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Review Article

Corrosive Injury of the Upper Gastrointestinal Tract: A Review

Abstract

Ingestion of a corrosive substance can produce severe injury to the gastrointestinal tract and can even result in death. The degree and extent of damage depends on several factors like the type of substance, the morphologic form of the agent, the quantity, and the intent. In the acute stage, perforation and necrosis may occur. Long-term complications include stricture formation in the esophagus, antral stenosis and the development of esophageal carcinoma. Endoscopy should be attempted and can be safely performed in most cases to assess the extent of damage. Procedure related perforation is rare. Stricture formation is more common in patients with second and third degree burns. This review summarizes our current knowledge and evidence based management of this unique but not uncommon pathology of the upper gastrointestinal tract.

Introduction

Worldwide, children represent 80% of the ingestion injury population globally, primarily due to accidental ingestion [1,2]. In contrast, ingestion in adults is more often suicidal in intent, and is frequently life-threatening [3]. Corrosive agents produce extensive damage to the gastrointestinal tract, which may result in perforation and death in the acute phase. Long term complications include stricture formation and development of esophageal carcinoma. Ingestion of corrosives is common in developing countries like India; more so in South India, where there is an easy over the counter access to strong corrosives like aquaregia and bathroom cleaning acid [2,3]. This review attempts to provide an overview of the pathophysiology, clinical features, investigations and evidence based management of corrosive injuries of the upper gastrointestinal tract; which in turn would provide a sound evidence based clinical foundation for the Primary treating doctor, the Gastroenterologist and the Surgeon.

Materials and Methods

A systematic search of the scientific literature was carried out using Medline and Embase for the years 1970–2015 to obtain access to all publications relating to the various aspects of corrosive/caustic substance ingestion. Bibliography of the retrieved studies were also reviewed. The search strategy was with the appropriate specific search terms, like “corrosive ingestion”, “corrosive poisoning”, “caustic ingestion”, “epidemiology & pathophysiology of corrosive ingestion”, “management”, “endoscopy”, “therapeutic endoscopy”, “surgery for esophageal & gastric strictures”, “acute management of corrosive ingestions”, “outcomes”, “mortality”, “morbidity” among others.

Epidemiology and pathophysiology

Ingested corrosives can be broadly classified into alkalis or acids. Alkaline material accounts for most caustic ingestions in Western countries, whereas injuries from acid are more common in developing countries, like India, where hydrochloric acid and sulfuric acid are easily accessible [4]. In southern parts of India, where jeweler

making is a common profession, aquaregia is a common offending agent. Lye is a general term used for alkali found in cleaning agents [2,3]. Solutions with a pH of less than 2 or greater than 12 are highly corrosive. Acids and alkalis differ in their mechanisms of tissue damage. Acids produce coagulative necrosis, with eschar formation that limits further penetration and depth of injury. Alkalis on the other hand, combine with tissue proteins and cause liquefactive necrosis, leading to a deeper penetration into tissues. Additionally, alkali absorption can lead to thrombosis in blood vessels, impeding blood flow to already damaged tissue. Accordingly, alkali ingestion may lead to more severe injury and complications, but this distinction is not clinically relevant in the setting of strong acid or base ingestion, both being able to penetrate tissues rapidly, potentially leading to full-thickness damage of the esophageal/gastric wall. Injury occurs quickly, depending on the agent’s concentration and time of exposure [4-7].

The conventional acceptance is that acids preferentially damage the stomach, due to the protective esophageal eschar. Depending on the concentration of the acid, extensive esophageal damage and perforations can occur even after acid ingestion. Strong acid are also be associated with a higher incidence of systemic complications, such as renal failure, liver dysfunction, disseminated intravascular coagulation and hemolysis. Esophageal injury begins within minutes and may persist for hours. Initially, tissue injury is marked by necrosis with swelling and hemorrhagic congestion. Four to 7 days after ingestion, mucosal sloughing and bacterial invasion are the main findings. At this time granulation tissue appears, and ulcers become covered by fibrin. During this period the wall is at its weakest and perforations may occur if the ulceration extends beyond the muscularis [4-7].

