https://doi.org/10.47811/bhj.30 **REVIEW ARTICLE**

# Management of corrosive oesophageal injuries: a review

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## ABSTRACT

With the increasing incidence of corrosive oesophageal injuries, especially in the developing countries, understanding the pathophysiology and the approach to its management in the emergency and elective settings has become of paramount importance for the surgeon. The approach to the management varies depending on the time of the diagnosis and the location of injury. Endoscopy plays a key role as the first step in the evaluation of such patients in the emergency as well as late elective setting. With the advancement in endoscopic interventions, there has been a shift of preference towards the less invasive and less morbid endoscopic techniques to address the late sequelae of corrosive oesophageal injuries. Refractory cases eventually merit surgery, even at the cost of the morbidity and mortality associated with such procedures. The surgical approach also varies depending on the location of the stricture, with the proximal 1/3rd strictures representing the most challenging location. A regular follow up and endoscopic surveillance are needed in these patients to screen for any late complications such as cancer or dysmotility.

**Keywords:** Caustic, Bypass, Endoscopic dilatation, Resection.

## INTRODUCTION

Oesophageal injuries related to the ingestion of caustic substances remain an important issue of concern in the Indian subcontinent and in the West. The magnitude of injury depends on various factors like the nature and volume of the substance ingested, the intent of ingestion (suicidal/accidental), the age of the patient and the duration of exposure[1]. The circumstance of the injury varies among children and adults; with 80% of paediatric injuries being accidental and the majority of the adult injuries being suicidal[2]. Most studies from the West have found alkalis (lye used to clean drains) to be most commonly implicated as the source of injury. However, the Indian data shows a different picture, with common household acids being the most frequent source of caustic injuries[3]. In a review of the literature published by Lakshmi et al. toilet cleaning fluid (hydrochloric acid) and Aqua regia or Goldsmith's solution (nitric acid) were the most common causes in this country[4]. A major problem in stating the true epidemiology of these injuries is the heavy under-reporting of the statistics, leading to a skewing of the data towards the better-resourced centres in the cities[5].

### Pathophysiology

Due to the difference in their chemical properties, acids and alkalis vary widely in the severity of the injuries they cause. According to the old literature, acids 'lick the esophagus and bite the stomach[6]. In contrast, alkalis tend to cause deep oesophageal damage due to their property of causing liquefactive necrosis. However, this dictum has been challenged by some authors, who have shown a greater incidence of oesophageal injuries with acid ingestion[7]. Acids tend to form an eschar after contact with the mucosa due to coagulative necrosis. This limits the further penetration of the caustic liquid into the deeper layers of the oesophagus[8]. On the other hand, alkalis tend to cause liquefactive necrosis and thereby penetrate deep into the layers of oesophagus causing perforation. The physical nature of the substance ingested also determines the location of the caustic injury[9]. The substances ingested in powder or crystal form tend to cause more damage to the posterior pharynx and upper oesophagus as a result of a prolonged contact time, while liquids tend to be more injurious to the body and lower end of the oesophagus and stomach. It has also been shown that injuries occurring in the fasting state tend to affect the gastric antrum whereas those in the post prandial state affect the gastric corpus[10].

Caustic injury takes place in three different phases[11]. In the first phase there is haemorrhage and congestion due to eosinophilic necrosis. This is followed by coagulative necrosis of the proteins in the initial 24-72 hours. In the first 4-7 days after injury, the surface gets covered with mucosal slough and granulation tissue with super-imposed bacterial infection. At this time the oesophageal wall is friable with a high risk of perforation. This is followed 1-3 weeks later by the third phase of regeneration, where fibrosis and scar formation takes place. Owing to the fragility of the oesophageal wall, endoscopic interventions are avoided in the first 5-15 days of caustic injury to avoid iatrogenic perforation[12]. Scar retraction begins by the 3rd week, which continues for months after the injury. Reactive Oxygen Species (ROS) generation leading to formation of malonedialdehyde has been implicated in causing stricture formation in caustic oesophageal injuries[13]. Injury to the Lower Oesophageal Sphincter (LES) causing a high chance of Gastro-

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Esophageal Reflux (GER) has also been shown in some studies. Hence, periodic screening for GER is recommended for all patients who have had caustic oesophageal burns[14].

