0	0	24	3	4 (17%)	..	2 (8%)
Javed et al ⁴⁹	2012	209	13 (6%)	12	0	1	1	2 (15%)
Chirica et al ¹	2012	1024	253 (25%)	197	27	11	18	42 (17%)	167 (66%)	51 (20%)
Wu et al ⁵⁰	2015	426	64 (15%)	40	17	29 (45%)

Table 1: Outcomes of emergency surgery for caustic injuries

stripping technique, is the most common emergency operation done for the treatment of severe caustic burns.^{1,22,23} Mortality and morbidity are high (table 1) and pulmonary complications are the most common adverse events.¹ If necrosis is confined to the stomach, total gastrectomy with preservation of the native oesophagus should be considered.⁵¹ Immediate oesophagojejunostomy reconstruction has been shown to be safe in a high volume referral centre, with leaks in 5–8%.⁵¹ However, for the occasional surgeon, a damage control procedure such as exclusion with or without external oesophageal drainage is likely to be safer and should be considered in this setting.²⁶ Partial gastric resections are usually avoided because ongoing necrosis might compromise outcomes. The justification of oesophagectomy with gastric preservation based on isolated oesophageal necrosis has been challenged.^{26,33} Concomitant necrosis of other abdominal organs (spleen, colon, bowel, duodenum, pancreas) justifies extended resections in 20% of patients who undergo oesophagogastricomy.^{1,21} Pancreatoduodenectomy⁵² or duodenal stripping⁴⁴ can be undertaken in the small number (6%) of patients who have duodenal necrosis (pancreatic involvement is uncommon); immediate pancreatobiliary reconstruction was recommended after pancreatoduodenectomy if the patient's condition allows.⁵² Pancreatoduodenectomy for caustic injuries has high mortality (39–50%) and morbidity (94–100%), and functional results are dismal.^{44,45,52} On rare occasions, direct mediastinal extension of oesophageal necrosis results in tracheobronchial necrosis; pulmonary patch repair through a right thoracotomy approach might be lifesaving despite very high mortality rates (45%).²⁴ The presence of extensive bowel necrosis warrants discussion of abandoning the operation on a case-by-case basis because of poor survival and compromised nutritional issues.²¹ Figure 3 shows the most common emergency operations.

Non-surgical management

After initial assessment, 70–90% of patients are eligible for a first-line non-surgical approach.^{1,6,10} Patients with low

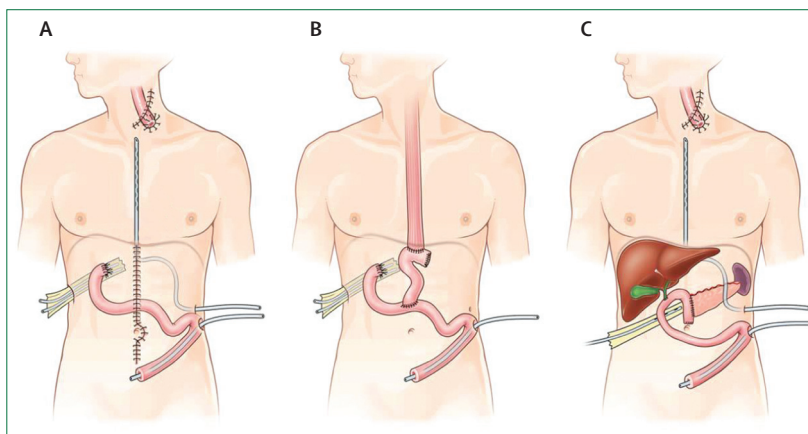


Figure 3: Emergency operations for caustic injuries

(A) Oesophagogastricomy, cervical oesophagostomy, feeding jejunostomy. (B) Gastrectomy with oesophagojejunostomy. (C) Oesophagogastricomy and pancreatoduodenectomy.

grade (endoscopy grade 1–2a, CT grade 1) injuries are suitable for early hospital discharge.^{3,9} Patients with more severe injuries (endoscopy grade 2b–3b, CT grade 2) require close monitoring.^{9,10} Deterioration of clinical signs and symptoms (abdominal pain, rebound tenderness, need for ventilator support, shock, neuropsychiatric deterioration) or laboratory test results (renal failure, leucocytosis, acidosis, thrombocytopenia) should prompt a repeat of the management algorithm and reconsideration of indications for surgery.^{1,26} In one study,¹ 18 (2%) of 784 patients managed conservatively by endoscopy alone were eventually switched to surgery, whereas in a later cohort, none of 143 patients managed by CT-endoscopy were switched.^{19,33}