Esophageal repair usually begins on the 10th day after ingestion, whereas esophageal ulcerations begin to epithelialize approximately 1 month after exposure. The tensile strength of the healing tissue is low during the first 3 weeks since collagen deposition may not begin until the second week. Hence, endoscopy is preferably avoided 5-15 days after ingestion. Scar retraction begins by the third week and may

continue for several months, resulting in stricture formation and shortening of the involved segment of the gastrointestinal tract [4-7]. Additionally, lower esophageal sphincter pressure becomes impaired, leading to increased gastroesophageal reflux (GER), which in turn may accelerate stricture formation [4,9]. Reactive oxygen species generation with subsequent lipid peroxidation may contribute either to the initial esophageal injury, or to the subsequent stricture formation [4,10].

Clinical Presentation

The clinical presentation depends upon the type, amount, and physical form of the substances. Solid alkali adheres to the mouth and pharynx producing maximum damage to these areas while relatively sparing the esophagus. Liquid rapidly passes through the mouth and pharynx and produces its greatest caustic effect on the esophagus. Hoarseness and stridor suggest a laryngeal or epiglottic involvement, and may be a harbinger for aerodigestive and high pharyngeal sequelae. Respiratory complications from caustic ingestion may result in laryngeal injury and upper airway edema, which may ultimately require a tracheotomy. Only 10- 30 percent of patients with esophageal burns have no oropharyngeal damage [4,6,11,12].

Symptoms of esophageal involvement include dysphagia and odynophagia, whereas epigastric pain and hematemesis may be manifestations of stomach involvement. Bleeding following corrosive ingestion is usually self-limiting; though massive hemorrhage from the stomach or duodenum has been reported a short time after corrosive ingestion, severe bleeding typically occurs at 2 wk, after ingestion [4,11,12]. However, the absence of pain does not preclude significant gastrointestinal damage. Perforation of the stomach or the esophagus can occur at any time during the first 2 weeks. Hence, any worsening of abdominal pain or the appearance of chest pain should promptly be investigated with a high index of suspicion. No one sign or group of signs is 100% accurate in predicting positive or negative endoscopies [13]. Late sequelae of corrosive ingestion, include stricture formation, gastric outlet obstruction and malignancy involving the injured segment of gastrointestinal tract. Strictures may become symptomatic within 3 months or may even manifest a year later. Ingestion of a liquid agent is most likely to induce stricture formation, which tend to be long. Esophageal carcinoma is a well-known sequel of corrosive ingestion. The latent period between the time of ingestion and the development of carcinoma may be as long as 58 years. There is a 1000-3000 fold increase in the incidence of esophageal carcinoma, and up to 3% of patients with carcinoma of the esophagus may have a history of caustic ingestion [11,12].

Management

Pre hospital measures

Gastric lavage or induced emesis is contraindicated because re-exposure of the esophagus to the corrosive agent may produce additional injury. Milk and water have been used as antidotes but their effectiveness remains unproven, moreover the heat generated by the chemical reaction may increase damage. Milk may also obscure subsequent endoscopy. Activated charcoal is also contraindicated for the same reason [4,14-16].

Initial management

Hemodynamic stabilization and adequacy of the patient's airway are priorities. Airway control is of paramount importance. Apart from it being the cornerstone of resuscitation, airway may most commonly be compromised by laryngeal edema or direct corrosive injury to the laryngeal apparatus. Airway control may be achieved by simple postural maneuvers; in cases of severe compromise, intubation or a tracheostomy maybe indicated [12,14-16]. Fiberoptic laryngoscopy allows intubation under direct visualization, avoiding "blind" intubation with the risk of bleeding and additional injuries [12,17].