Most patients present in the emergency setting with features of dysphagia, drooling and stridor. However, the severity of the symptoms cannot always be correlated with the extent of injury[15]. Stridor and drooling have been found to be highly indicative of significant oesophageal injury. Respiratory tract injuries may result secondary to laryngeal injury and upper airway oedema, which may ultimately need a tracheostomy[16]. However, lower respiratory tract injuries have been uncommon, indicating the effectiveness of the pharyngo-glottic mechanisms in preventing aspiration of the contents into the lower respiratory tract[17].

**Evaluation and Assessment**

Routine laboratory tests are not of any value in assessing caustic oesophageal injuries. Rigo et al. have shown that a high WBC count (>20000/mm3), elevated CRP, advanced age and the presence of oesophageal ulcers to be predictors of mortality in adults in the emergency setting[18]. Another study by Cheng et al. showed an arterial pH <7.22 and a base excess < -12 to be useful indicators of the need for emergency surgery in these patients[19]. However, these tests lack sensitivity and specificity and therefore, have limited applicability in the management of oesophageal injuries.

**I. The role of Endoscopy**

Oesophago-gastroduodenoscopy is considered to be the most crucial investigation in the initial 12-48 hours of corrosive injuries[20]. Gentle manoeuvring and insufflation are recommended during this procedure. Although, it is recommended to avoid endoscopy in the initial 5-15 days of injury due to the high friability of the oesophageal mucosa, Tiryaki et al. have performed early bougienage and dilatation without complications during this time frame and shown superior results in decreasing the rates of stricture formation[21]. Correlation of the degree of injury to the upper aero-digestive tract with the grade of severity of gastric/ oesophageal injuries have also shown by some authors. Aronow et al. found that the presence of significant lip and oropharyngeal injuries precluded a more severe grade of oesophago-gastric injuries[22]. It is recommended that adults with a history of ingestion with suicidal intent should undergo endoscopy as a routine because of the larger amount of the content usually ingested and thereby, the possibility of a more severe grade of injury. Severe oesophageal injuries have been reported in about 12-19.3% of asymptomatic children with a history of caustic ingestion[23]. However, significant damage on endoscopy is seldom found in the absence of symptoms.

The amount of symptoms produced, the nature of caustic substance ingested and the presence of upper aerodigestive and ENT injuries must be considered, when deciding for an early endoscopy[24]. Radiological suspicion of perforation and significant supraglottic or epiglottic burns is a contraindication to performing an early procedure[25].

The severity of esophageal injuries on endoscopy is graded according to the recommendations by Zargar et al (Table 1)[12].

**Table 1. Endoscopic classification of corrosive injuries (Zargar et al)[12]**

| Grade | Description |
|---|---|
| Grade 0 | Normal |
| Grade 1 | Superficial mucosal oedema and erythema |
| Grade 2A | Superficial ulcerations, erosions and exudate |
| Grade 2B | Deep, discrete and circumferential ulcerations |
| Grade 3A | Focal necrosis |
| Grade 3B | Extensive necrosis |
| Grade 4 | Perforations |

The degree of oesophageal injury on endoscopy is correlated with the mortality and the extent of systemic complications, with an increase in each grade being associated with a nine fold increase in morbidity and mortality (Figure 1 & 2). In their study, Zargar et al. found an uneventful recovery in patients with grade 0, 1 and 2A burns. Higher grades of burns (grade 2B and 3) developed