The optimal time to resume eating after corrosive ingestion is unknown. Correlations between oral re-alimentation and risks of perforation and of delayed sequelae have not been clearly established. Thus, most clinicians reintroduce oral feeding as soon as patients are able to swallow normally,^{2,3,17} although complex re-alimentation algorithms have also been proposed.³⁴ The role of nutritional support is paramount in the acute

phase and subsequently until effective dilatation or oesophageal reconstruction is achieved. When oral alimentation is not feasible, **total parenteral nutrition** and **early enteral feeding**, through **nasojejunal** tubes or **jejunostomy**, is recommended.⁵³ Control of the psychiatric condition and psychological support are mandatory before hospital discharge, regardless of the severity of corrosive injuries.¹⁷ Although regular follow-up is recommended for early detection and timely treatment of sequelae such as oesophageal stricture, the frequency of outpatient visits and the best way (clinical examination, endoscopy) to conduct follow-up are still open to debate.

Delayed sequelae of corrosive ingestion

Late sequelae of corrosive ingestion occur after variable periods, are disabling, and can be life-threatening. The most common sequelae include **haemorrhage**, **fistula formation** (**tracheobronchial**, **aortoenteric**), **pulmonary complications**, **stricture development**, and **malignancy**.

Bleeding is a **rare** complication, occurring in around 3% of patients, and usually occurs **3–4 weeks** after ingestion.⁵⁴ In one study,⁵⁴ **sentinel bleeding preceded by 2 days** the occurrence of massive gastric or duodenal haemorrhage. Management includes resection or **embolisation** of the bleeding site but mortality (16%) and morbidity (75%) are high.⁵⁴

Fistulisation into adjacent organs can occur at any time after massive ingestion of strong corrosive agents. Chronic tracheo-oesophageal fistula is a rare complication (in 3% of patients). Management includes repair of the airway defect and oesophageal reconstruction usually by a staged surgical approach.^{55,56} In one study,⁵⁵ aorto-oesophageal fistula occurred 5 days to 2 months after ingestion in three (0.2%) of 1260 patients and is almost universally fatal.

In one study,²⁷ **aspiration pneumonia** was reported in **4.2%** of patients after caustic ingestion; risk factors included advanced age, hesitation in swallowing with prolonged oropharyngeal storage, and emergency nasogastric tube positioning. **Small airway obstruction** by sloughing, exudation, ulceration, and granulation tissue leads to recurrent atelectasis and pneumonia; treatment is difficult and **mortality reaches 60%.**²⁷

Stricture formation is by far **the most common**, disabling, and resource-consuming late complication.^{2,3,11,12,17} Strictures usually **develop within 2 months** (3 weeks to 1 year) after ingestion and their development is **reliably predicted** by both **emergency endoscopy**⁹ and **CT.**¹⁴ Several strategies have been proposed for stricture prevention, but the clinical benefits have not been clearly demonstrated. Studies in human beings **failed to prove** the effectiveness of **antibiotics**⁵⁷ and of systemic or intralesional **steroid** administration^{57,58} to prevent strictures. The use of intraperitoneal injections of fluorouracil,⁵⁹ antioxidant agents,⁶⁰ octreotide,⁶¹ and cytokines⁶² have been tested in animal models but not in

human beings. Oesophageal stenting for stricture prevention has been attempted without gaining wide acceptance.^{3,28,63} Most recommendations on stricture prevention and treatment rely on small retrospective studies, underscoring the need for well-designed research on this topic.

Gastric strictures are uncommon because of the large diameter of the stomach and are mostly caused by acids.⁶⁴ The antrum is most commonly involved (in 75–80% of cases), but hourglass (15%) and diffuse gastric (5%) strictures have also been described.^{64,65} Half of patients have concomitant oesophageal strictures and gastric outlet obstruction might be unmasked after treatment of oesophageal involvement. Although successful management of gastric outlet obstruction by endoscopic balloon dilatation has been reported in patients with short strictures (<15 mm),⁶⁶ perforation (46%) and failure (55%) are common.⁶⁷ Evidence supporting the use of stents in the management of gastric outlet obstruction is scarce. Resection or bypass 3–6 months after ingestion has low morbidity (10–15%) and mortality (0–4%) and the success rate is high.¹⁷