Investigations

In the acute phase, a plain chest radiograph may reveal air in the mediastinum or below the diaphragm suggesting esophageal or gastric perforation. To confirm and localize a perforation, water-soluble contrast agents like gastrograffin are used as they are less of an irritant to the mediastinum compared to barium sulfate [4,14-16]. Barium studies may be helpful as a follow-up measure and for the evaluation of complications. It is radio opaque, provides greater radiographic details than water-soluble contrast agents, and has lower risk of aspiration pneumonitis. A CT scan offers a more detailed information regarding the transmural damage and the extent of necrosis [4,18].

Esophagogastroduodenoscopy is considered crucial and usually recommended in the first 12-48 hours after caustic ingestion, though it is safe and reliable up to 96 hours after the injury; gentle insufflation and great caution are mandatory during the procedure [4,12,19-21]. Endoscopy and even dilatation have been performed without consequences from 5 to 15 days after corrosive ingestion; though potentially hazardous due to tissue softening and friability during the healing period. Every attempt must be made to assess the esophagus, stomach, and duodenum provided it can be done safely. Passage of the scope should be limited to the level of the first signs of a circumferential second or third degree esophageal burn [4,12,19-21].

All adult patients must undergo endoscopy after suicidal ingestion, because of the larger amount of more corrosive agents swallowed compared with unintentional injuries. There are no strict guidelines as to who needs endoscopy and who does not [4,20]. Ten to 30 percent of caustic ingestions globally do not show any upper gastrointestinal injury, hence the indication for early endoscopy should be made on a case-by-case basis, with consideration of symptoms, otorhinolaryngeal injuries, and the amount and nature of the ingested substance. [4,12,19-22]. Apart from accurately assessing the degree and extent of the corrosive injury, endoscopy predicts the risk of systemic complications and death; with each increased injury grade correlated with a 9-fold increase in morbidity and mortality [4,12,19-21].

Contraindications to endoscopy are a radiologic suspicion of perforation or supraglottic or epiglottic burns with edema, which may be a harbinger of airway obstruction. A third degree burn of the hypopharynx is a further contraindication for endoscopy [4,12,19-21]. Evaluation of the esophageal wall by endoscopic ultrasound

(EUS) using a miniprobe has been shown to be safe, though it does prolong examination time without showing any difference with endoscopy in predicting early complications [23,24]. The destruction of the muscular layers of the esophagus observed at EUS seems a reliable sign of future stricture formation; furthermore, ultrasound examination with a radial probe may predict the response to dilatation, which usually requires more sessions when the muscularis propria is involved at EUS. In spite of these encouraging reports, the role of US examination in caustic injuries is still under evaluation [23,24].

Correlation between laboratory values and the severity/outcome of injury is poor. A high white blood cell count (> 20000 cells/mm³), elevated serum C-reactive protein, age and the presence of an esophageal ulcer have been considered predictors of mortality in adults; an arterial pH less than 7.22 or a base excess lower than -12 have been considered indication of severe esophageal injury [25,26].

Conservative Management

Oral intake is encouraged in patients whose injuries are graded 1 or 2a. In more severe cases of damage (grades 2 or 3), observation in an intensive care unit and nutritional support is required [4,12,26]. Stricture formation is the most important complication of corrosive damage to the esophagus. Attempts to prevent stricture formation include steroid use, stenting, use of indwelling nasogastric tube, and early dilatation [4,11,12,26].

To date, the efficacy of proton-pump inhibitors and H₂ blockers in minimizing esophageal injury by suppressing acid reflux has not been proven, though an impressive endoscopic healing after omeprazole infusion has been observed in a small prospective study [4,27]. Although animal studies had shown that the use of steroids may decrease the incidence of stricture formation, studies in humans have been inconclusive so far. A metaanalysis of studies between 1991 and 2004, and an additional analysis of the literature over a longer period from 1956 to 2006 did not find any benefit of steroid administration in terms of stricture prevention [4,28,29]. Steroids are usually reserved for patients with symptoms involving the airway. The results of a meta-analysis in 361 subjects from a total of 13 studies produced more encouraging results. Steroids are usually given for at least 3 weeks. Systemic administration of steroids is ineffective in preventing strictures. Intralesional triamcinolone injections have been proposed to prevent strictures, but optimal dose, frequency, and best application techniques are yet to be defined. The use of corticosteroids continues to be a debatable issue [4,26,28,29].