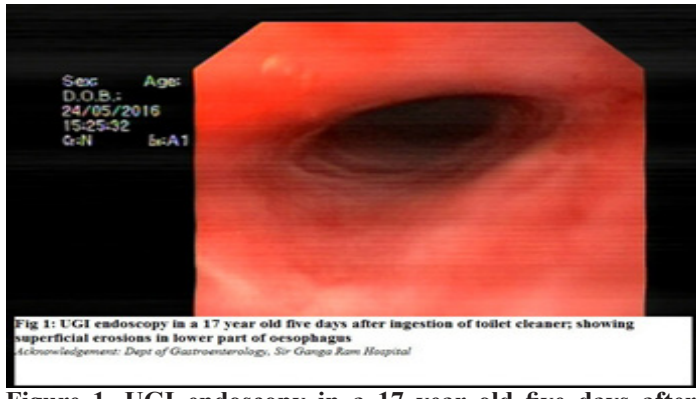


**Figure 1. UGI endoscopy in a 17 year old five days after ingestion of toilet cleaner; showing superficial erosions in lower part of oesophagus**

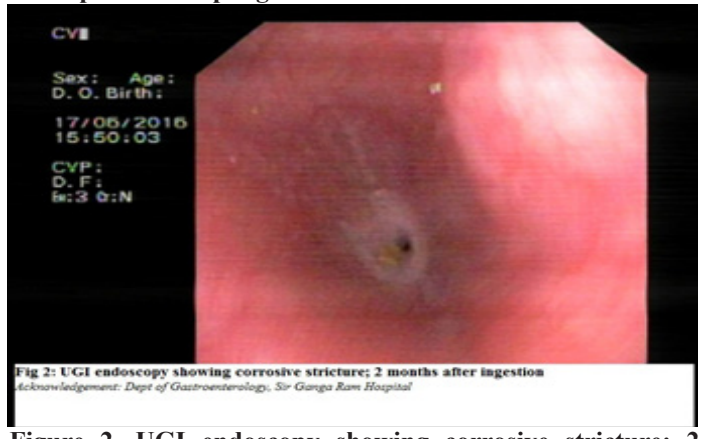


**Figure 2. UGI endoscopy showing corrosive stricture; 2 months after ingestion**

sequelae in the form of cicatrisation, necessitating subsequent endoscopic management. The extent of necrosis did not correlate with the endoscopic grade of injury. This was substantiated further by a report published by Keh et al. suggesting the need for better criteria to guide decision making in these cases[26].

**II. Role of radiological investigations**

A plain radiograph of the chest and upper abdomen has been suggested for detecting pneumoperitoneum or pneumomediastinum, indicating gastric or oesophageal perforation respectively[27]. Contrast X rays using gastrografin or barium has been suggested to confirm the presence of any oesophageal perforation[28]. The choice of the ideal contrast agent is a matter of debate. In the acute setting, gastrografin is the preferred agent owing to its less irritant effect on the mediastinal tissues. In the diagnosis of late sequelae, barium has been shown to be superior owing its better ability to provide radiographic details. Also, aspiration of gastrografin has been shown to have more deleterious effects in the chronic setting compared to barium (Figure 3 & 4).

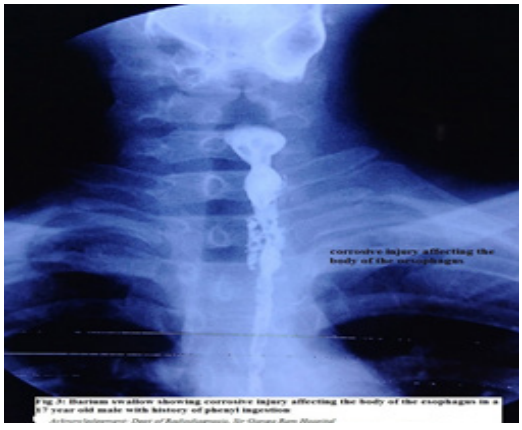


**Figure 3. Barium swallow showing corrosive injury affecting the body of the oesophagus in a 17 year old male with history of phenyl ingestion**