Traditionally, **corrosive strictures** can involve all **oesophageal segments**, are **multiple, long, irregular**, and have long stabilisation delays.⁶⁸ The main treatment goal should be the improvement of symptoms and of the nutritional status, rather than the conservation of a large oesophageal lumen patency. **Endoscopic dilation is the first-line management option.**³ Dilation can be started safely after healing of acute injuries, usually between the **third and the sixth week**; **later** management might compromise outcomes because of oesophageal wall **fibrosis** and collagen deposition.⁶⁸ CT or endoscopic ultrasound wall thickness can predict the response to dilatation.⁶⁸ **Savary bougies** are preferred to balloon dilators,⁶⁹ although studies have shown no clear advantage of one method over the other.⁷⁰ Even for experienced clinicians, **perforation rates** after dilatation of corrosive strictures are **higher** than other benign strictures (**4–17%** vs **0.1–0.4%**).³ Oesophageal perforations in this context are usually **contained** and can benefit from **non-operative** management; it is unclear whether perforation should preclude further dilation attempts.⁷¹ **The interval between dilations** varies between **1 week and 3 weeks**, and **three to five sessions** are expected to provide satisfactory results.³ A cutoff of five to seven failed sessions has been proposed for stopping dilatations and considering reconstructive surgery.⁷² Worldwide, such decisions are nevertheless influenced by several other factors related to the patient (eg, age, malnutrition, operative risks), the physician's expertise, and the availability of alternative surgical options. Overall, **roughly half** of **dilations** for caustic strictures are **successful**, which is significantly lower than for other benign strictures (75–80%).³

The advent of interventional endoscopy has renewed the interest of **intraluminal stenting** to prevent stricture recurrence after dilatation.^{73–78} Although encouraging

results have been reported with silicone rubber, polyflex, and biodegradable stents, their widespread clinical use is currently hindered by issues such as hyperplastic tissue growth, removal difficulties, high migration rate (25%), high recurrence rate (50%), low availability, and high costs.^{3,28} Therefore, the place of endoscopic stenting for corrosive oesophageal strictures is yet to be defined and controlled studies are needed. Intralesional steroid injections increase the effects of endoscopic dilation⁷⁹ and topical mitomycin⁸⁰ can be effective for the treatment of complex strictures; such combined approaches warrant discussion before contemplating surgery.

Pharyngeal strictures have been reported in 0·7–6% of patients after caustic ingestion and have been attributed to prolonged contact of the caustic agent with the superior aerodigestive pathways after massive ingestion, forced vomiting, hesitation before deglutition, or ingestion of solid crystal forms.⁸¹ Scarring involves the hypopharynx, the glottis, and the base of the tongue, and may interfere with the mechanisms of deglutition and respiration rendering management difficult. Endoscopic treatments are usually ineffective; some patients may eventually recover nutritional and respiratory autonomy after complex surgical repair.⁸¹ Corrosive induced microstomia and glossopalatine synechia are rare, but can compromise functional outcomes; prevention after ingestion by daily fatty dressings and regular tongue mobilisation is important.

Risks of oesophageal malignancy after caustic ingestion are thought to be 1000–3000 times higher than in the general population.² Up to 30% of patients with caustic injuries develop oesophageal cancer, with a latency period of up to 40 years.⁸² The risk is probably overestimated because of several confounding factors such as alcohol misuse and smoking habits.³ Associations between caustic gastric injuries and cancer are less well established.² Although specific screening programmes seem justified after caustic ingestion, there are no reliable guidelines addressing the topic.

Oesophageal reconstruction

Oesophageal reconstruction is required to restore digestive continuity after emergency oesophageal resection and in patients with strictures that were not eligible for or had failed dilation. Although emergency surgery can be done in admitting facilities, patients that require complex reconstruction benefit from early referral to expert centres.⁸³

One stage reconstruction after emergency oesophagectomy is not advisable because subsequent development of pharyngeal strictures might compromise outcomes and render reconstruction futile.¹ A minimum 6 months' delay in reconstruction enables injuries to stabilise and has been associated with decreased rates of cervical anastomotic strictures and with improved functional outcomes.^{72,84} Control of psychiatric disease when present is essential, especially if concomitant