With regard to the use of antibiotics, the data is not very clear. Although in animals, antibiotics have shown to decrease infection in steroid treated esophageal burns, no controlled trials in humans are available. The consensus however appears to be that patients treated with steroids should be treated with antibiotics as well. Prophylactic antibiotics, in the absence of steroid therapy are not advocated [4,26,28].

The insertion of nasogastric tube early in the course of the treatment has been suggested to ensure patency of the esophageal lumen but one needs to be cautious because a nasogastric tube itself can contribute to the development of long strictures and routine use is

not warranted [4,12]. Any esophageal catheterization may be a nidus for infection and nasogastric placement may worsen gastroesophageal reflux, with a consequent delay in mucosal healing. However, enteral nutrition through a nasogastric tube has been demonstrated to be as effective as jejunostomy feeding in maintaining nutrition in such patients, with a similar rate of stricture development [30,31]. Therefore, after caustic injuries the placement of a nasogastric tube may be considered, but the decision should be made with caution and done on a case-by-case basis [4,12,14,31].

Diverse other agents such as Sucralfate, Heparin, Mitomycin C, epidermal growth factor (EGF), Anti-oxidant treatment (vitamin E, H₁ blocker, mast cell stabilizer, methylprednisolone) and caffeic acid phenethyl ester (CAPE) have been shown in animal studies to decrease the incidence of stricture formation but studies in humans are awaited [32-38].

Endoscopic management

Specially designed silicone rubber or polyflex stents have been found helpful in preventing stricture formation but the efficacy is less than 50%, with a high migration rate (25%). Patient selection remains a challenge and the development of hyperplastic tissue is a concern [39-41]. Biodegradable stents (poly-L-lactide or polydioxanone) are under evaluation for benign strictures, with a 45% success rate at 53 months in a patient population with only two caustic strictures, a migration rate of around 10%, and a significant hyperplastic tissue response. Moreover, cost and minimal experience in caustic strictures make the use of biodegradable devices questionable, especially in developing countries [42-44]. Timely evaluation and dilatation of the stricture play a central role in achieving a good outcome. Late management is usually associated with marked esophageal wall fibrosis and collagen deposition, which makes dilatation more complex [7,11,12,16]. Dilatation can be carried out with balloon or bougies (usually Savary) without a clear advantage for each method. However, the failure rate after pneumatic dilatation is higher in caustic ingestion-related strictures than in other benign strictures; Savary bougies are considered more reliable than balloon dilators in consolidated and fibrotic strictures such as old caustic stenosis or in long, tortuous strictures, and may offer the operator the advantage of feeling the dilatation occurring under his hands [45-50].

Dilatation should be avoided from 7 to 21 d after ingestion for the risk of perforation, though early, prophylactic dilatation with bougienage has been reported to be safe and effective even in this period [51]. The perforation rate after dilatation of benign esophageal strictures varies between 0.1% and 0.4%, but for caustic strictures it fluctuates from 0.4% to 32.0%, dropping from 17.6% to 4.5% with increased experience [4,16,45-50]. The interval between dilatations varies from less than 1 to 2-3 weeks and usually 3-4 sessions are considered sufficient for durable results, although the number of dilatations required may be unpredictable and quite high. A cut-off value for unsuccessful dilatation treatment may be difficult to define, especially in developing countries, where alternative surgical options are not widely available. A good nutritional state is crucial for a successful outcome, especially in children, and both an improvement in nutritional status and sustained esophageal patency should be considered reference points for a successful dilatation [4,16,45-50].