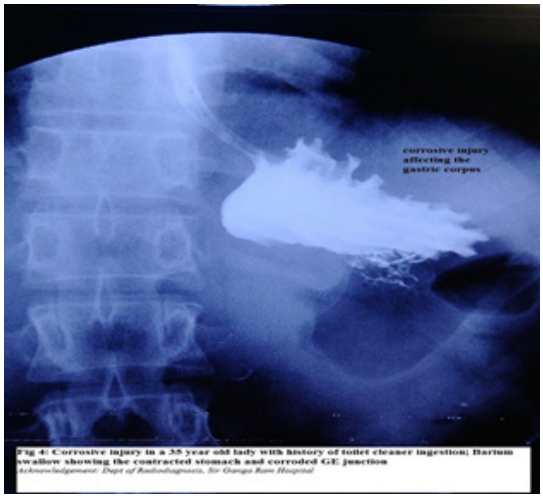


**Figure 4. Corrosive ingestion in a 35 year old lady with history of toilet cleaner ingestion; Barium swallow showing the contracted stomach and corroded GE junction**

Identifying the extent of damage to the muscular layers of the oesophagus on Endoscopic Ultrasonography (EUS) also seems to be a reliable sign to predict the chances of stricture formation in the future[29]. Examination using a radial probe may serve as a guide to assess the response to dilatation. However the definitive role of EUS in corrosive esophageal injuries has yet to be elucidated.

A contrast enhanced CT scan provides a greater anatomical detail of the extent of transmural damage to the oesophagus and stomach compared to early endoscopy. Also, it is better in identifying complications such as perforation. A CT scan with thin layer oral contrast has a definite role in localising the site of an oesophageal perforation in the acute setting. A comprehensive CT grading scheme has been proposed by Ryu et al. to predict the development of oesophageal strictures (Table 2)[30].

**Management of acute corrosive injuries**

This involves immediate haemodynamic stabilisation of the patient as per the ABC protocol of resuscitation.

The important recommendations can be summarised as follows:

- **Establishment of a secure Airway:** 'Blind intubation should be avoided in all circumstances'. Fibreoptic laryngoscopy allows intubation under guidance and is the preferred method of securing an airway in these patients.
- **Establishment of the Circulation:** This is achieved by inserting large bore intravenous cannulae in the emergency setting. Most patients present with severe dehydration due to extensive third space losses and therefore, need immediate volume resuscitation.
- **Role of gastric lavage:** Gastric lavage and emesis are contraindicated due to the further risk of re-exposure of the surface to the corrosive agent thereby aggravating the damage and also, increasing the risk of aspiration.
- **Role of diluting agents:** Administering weak acids in alkaline burns or vice versa is contraindicated. This can generate large amounts of heat due to the exothermic nature of the neutralisation reaction, thereby increasing the damage. Similarly, milk and activated charcoal have no role and are not recommended due to their obscuring the endoscopic

**Table 2. CT grading of corrosive oesophageal injuries (Reproduced from Ryu et al)[30]**

| Grade | Description |
|---|---|
| Grade 1 | No definite swelling of the oesophageal wall |
| Grade 2 | Oedematous wall thickening without peri-oesophageal involvement |
| Grade 3 | Oedematous wall thickening with peri-oesophageal soft tissue infiltration with a well-demarcated interface |
| Grade 4 | Above together with blurring of the tissue interface or localised fluid collection around the oesophagus or descending aorta |

view.

- **Role of nasogastric (NG) tubes:** Blind placement of NG tubes is contraindicated due to the risk of creating false tracts and perforation. However, these can be placed under endoscopic guidance and may act as stents for circumferential burns and also prevent vomiting[31].
- **Role of corticosteroids:** Pelclova et al. carried out a meta-analysis of 10 studies comprising of 572 patients to study the role of steroids in the prevention of stricture formation[32]. They found no significant difference between the steroid treated and the non-treated groups (35.1% vs 33.3%; $p>0.05$). Also, steroids were associated with significant side effects. Therefore, their use is only reserved for patients with severe airway symptoms.
- **Role of proton pump inhibitors:** Though no definite role has been found, a study by Cakal et al. found a superior rate of endoscopic healing among those treated with intravenous omeprazole[33].
- **Role of early surgery:** Emergency surgery is indicated on clinical grounds rather than being guided by the radiological findings. In the presence of gastric/oesophageal perforation, immediate laparotomy followed by gastrectomy or oesophagectomy with cervical oesophagostomy and a feeding jejunostomy is the management of choice[34]. The presence of signs of multi-organ failure and Disseminated Intravascular Coagulopathy (DIC) or severe metabolic acidosis also suggests the need for surgical management[35]. However, these findings usually appear in severe grades of oesophageal injuries (grade 3 or 4) and may already be associated with a high morbidity and mortality.

