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AN UPDATE ON CAUSTIC GASTROINTESTINAL INJURY WITH MINIMALLY INVASIVE TREATMENT OPTIONS

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Abstract: Caustic injury of the gastrointestinal tract (GI) has remained a costly and devastating problem to the bi-modal population affected, however it has seen encouraging evolutions in its care. Caustic injury is defined as an ingested acid or base that results in a wide range of pathology in the upper GI tract, mainly the upper airway, esophagus and stomach. To yield an update on caustic injuries with succinct recent innovations in care, previously published articles, with an emphasis on more recent articles were searched for using Pubmed and MEDLINE. Major advancements in technology have improved the treatment of the acute phase of injury leading to a more uniform methodology in diagnosis, less morbid acute temporizing measures and improved staging of acute injury with the help of advancements in computed tomography and endoscopic interventions. With the advancement of endoscopy and laparoscopy, acute treatment and chronic complications of caustic injury have been reassuringly improved. Endoscopy and laparoscopy have also aided in therapeutic advancements with the implementation of endoscopic dilation of strictures and minimally invasive techniques for gastro esophageal bypasses and resections. Emerging are biochemical platforms such as bioscaffolds to promote esophageal healing and topical anti-cytotoxic agents to treat esophageal strictures. Treating caustic injury has seen recent advancements with the aid of minimally invasive diagnostic and therapeutic modalities to help in the acute and chronic phases of care as improvements in clinical outcomes with novel biocellular techniques emerging.

Key Words: Gastrointestinal tract, cytotoxic agents, Endoscopy and laparoscopy

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Introduction & Epidemiology

Gastrointestinal (GI) injury secondary to caustic chemical ingestion continues to be a devastating injury to the individual as well as a financial burden to the health care system. Injuries range from mild to catastrophic in the acute and chronic state secondary to alkali or acid ingestion. There is an estimated incidence of

five to fifteen thousand corrosive ingestions per year by the Centers for Disease Control with eighty percent of admissions being in the pediatric population. Caustic GI injuries are considerable and cost nearly twenty-three million dollars to treat annually[1-3]. While the incidence has been decreasing in the United States, other countries, specifically developing countries, have seen a steady increase of injuries per year, resulting in an overall mortality rate of 1.5% [4-7]. Caustic injuries are generally divided into categories of acidic or alkali, and intentional/voluntary or unintentional/accidental ingestion. There is a bi-modal distribution, occurring first in the pediatric population and secondly in the adult population[4,5,8,9]. Accidental ingestion is credited for the majority of cases as it is characteristically associated with the pediatric population (ranging from ages two to five), and related to a bottle mislabeling or accidental household cleaning supply ingestion[3,5,7-9]. Typically in this pediatric population, smaller quantities of chemicals are swallowed thus leading to an overall less complicated disease course [3,5,7,9]. The adult population accounts for the remainder of corrosive chemical ingestions, usually intentionally as an act of suicide[5-7,9,10].

Voluntary ingestion is classically associated with larger quantities of chemical consumption and subsequently causes a more serious degree of injuries with a protracted disease course requiring multiple and continued interventions from chronic complications[5,7,10].

The clinical outcomes from caustic consumption range in severity and depend on the quantity, concentration, contact duration and pH of the chemical ingested [3-5,10-12].

Extremes of acids and bases have been shown to cause the most damage; acids with a pH less than 2 and bases with a pH above 12 [3-5,10,11,13]. Acids, such as hydrochloric acid and cyanide, cause a contact coagulative necrosis along the GI tract with eschar formation that auspiciously forms a protective barrier to prevent deeper injury to the surrounding tissues (table 1) [3-6,10,11,13]. Conversely, basic substances, such as lye and ammonia, cause liquefactive necrosis and saponification allowing the damage to penetrate into deeper tissues and surrounding muscles secondary to ongoing chemical reactions and destruction of tissue-proteins (table 1) [3-6,10-12]. One exception remains in hydrofluoric acid, as it is the one acid that causes liquefactive necrosis and can result in cardiac arrhythmias secondary to calcium electrolyte disturbances secondary to fluoride absorption [13].

The clinical sequela of caustic ingestion ranges from superficial mucosal injuries to full-thickness perforation, hypokalemia, arrhythmias, respiratory distress, sepsis and death in the acute period while long-term survivors can undergo stricture-formation and carcinoma [3-5,8].

Over the years laparoscopic surgery as well as endoscopic interventions have evolved and led to dramatic advancements in the field. These advancements have made it possible to treat both the acute and chronic ailments that are associated with caustic ingestion on a minimally invasive platform sparing the morbidity and pain associated with open operations.

Methods: A search on Pubmed and MEDLINE for keywords including “caustic,” “Corrosive,” “esophageal,” “gastric,” “acid,” “basic,” “base,” “aerodigestive,” “Chemical,” “burn,” “gastrointestinal,” and “substance” was performed for published peer-reviewed articles. Searches were limited to the English language with an emphasis towards more recent articles. Each paper was analyzed for their contributions to the epidemiology, diagnosis, management, and outcomes of caustic gastrointestinal injuries. Both adult and pediatric literature was included.