In the past, patients with antral stenosis have required surgery, either pyloroplasty or gastroenterostomy. However, some cases may be successfully managed with endoscopic dilatation and this may be attempted prior to surgery [4,16,52].

Surgery

Surgery plays a key role as both an emergency measure and later also in delayed reconstruction. In the acute phase, it is clear that patients with evidence of perforation require immediate surgery [16,53-55]. Patients with shock, acidosis, and coagulation disorders and those who have ingested large amounts of corrosives, usually tend to have severe injury on laparotomy and early surgical intervention may prove beneficial [56].

Early surgery

Patients with clinical or radiological evidence of perforation require immediate laparotomy, usually followed by esophagectomy, cervical esophagostomy, frequently concomitant gastrectomy and even more extensive resections, and jejunostomy feeding may be required [4,16,54]. These injuries also result in metabolic abnormalities such as severe acidosis and dehydration. Loss of the gut mucosal barrier coupled with peritoneal and mediastinal contamination results in severe sepsis. Management of these patients involves urgent resuscitation with correction of fluid and electrolyte and acid-base abnormalities, administration of broad spectrum antibiotics and immediate surgical exploration [4,16,55-59].

Indications for emergency surgery rely more often on clinical grounds than on radiological findings; in the presence of doubtful clinical features a decision to perform laparotomy is likely more advantageous for patients than a conservative attitude especially in patients who ingested large amounts of corrosive substances [4,16,55-59]. Laboratory and endoscopic criteria for emergency surgery have been suggested, including disseminated intravascular coagulation, renal failure, acidosis and third degree esophageal burns. Unfortunately, these are often late findings and surgery may improve mortality and morbidity in grade 3A injuries only. Severe injuries of the stomach at endoscopy require careful monitoring with a low threshold for laparotomy [56]. Conservative management of severe gastric injuries at laparotomy, with partial or total conservation of the stomach, has been recently advocated by some in the absence of clinical and biological signs of severity [56,59]. The need to perform surgery for caustic injuries has a persistent long-term negative impact both on survival and functional outcome.

Diagnostic Laparoscopy acts as an ideal bridge between a formal laparotomy and conservative management. It helps assess the abdominal viscera in patients who have equivocal abdominal findings in a background of features of sepsis. Laparoscopy has been proposed when gastric perforation is highly suspected. The minimally invasive approach has two caveats: unless in very expert hands, it is not a substitute for a comprehensive abdominal exploration, particularly in the posterior aspects of the stomach and duodenum, and it can extend the operative time excessively in a situation where time is a major determinant of outcome. However, it might be considered a useful tool when the stomach cannot be evaluated by endoscopy. Experience is still limited and laparoscopy may be neither feasible nor helpful in such dramatic circumstances [4,53,60]. All injured organs must be

resected if possible, during the first operation. A massive intestinal necrotic injury represents a reasonable limit for resection. Emergency surgery may be required in the case of severe, uncontrolled late gastric bleeding, usually 1-2 wk after ingestion [4,16,55-59].

Late surgery

When esophageal dilatation is not possible or fails to provide an adequate esophageal caliber in the long-term, esophageal replacement by retrosternal stomach or, preferably, a colonic interposition should be considered [61]. Mortality and morbidity are low in expert hands. Unless the esophagus was resected at the time of the initial episode of ingestion, the surgeon has the option of simply bypassing the strictured esophagus and leaving it in situ. In most instances it is possible to bring up the esophageal substitute via a substernal route and perform the proximal anastomosis in the neck or pharynx. Esophageal bypass avoids the need to dissect out a densely scarred esophagus with the attendant risk of injury to the great vessels, thoracic duct, and the trachea or left main bronchus and the inevitable consequence of vagal injury [61-65].