There is no role of conservative procedures such as simple closure of a gastric perforation[36]. However, radical surgery in the form of oesophagectomy or total gastrectomy have their own attendant complications, when performed in the emergency setting. Minimal resection of the affected organs with a second look surgery is not recommended. An extended resection, including the pancreas or small bowel may be required in some cases. However, an extensive colonic resection may compromise the future graft that would be required for oesophagocoloplasty. In such circumstances, difficult vascular microsurgery for atypical grafts may be required in the future[37].

For perforations into the mediastinal cavity, intercostal drainage tubes are recommended in addition to the above measures.

**Management of chronic oesophageal strictures**

**I. Measures for stricture prevention**

- **Role of systemic antibiotics:** Previous reports have suggested some decrease in the incidence of stricture formation with the use of broad spectrum antibiotics. However, their value in the setting of caustic ingestion with no focus of infection has not been validated by any recent study. If steroids are being used for concomitant airway injury, antibiotics may have a prophylactic role[38].
- **Role of Mitomycin C:** Mitomycin C is an alkylating agent with DNA crosslinking properties. Uhlen et al. have shown its applicability in preventing stricture formation when injected into the oesophageal mucosa or applied topically, though at the expense of deleterious side effects[39]. A recent systemic review by Berger et al. has also shown promising results in long term, but studies determining the optimum dose and the duration of the drug are still lacking[40].
- **Miscellaneous agents:** Anti-oxidants like vitamin E and phosphatidylcholine have been shown to inhibit collagen formation and there is experimental evidence that they decrease stricture rates[41]. Agents like IFN alpha and octreotide have also been studied in this regard[42]. However, they are yet to be validated for use.
- **Role of intraluminal stents:** Patient selection for intraluminal stenting is challenging. Early stent placement within the first month of oesophageal injury is the usual recommendation[43]. Evrard et al. found a relief of dysphagia in 81% of patients using polyflex stents[44]. In another series of 15 patients, Repici et al. found a favourable result in 80% of the patients with the use of these stents after a median follow up of 22.7 months[45]. However, a study by Holm et al. has revealed less optimistic results[46]. Stent migration was the most frequent complication noted (62%), followed by the ingrowth of hyperplastic granulation tissue (17%).

Atabek et al. have shown promising results with the use of Poly Tetra Fluoro Ethylene (PTFE) stents with an around 72% efficacy at 9-12 months of median follow up[47]. Biodegradable stents like polydioxanone and polylactide have been shown to have around 45% success rates with a failure rate of around 10%[48]. However, these are associated with an increased chances of hyperplastic tissue ingrowth.

For the complex hypopharyngeal strictures, 10-12 mm cervical Niti S stent placement with periodic exchanges at intervals of 6 weeks to 3 months is recommended[49].

**II. Management of corrosive strictures (non-surgical)**

- **Role of endoscopic dilatation:** Early endoscopic dilatation is recommended. Delay in dilatation is associated with higher chances of collagen deposition and subsequent scar and stricture formation.

Maloney (bougies), Through the Scope (TTS) balloon and Savary-Gillard (polyvinyl chloride) dilators are among the options available.

The Savary dilators are considered superior to the balloon dilators in old, long and tortuous and fibrotic strictures and may offer a better tactile sensation of dilatation to the operator[50]. Also, Savary dilators exert both radial and longitudinal forces on the stricture unlike the balloon dilators, which exert only radial forces[51]. The usual rate of perforation after dilatation

for benign oesophageal strictures varies between 0.1% and 0.4%[52]. However, this increases up to 32% in case of corrosive esophageal strictures. Balloon dilation may be associated with a 5-8% perforation rate in benign oesophageal strictures, which may increase up to 32% in case of caustic strictures[53]. These rates are variable according to the operator experience. The use of balloon dilators in proximal hypopharyngeal strictures may be associated with extrinsic compression of the larynx leading to airway compromise.