Diagnosis of Injury: Acute Caustic Injury and Pathophysiology- Acidic solutions cause coagulative necrosis to the lining of the GI tract (one exception being hydrofluoric acid causing liquefactive necrosis) with stronger acids causing more intense injuries. Microscopic examination shows detachment of esophageal epithelium from the lamina propria and thrombosis of intramural vessels in addition to coagulation necrosis [10]. In addition to GI tract

erosion, the unpleasant taste of acidic solutions may limit the quantity of ingestion but will also cause gagging and possible aspiration to the upper respiratory tract with airway compromise[6]. As mentioned, acids form an eschar than can limit injury to deeper tissues in the neck and upper abdomen. However, because it tends to pool in the stomach it predisposes to gastric perforation[6]. Alkali solutions form liquefactive necrosis and typically cause more severe injuries than acids. This is because of a blunted pain reaction secondary to alkali solutions' nerve dissolving properties allowing for larger quantities to be ingested[13]. Since alkalis bind to or pharynx and esophageal tissue their injuries tend to occur proximal to the stomach and gastric perforation/stricture is generally spared. An example of the caustic nature of these compounds is evident in the fact that a 30% solution of caustic soda/sodium hydroxide can cause a full-thickness esophageal injury with only one second of contact time to esophageal tissue. In addition, alkaline disk batteries can cause esophageal perforation if lodged in the esophagus for less than an hour and is a major concern in the pediatric population[13,14]. Injuries to the face, lips and mouth may be obvious but once stabilized evaluation of the oropharynx; glottis, esophagus and stomach are needed to stage the severity of the injury. Obtaining information on the type of substance ingested, amount and timing is paramount[13]. Children commonly present with vomiting however most present with a combination and range of symptoms including nausea, vomiting, stridor, hoarseness and dysphasia along with chest and abdominal pain[6]. Retrosternal chest pain and abdominal pain are often signs that perforation has occurred[13]. Experimental models have shown that pyloric spasm after caustic ingestion limits the corrosive substance from entering the duodenum but causes severe gastroesophageal reflux that extends the contact time to the upper GI mucosa and perpetuates the damage[13,15].

With treating all trauma-related patients, efforts must be made to secure an adequate airway before continued edema to the oropharynx causes upper airway obstruction and intubation is exponentially more difficult. Fiberoptic intubation is often used to avoid injuries to the friable mucosa while simultaneously affording the opportunity to assess the nature of the chemical burn; black slough or gray opaque membranes being acids and alkalis; respectively[4,6,13]. Occasionally airway edema maybe so severe it will require a surgically placed tracheostomy[13]. Volume resuscitation must be initiated as all patients will present with some degree of shock even if only secondary to pain and the sympathetic response[13]. The role of antibiotics and steroids during the acute period have not yet been defined, but can be considered[16]. The use of systemic steroids in the acute period remains controversial; some trials have suggested decreased stricture formation with steroids while others report the opposite findings. A landmark study from the New England Journal of Medicine, published in 1990 concluded no overall difference in stricture formation with the use of steroids in the acute period[6]. Dilution (with water or milk) or neutralization should not be attempted and may in fact only cause more injury secondary to an exothermic chemical reaction with the ingested substance[6,13]. Chest and upright abdominal radiographs should obtained to rule out esophageal/meditational perforation, gastric perforation and the presence of aspiration[6]. Once stabilized, modalities to further evaluate mucosal damage are indicated. Historically, contrast radiographs were used to assess areas of perforation including barium esophogram and technetium-labeled sucralfate, however their false- negative rate can approach 60% and has a limited ability to show the extent of injury[13,14]. Additional limitations of contrast studies lie in the properties of the two agents used; barium and gastrogaffin. Barium can cause a significant inflammatory reaction in the intra-peritoneal cavity if used in the presence of a gastric perforation, yet has minimal pulmonary complications if aspirated. Conversely, while gastrogaffin is well tolerated in the peritoneum it can cause significant pulmonary complications if aspirated[13].

Endoscopy in the Acute Phase

The use and timing of endoscopy in acute caustic injury remains controversial, however most would agree that a burn from a strong alkali substance would mandate endoscopy[1,6,15]. Some advocate for early endoscopy (within 12 hours, and no more than 24 hours) to assess for the extent of damage with a flexible endoscope while others Maintain that computed tomography (CT) could replace invasive endoscopy and avoid the complications associated with the procedure[5,11,13,17]. Nevertheless, endoscopy remains to be considered the goal standard of diagnosis[12].

Although the data is not conclusive, it is generally advised not to perform endoscopy after the first few days because of the evolving inflammatory process resulting in weak, friable tissues that are at increased risk for perforation[5,6]. Caution must be used when approaching full-thickness burns and avoid passing the scope distal to the site of the lesion for fear of exaggerating the injury [5,6,11,13].