The disadvantage of bypass is that the remaining esophagus is prone to undergo cystic dilation, with occasional rupture. It is inaccessible to endoscopic examination. If it is not disconnected from the stomach, it may be subject to severe acid reflux without the buffering effect of saliva. Finally, the esophagus has an increased risk for cancer after caustic injury. The magnitude of the risk is debated, but it is alleged that the risk is 1000 times that of the general population. It tends to occur many years after the injury, often more than 30 years later [4,16,61-65]. Resection of the esophagus after transmural caustic injury can be a formidable undertaking and an increased mortality as a consequence of attempted resection outweighs the theoretical advantage of reducing the cancer risk. Thoracotomy is usually required because the dense periesophageal scarring, as a result of both the injury itself and possibly superimposed microperforations from numerous dilatations may be difficult and dangerous to resect via the transhiatal route. Hence if esophagectomy is to be performed, it should be done in a high-volume center where experienced surgeons and intensive care is available [4,8,16,61-65].

Choice of substitute

Options for substituting the damaged esophagus include the stomach, colon or the jejunum. Gastric pull-up requires only one anastomosis, is generally quicker, and is increasingly being performed laparoscopically. However, the functional results tend to deteriorate over time with the development of symptomatic reflux, stricture, and columnar metaplasia above the anastomosis in the proximal esophageal remnant [4,8,16,62-67]. In contrast, colon interposition is a more extensive procedure that requires three anastomoses, but the functional results remain stable or improve with time. Colon interposition is also associated with a lower incidence of stricture than gastric pull-up. The stomach may often become unavailable due to intrinsic damage by the caustic agent, leading to scarring and foreshortening [4,8,16,62-67].

The short mesentery of the jejunum generally precludes a jejunal limb from reaching to the cervical esophagus or pharynx. It is best to bring the limb of jejunum into the middle or upper mediastinum

and then bridge the gap by harvesting a free flap of jejunum and anastomosing the artery and vein to the external carotid and jugular vein, respectively. The distal end may be anastomosed to the upper limit of the Roux limb of the jejunum, this is done in a staged manner to let the graft mature and the blood supply develop for several weeks before performing the proximal anastomosis in the pharynx [4,8,12,16,62,67]. Early attempts with pedicled cervical skin flaps were associated with a very high failure rate because of leakage and stricture. A myocutaneous flap harvested from the pectoralis major muscle and based on the pectoral branch of the acromiothoracic artery may be tunneled under the clavicle and sutured into a pharyngeal defect, but this flap is too bulky to be used for a circumferential defect [4,68]. Reconstruction is advisable at the end of the evolving scarring process, usually after 6 mo, although the optimal timing of reconstruction has been reported from 2 months to years [4,8,12,16,62,67].

Gastric strictures

Chronic corrosive gastric injury was classified into the following five types. Type I: short ring stricture of the stomach within one or two centimeters of the pylorus; type II: stricture extending proximally up to the antrum; type III: mid gastric stricture involving the body of the stomach and sparing the proximal and distal parts of the stomach; type IV: diffuse gastric involvement producing a linitis plastica like appearance; and type V: gastric stricture associated with a stricture of the first part of the duodenum [69]. The ideal time for surgical intervention for a chronic corrosive gastric injury is debatable. It is better to postpone surgery resort to jejunostomy feeds to improve the general fitness status and allow the gastric stricture to stabilize. This may take up to several months. This period also enables the mucosal lesions to heal, so that surgical anastomosis can be carried out with greater safety. The preferred operation depends on several factors: (1) the general condition of the patient, (2) the need for a concomitant esophageal reconstruction, and (3) the type of chronic gastric injury [69-71]. In type I gastric injury, a limited resection with a gastroduodenal reconstruction is performed. The strictures are short, and hence the extent of gastric resection required is minimal. The stomach and the duodenum can be brought together in most instances without tension. Type II or III gastric injury is best treated by a distal gastrectomy and an antecolic Polya reconstruction. A retrocolic GJ may interfere with the middle colic arcade and make mobilization of the colon at a later date for esophageal bypass more difficult or sometimes impossible. Type IV gastric injuries can be managed by a total gastric resection. A type V gastric injury that extends into the duodenum or has a separate stricture of the duodenum is more difficult to manage. Resection in such instances involves a major procedure in a patient with poor general condition. Such injuries are best managed by an antecolic dependant gastrojejunostomy. However, besides seriously compromising the general condition of the patient, they are almost always associated with severe esophageal injuries. These strictures are hence treated with a colonic bypass for the esophagus and anastomosing the distal end of the colon end-to-side to the proximal jejunum, leaving the stomach in situ [4,16,69-71].