Dilatations are usually carried out at intervals of less than 2-3 weeks, with 3-4 sessions being sufficient for good results[54]. However, this is guided by the pre-dilatation stricture length and diameter. The 'rule of 3', that entails keeping the dilatation diameter up to 3 mm or less at every session should be adhered to[55]. A good nutritional status is essential to ensure a favourable outcome of dilatation and should be ensured by nutritional rehabilitation by the nasogastric or jejunostomy routes[56].

III. Surgical management of corrosive oesophageal strictures

Strictures refractory to dilatation such as those which are long (>10 cm) and tortuous, multiple sequential strictures, or ones in whom 4-5 sessions of dilatation have failed need to be treated by surgery. The basic tenet of surgical management is replacement of the native oesophagus by the stomach or colon. The proximal pharyngoesophageal strictures constitute an a typical scenario and therefore, need to be explained as a separate entity.

The recommendations can be outlined as follows:

- Surgical technique: Resection vs Bypass: In a retrospective review of 176 patients with corrosive oesophageal strictures by Javed et al. 64 underwent resection and 112 underwent bypass, with the native oesophagus left in situ[57]. The authors found no significant difference among the two groups based on intra operative variables and the risk of post-operative complications. The proponents of resection have suggested a higher risk of malignancy in the retained scarred esophagus as well as a high chances of complications like a mucocoele and GER in the latter[58]. However, a study by Gerzic et al. found a higher mortality (11% vs 5.9%) in the resection alone group[59]. The proponents of bypass favoured the same by suggesting a higher morbidity associated with resection due to dense peri-oesophageal adhesions and higher chances of a recurrent laryngeal palsy[57,59].
- Choice of conduit: Gastric vs Colonic: Most of the studies quoted in the surgical literature have shown superior results with the use of colonic conduits in corrosive esophageal strictures. However, proponents of the gastric conduit have suggested better anastomotic healing in the same owing to a more uniform and predictable vascularity of the stomach[60]. The proponents of colonic conduits suggest lower chances of reflux and metaplasia thereby, giving better functional outcomes[61]. Javed et al. reported a lower incidence of conduit necrosis (0.9% vs 10%; p=0.006) and lower mortality (2.8% vs 11.6%; p=0.025) with the use of a gastric conduit[57]. Gupta et al. in their study of 51 consecutive patients showed no statistically significant difference in the rates of anastomotic leak and stricture among the two groups[62].
- Approach: Transhiatal vs Transthoracic: In a series of 51 consecutive patients, Gupta et al. demonstrated a favourable outcome with a transhiatal resection approach in 49 patients[62]. With slow and meticuluous dissection, especially in the upper third of the oesophagus along the anterior aspect, the transhiatal approach has favourable results in expert hands.
- The route of the conduit: The posterior mediastinal route is the longest and the most favourable route of oesophageal transposition. However, in the rare event of sclerosingmediastinitis affecting the posterior mediastinum, other routes such as the substernal or subcutaneous routes may be considered.
- Mid colon oesophagocoloplasty:

Midcolonoesophagocoloplasty is a novel surgical technique, that has gained wide acceptance due to its ease of harvesting and the low rates of conduit necrosis. The graft includes the entire transverse colon with parts of the proximal right and left colon including the territories supplied by the middle colic branches. Ananthakrishnan et al. have shown a very low incidence of conduit necrosis (1 out of 112 cases studied)[63]. This may be attributed to the versatility of the conduit to accommodate to variations in graft vascularity. Because of the sufficient length of the conduit, there is a lower incidence of stenosis and salivary fistula formation. The anastomosis is performed in a wide side-side fashion, thereby avoiding the terminal end of the colon graft to be a part of the anastomosis.