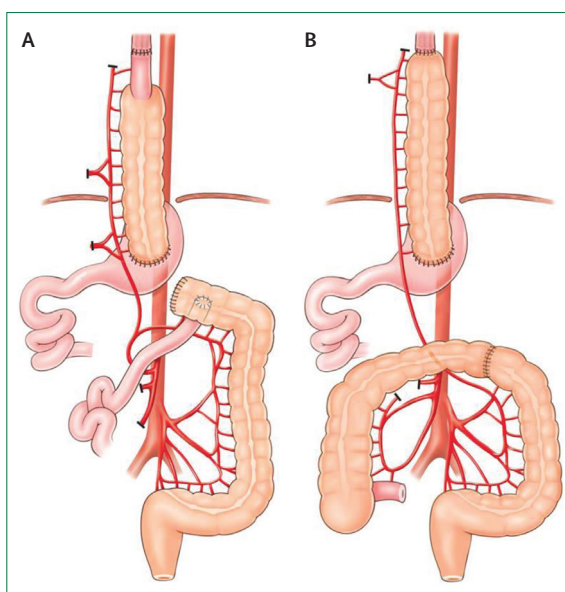


Figure 4: Oesophageal reconstruction

The stomach had been preserved initially and digestive continuity subsequently restored by colonic interposition: (A) right colon, (B) left colon.

pharyngeal reconstruction (which requires active patient participation throughout the re-education process) is considered.⁸¹ If the use of a colonic substitute is considered, most authors recommend preoperative colonoscopy to rule out malignancy.^{72,85} Angiographic studies of the vascular pedicle before colon interposition have been advocated,⁸⁶ but are not used routinely because they do not replace intraoperative vascular clamping tests.^{72,83} Angiography might be useful for strategy planning after failed primary reconstruction.⁸³ Otolaryngological assessment including fibre-optic nasopharyngoscopy, hypopharyngoscopy, and direct laryngoscopy under general anaesthetic is necessary. Failure to detect pharyngeal strictures before oesophageal reconstruction invariably results in functional failure.⁷²

The choice of the oesophageal substitute after caustic ingestion is a matter of debate. Colon interposition (figure 4) is the most common reconstructive procedure.^{2,3} The issue of which colon segment (right vs left) should be preferred has never been addressed by randomised controlled studies. Despite pros and cons for the use of either right or left colon transplants, results of large series originating from expert centres show similar results.^{72,84,85,87–91} Operative mortality of colon interposition ranges between 0% and 10% and morbidity ranges between 19% and 63%. Specific complications include graft necrosis (0–14%) and cervical anastomosis leakage (6–28%; table 2). Retrosternal oesophagocoloplasty is the most common procedure and debate persists as to whether to resect^{96,102} or bypass^{72,84} the native oesophagus (if still present) at the time of reconstruction. Disagreement concerns mainly the risks of malignancy and mucocoele in the retained bypassed oesophagus and

	Year	Patients with corrosive injuries (n)	Oesophageal substitute	Operative mortality (n, %)	Operative morbidity (n, %) (%)	Graft necrosis (n, %)	Cervical leakage (n, %) N (%)	Late morbidity (n, %)	Anastomotic stricture (n, %)	Reflux (n, %)	Redundancy (n, %)	Success† (n, %)
Hong et al ⁹²	1963	81	Colon	6 (6%)	..	7 (9%)	23 (28%)	..	13 (16%)	12 (15%)	..	67 (84%)
Chien et al ⁹³	1974	60	Colon	1 (2%)	21 (35%)	0	6 (10%)	..	15 (25%)	6 (10%)	1 (2%)	..
Wu et al ⁹⁴	1992	75	Colon, stomach	0	14 (19%)	0	5 (7%)	..	3 (4%)	..	2 (3%)	..
Wain et al ^{91*}	1999	20/52	Colon	2 (4%)	27 (52%)	5 (10%)	3 (6%)	..	24 (46%)	3 (6%)	2 (4%)	57 (90%)
Bassiouny et al ^{95†}	2001	100	Colon	3 (3%)	..	0	8 (8%)	..	7 (7%)	..	6 (6%)	95 (95%)
Popovici et al ^{88*}	2003	284/347	Colon	16 (5%)	..	4 (1%)	24 (7%)	12	22 (6%)	..	2 (4%)	202 (80%)
Gupta et al ⁹⁶	2004	51	Colon, stomach	0	..	0	10 (20%)	..	30 (59%)	51 (100%)
Han et al ⁹⁷	2004	68	Colon, stomach	0	17 (25%)	0	9 (13%)	..	3 (4%)	65 (96%)
Knezevic et al ⁸⁴	2007	336	Colon	4	89 (27%)	8 (2%)	31 (9%)	47 (14%)	15 (4%)	..	14 (4%)	233 (82%)
Deng et al ⁹⁸	2008	85	Colon	7 (8%)	39 (47%)	2 (2%)	15 (18%)	..	7 (8%)
Javed et al ⁹⁹	2011	176	Colon, stomach	11 (6%)	..	8 (5%)	22 (13%)	..	33 (19%)	25 (15%)	..	143 (81%)
Boukerrouche et al ^{100*}	2013	57/60	Colon	2 (3%)	27 (45%)	2 (3%)	10 (17%)	11 (18%)	5 (8%)	2 (3%)	2 (3%)	58 (97%)
Ezemba et al ¹⁰¹	2014	21	Colon	2 (10%)	11 (52%)	3 (14%)	4 (19%)	..	3 (16%)	4 (19%)	..	15 (80%)
Chirica et al ⁸¹	2015	238	Colon	7 (3%)	150 (63%)	12 (5%)	50 (21%)	98 (41%)	49 (21%)	21 (9%)	..	166 (76%)