Pharyngoesophageal strictures

Pharyngoesophageal strictures (PES) raise difficult therapeutic problems due to the site of stricture, the possible association with

laryngeal injury and the presence of downstream esophageal strictures [68]. Reconstruction of a segment of the esophagus distal to the pharyngoesophageal junction and performance of anastomosis at this site is met with better results. Dilatation still remains the first choice therapeutic modality, although the failure rate of dilatation is higher as there is no lumen to enable passage of a guide wire for use of over-the-wire dilators [4,16,68]. When a balloon dilator is used, the extreme proximal nature of the stricture results in the proximal part of the inflated balloon occluding the larynx. This causes acute respiratory embarrassment and necessitates abandoning of the procedure. If a patent segment of the esophagus can be demonstrated below the pharyngoesophageal stricture, an esophagostomy can be established through a right neck approach along the anterior border of the sternocleidomastoid distal to the PES. After a week, a guide wire passed transorally across the stricture exiting through the esophagostomy can be used for Savary-Gilliard dilatation. The stricture is kept open between dilatations by leaving a nasogastric tube to exit out through the esophagostomy. Once the lumen of the pharyngoesophageal stricture is stabilized, an esophagocoloplasty can be done through a left-sided neck approach. If the pharyngoesophageal stricture is the only segment of the esophagus to be narrowed, stabilization of the stricture by dilatation through an esophagostomy can be followed by asking the patient to progressively swallow liquids, semisolids and solids [68,72,73]. Patients with only synechiae between the arytenoids and the posterior pharyngeal wall benefit from repeated excision of the granulation tissue, cauterization, and adhesiolysis under anesthesia [1,12,68,72].

If the pharyngoesophageal stricture is longer and extends up to several centimeters into the cervical esophagus, an island myocutaneous flap may be beneficial. In those with additional distal non-dilatable strictures, the myocutaneous flap inlay which is done by an approach through the right side of the neck can be followed by a second stage esophagocoloplasty through the left side of the neck [73]. Patients who have extensive laryngeal scarring requiring a permanent tracheostomy are easier to manage. Since the risk of aspiration is eliminated they can be treated by a colonic bypass with the proximal anastomosis being made to the lateral wall of the pharynx [74]. Those in whom none of the above is feasible due to an unfavourable anatomy of the stricture and the aerodigestive tract; a permanent feeding jejunostomy or gastrostomy is the only option [71-74].

Conclusion

Ingestion of corrosive substances is increasingly reported in developing countries, due to lack of education and prevention. The relationship between symptoms and severity of injury may be vague, and patients should be carefully monitored, since esophageal or gastric perforations can occur at any time during the first 2 weeks after ingestion. Endoscopy is considered a cornerstone in the diagnosis of corrosive ingestions, yet the indication for early endoscopy should be made on a case-by-case basis. Timely and early surgery may be the only hope for patients with severe injuries, and an aggressive attitude should be considered in such patients. Main late sequelae include esophageal strictures, often accompanied by undernourishment. The likelihood of a gastric outlet obstruction should always be kept in

mind. Endoscopic dilatation is usually successful in achieving a patent esophageal lumen, but in complex strictures several attempts must be carried out. A cut-off value for unsuccessful dilatation treatment may be difficult to define, especially where alternative surgical options are not widely available. Even though mortality and morbidity of esophageal replacement in patients not responding to dilatation are low in expert hands, corrosive strictures are a complex problem which needs a dedicated multidisciplinary team management for successful outcomes.

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