- **Ileocoloplasty vs Coloplasty**

Gerzic et al. have compared ileocolopasty and coloplasty as surgical bypass conduits for corrosive strictures[59]. In their series of 176 consecutive patients with corrosive strictures, they reported a higher incidence of early post-operative complications such as anastomotic leakage, pneumothorax, wound infection and cardiopulmonary complications among the ileoocoloplasty group (50.84% vs 25.64%). Also, the incidence of late post-operative complications such as stenosis of the cervical anastomosis and oesophageal mucocoele was found to be higher in the ileocoloplasty group (33.89% vs 7.69%).

**IV. Management of difficult pharyngo-oesophageal strictures**

Due to the site of involvement and the possibility of associated laryngeal injuries, the proximal esophageal strictures pose a special problem for the surgeon. According to the reports by Wu et al. they comprise around 24.1% of all the corrosive oesophageal injuries[64]. The severity increases with the degree of airway narrowing. Ananthakrishnan et al. studied the role of various therapeutic options for Pharyngo Esophageal Strictures (PES) in a series of 51 patients over a period of 30 years (1977-2006)[65]. In the absence of stricture distal to the pharyngoesophageal junction, a staged procedure in the form of cervical oesophagostomy with

serial dilatations of the PES, followed by a definitive surgery in the form of a pedicled flap using a Pectoralis Major Myocutaneous (PMMC) flap or Sternocleidomastoid flap (SMMC) is suggested.

In the event of a stricture distal to the PES, serial dilatations are recommended subsequent to a cervical oesophagostomy. In the event of undilatable stricture or failed dilatation, an oesophagocoloplasty can be recommeneded in addition to the flap procedures.

Another group of patients may have total laryngeal involvement with a permanent tracheostomy. This group are treated with midcolon bypass procedure with anastomosis to the posterior pharynx (pharyngocoloplasty).

In patients with no demonstrable lumen beyond the PES, the options may be in the form of a permanent gastrostomy or jejunostomy for feeding.

**Follow up**

The corroded oesophagus remains at risk of developing neoplastic changes. Both squamous cell carcinoma and adenocarcinoma may develop at a frequency, about 1000-3000 times more than the general population. The incidence of cancer ranges between 2% to 30%, with an average duration of onset ranging between one to three decades from the primary insult[66]. Bypass procedures do not prevent the risk of developing cancer. Endoscopic surveillance is thereby recommended in all patients.

Another problem that might be observed on a long term follow up is dysmotility. This is more prevalent if the lower 1/3$^{rd}$ is affected, which could be due to the possible damage to the vagus by the cicatrisation process[67]. Gastric emptying time of liquids is significantly affected in caustic injuries to the lower third of the esophagus[68].

## CONCLUSIONS

Corrosive esophageal injuries are on a rising trend, especially in the developing countries. This could be attributed to factors such as lack of education and lower socioeconomic status of our people as well as the ready access to more powerful toilet cleaners. Among the upper sections of the society, factors like psychiatric ailments and resultant suicidal tendencies are important causes. The spectrum of injury varies widely from the time of its initial presentation to the development of late sequelae.

Early endoscopic diagnosis and needful intervention form the primary steps in the emergency setting after haemodynamic and airway resuscitation of the patient. The decision to proceed with emergency surgery is chiefly guided by the clinical parameters and must be weighed against the attendant complications and the associated morbidity and mortality. Nutritional rehabilitation in the form of feeding jejunostomy is of prime importance during the waiting period till the definitive surgery.

Endoscopic serial dilatations of corrosive strictures form the first step in the management of uncomplicated (short segment, middle/lower 3$^{rd}$) strictures. Strictures refractory to endoscopic dilatation necessitate definitive surgery. The decision between resection and bypass is a matter of debate and varies between different centres. However, bypass procedures do not eliminate the corroded oesophagus, thereby leaving behind a nidus for malignant change. Upper oesophageal or pharyngo-esophageal strictures form a specially challenging situation and require a combine approach by gastrointestinal and plastic surgeons.

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