* Also included patients with cancer; the ratio corrosive ingestion cases:whole cohort are shown; results of oesophageal reconstruction are provided for the whole series. †In children. ‡Crude data and percentages as reported by the authors; the denominator can be either the whole cohort or the number of patients available for follow-up.

Table 2: Outcomes after oesophageal reconstruction for caustic injuries

the operative risks of oesophagectomy in the scarred mediastinum.

A report comparing colon and stomach interposition for corrosive injuries showed that the duration of surgery, blood loss, incidence of conduit necrosis, and in-hospital mortality were significantly lower after use of a stomach conduit.⁹⁹ Use of gastric conduits in the setting of corrosive oesophagitis is nevertheless hampered by the presence of concomitant gastric involvement (need for emergency resection, stricture formation) after caustic ingestion. Length issues and the complexity of the surgical procedure usually limit first-line use of the jejunum for oesophageal replacement; salvage utilisation has been reported after failure of primary reconstruction.⁸³ Surgical management of pharyngeal strictures is usually recommended as a one-stage procedure at the time of oesophageal replacement and the difficulty of the situation is mirrored by the diversity of surgical procedures described (table 3).^{105,112,113} In one report,⁸¹ pharyngeal reconstruction by colopharyngoplasty resulted in similar operative morbidity and mortality as oesophagocoloplasty, although functional outcomes were significantly worse.

Late complications after oesophageal reconstruction are common and jeopardise outcomes. In one study,⁷² late morbidity was recorded in half of patients after colon interposition and graft-related dysfunction accounted for half of functional failures. Stenosis of the cervical anastomosis is common (4–59%) and usually complicates leakage.⁷² Complications such as redundancy, ulceration, reflux, and transplant-specific disorders (cancer,¹¹⁴ diverticulitis¹¹⁵) are rare but occasionally compromise reconstruction outcomes.^{116–118}

Surgical correction of graft-related complications has been successful in select patients; in extreme situations redo reconstructions with a novel substitute provide similar results to primary reconstruction.⁸³ In one study,⁸¹ inability to control the underlying psychiatric disease accounted for half of functional failures and 15% of patients attempted suicide again (one third by caustic reingestion) after reconstruction. These data underscore the difficulties in selecting patients for reconstruction and the necessity for long-term psychological support.

Most patients (75–100%) regain some degree of nutritional autonomy after oesophageal reconstruction for corrosive injuries (table 2). Factors that negatively affect functional outcomes include old age, severe psychiatric disorders, massive ingestion requiring emergency tracheotomy and extended visceral resections, short delays in reconstruction, and pharyngeal involvement. The lack of reliable definitions of functional success after oesophageal reconstruction precludes valid comparison between published series and calls for an international expert consensus conference on the topic.

Caustic ingestion in children

Caustic injuries in children are most likely caused by accidental ingestion, with boys more often involved than girls.²⁸ Injuries are usually mild with emergency surgery rarely indicated and the overall mortality is lower than in adults. Endoscopic assessment remains the gold standard in this population to diagnose severe injuries (0·5–14%) and predict risk of stricture (6–10%).²⁸ Dilation is the cornerstone of treatment of

	Year	Patients (n)	Technique	Laryngectomy (n, %)	Simultaneous oesophageal reconstruction (n, %)	Operative mortality (n, %)	Operative morbidity (n, %)	Cervical leakage (n, %)	Dilation (n, %)	Surgical revision (n, %)	Median follow-up (range)	Definitive tracheotomy (n, %)	Success* (n, %)
Tran Ba Huy ¹⁰³	1988	18	End-to-side ileopharyngeal anastomosis	11	18 (100%)	0	..	4 (22%)	15 (83%)	13 (72%)	.. (1.5 years to 8 years)	4 (22%)	11 (61%)
Park ¹⁰⁴	2001	8	Side-to-side hypopharyngoileal anastomosis	0	8 (100%)	0	2 (25%)	0	1 (13%) (35 months to 67 months)	0	7 (88%)
Anantha-krishnan ¹⁰⁵	2001	4	Island pectoralis major myocutaneous flap	0	0	0	0	2 (50%)
Wu ¹⁰⁶	2001	50	Hypopharyngo-enterostomy	1	50 (100%)	1 (2%)	8 (16%)	3 (6%)	..	6 (12%)	42 (84%)
Jianj ¹⁰⁷	2005	14	End-to-end colopharyngeal anastomosis	1 (7)	14 (100%)	0	-	4 (28%)	2 (14%)	1 (7%)	4 years (6 months to 10 years)	1 (7%)	14 (100%)
Anantha-krishnan ¹⁰⁸	2007	4	Sternocleidomastoid muscle myocutaneous inlay flap	0	0	0	2 (50%)	2 (50%)	0	2 (50%)
Huang ¹⁰⁹	2009	10	Laryngotracheal-colocolic anastomosis	10	10 (100%)	0	..	0	0	0	8 months (3 months to 3 years)	10 (100%)	9 (90%)
Radovanovic ¹¹⁰	2009	83	End-to-end and side-to-end colopharyngeal anastomosis	..	83 (100%)	5 (6%)	14 (17%)	4 (5%)	4 (5%)	4 (5%)	16 years (1 year to 30 years)	..	72 (87%)
Vimalraj ¹¹¹	2011	21	Transgastric retrograde dilatation	0	7 (33%)	0	2 (10%)	..	10 (48%)	1 (5%)	..	2 (10%)	11 (52%)
Chirica ⁸¹	2015	116	End-to-end colopharyngeal anastomosis	29 (25)	116 (100%)	2 (2%)	75 (65%)	20 (17%)	13 (11%)	17 (16%)	61 (57%)

*Crude data and percentages as reported by the authors. The denominator can be either the whole cohort or the number of patients available for follow-up.

Table 3: Outcomes of pharyngeal reconstruction for caustic injuries

caustic strictures in children and should be pursued for years if surgery can be avoided; a feeding gastrostomy might provide a retrograde approach for dilation of complex strictures.¹¹⁹ Results of oesophageal reconstruction in children done in expert centres are good but removal of the native oesophagus is recommended to avoid malignancy.⁹⁵

Management of caustic ingestion in low-resource settings

In low-income countries, specific issues including delays in management, limited access to medical expertise and technology, and poor follow-up have a negative effect on clinical outcomes.¹¹⁹ These factors should be taken into account when planning management of caustic injuries in low-resource settings. Children are often involved and oesophageal strictures requiring repeat dilations are the most common form of presentation.¹¹⁹ Introduction of complex endoscopic treatments and surgical procedures in the absence of a suitable hospital environment warrants caution. Under such circumstances, simple interventions such as placement of a surgical gastrostomy should be favoured because they might help avoid malnutrition and prolong survival.¹¹⁹

Future directions

Many questions remain unanswered regarding the optimal management of corrosive ingestion and further research is needed to standardise patient management. An international project is in progress under the auspices of the World Society of Emergency Surgery to develop a register that enables collection of clinical data after corrosive ingestion from many institutions.¹⁷ Such a register would enable large scale evaluation of clinical outcomes and of prognostic factors and alleviate some of the research difficulties induced by the low incidence of caustic ingestion. Research directed at assessment of quality of life after caustic ingestion is scarce,^{81,120} even though quality of life is a major concern in modern medicine. This shortage is of special concern because complex reconstructive surgical procedures are done with an exclusively functional purpose in these patients. Preventive strategies, such as the introduction of safety bottle tops, crystal rather than liquid forms of corrosive agents, appropriate labelling and packaging, and providing wide publicity in the media have proven effective in several countries.²⁸ Despite these convincing data, public health programmes directed at education and the establishment of effective measures limiting access to strong corrosive agents are lacking in several low-income and high-income countries.

Contributors

MC designed the study, searched the scientific literature, analysed data, and wrote and revised the report. LB designed the study, analysed data, and wrote and revised the report. MDK and ES designed the study, and wrote and reviewed the report. PC designed the study, searched the scientific literature, analysed data, and wrote and revised the report.

Declaration of interests

We declare no competing interests.

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