The major trepidation of flexible endoscopy is extending the length of a full-thickness injury or converting a partial thickness injury into a full-thickness injury[12].

Additional diagnostic and therapeutic benefits of early endoscopy are to rule out serious injury, which could facilitate admission to an observation unit or even discharge, to direct visually guided placement of a nasogastric tube to aspirate the caustic substance or deploy an esophageal stent[6,13]. Crain et al. analyzed 14 patients with acute caustic injury who underwent acute phase endoscopy to determine the severity of disease. The presence or absence of vomiting, drooling, and stridor along with oropharyngeal burns identified on endoscopy was directly related to the severity of injury. Fifty percent of patients with two or more findings (vomiting, drooling, and stridor) were found to have serious esophageal injury compared to patient with only 1 sign which related to minimal esophageal injury on endoscopy displaying a correlation between signs/symptoms and esophageal injury[18].

In the asymptomatic patient esophagogastroduodenoscopy (EGD) remains a valuable tool. This is especially true in the pediatric population who typically ingest small amounts of chemicals accidently and are usually diagnosed with first-degree injury and thus can be treated conservatively and spared an invasive procedure[4,11].

Conversely, suicide attempts often ingest large quantities of chemicals and therefore present with more serious injuries justifying routine use of endoscopy in that particular high-risk group. Despite its potential drawbacks it remains the most accurate modality of identifying lesions in the esophagus, stomach and duodenum and additionally staging them appropriately to determine treatment strategies[6,8,12].

Accurate staging has been shown to confer prognostic information about stricture development and other long-term complications (table 2)[1,4-6,11,12]. Specifically, grade 3b lesions found on EGD were associated with prolonged length of stay, admission to intensive care unit, and development of additional gastrointestinal and systemic complications[12]. Additional findings of grade 3 lesions show stricture formation exceeding 80% while only a third of grade 2 lesions developed complications such as perforation, pyloric stenosis or regurgitation[12]. While some studies advocate mandatory EGD for alkali burns, Arevalo-Silva et al. reported more extensive mucosal injuries to the esophagus secondary to acidic burns[4]. Additionally, their findings demonstrate a higher proportion of second, third, and fourth degree burns in the acidic group compared to the alkali group as well as a high degree (five out of six patients) of stage four patients requiring operative intervention (gastric pull-up, colonic interposition)[4]. Clearly, though no consensus has been reached, the risks and benefits of EGD need to be evaluated and tailored to each patient.

Surgical Intervention: Surgical intervention in caustic ingestion remains an option for any acute esophageal or gastric perforation. The goals of operative intervention are source control, diversion, debridement of necrotic tissue, drain placement and nutritional support

(feeding gastrostomy or jejunostomy tube). Either diagnosed on physical exam or by imaging, the signs and symptoms may vary but will inevitably include pain and progression to peritonitis or mediastinitis if the perforation occurs in the intra-abdominal or intra-thoracic cavity; respectively. The degree of systemic inflammatory response (SIRS) or septic shock must be addressed and the patient resuscitated accordingly. Timing and extent of injury are considered the two most important pieces of information to determine the operative approach which may range from debridement to esophagectomy[19]. Contained perforations without evidence of extravasations (on contrast imaging) into body cavities and without signs of sepsis maybe treated conservatively with nil per os (NPO), intravenous fluid and broad-spectrum antibiotics. Stable patients with small lesions and/or controlled perforations identified on flexible endoscopy maybe managed with clips, vacuum therapy or stents; all of which have been studied in animal models and show good results in preventing strictures[19,20]. Surgery is reserved for patients who are septic, show evidence of free perforation into the mediastinum/abdomen, or require operative placement of nutritional support [19,21].

The role of laparoscopy remains undefined during the acute phase however in a stable patient it maybe considered A niche for laparoscopy in the acute care algorithm is its ability to ascertain information in the abdomen about 2nd or 3rd degree burns, rule out perforation and place surgical nutrition access minimally invasively (jejunostomy or gastrostomy feed tube)[13]. In the acute period (under 24 hours), in a stable patient, primary repair maybe attempted if the injury is small as long as it is coupled with proximal and distal esophagomyotomy, debridement of non-viable tissues and buttressing from a tissue flap, pleura, muscle, diaphragm or stomach A feeding tube should be placed as well, with either a gastrostomy tube/jejunostomy tube or nasogastric tube for long-term nutritional support. For free perforations in an unstable patient, the placement of interventional-radiology drains for source control coupled with antibiotics maybe the only option and will alleviate the need for surgery temporarily. Many of these patients will still need operative debridement and a definitive operation[19]. The ability to use minimally invasive techniques (laparoscopy and/or thoracostomy) is strictly dependent on the stability of the patient and the comfortability of the surgeon with the modality. Chronic complications of caustic ingestion have been reported to be repaired minimally invasively but their role in the acute process currently remains undefined [22,23].

If esophagectomy or gastrectomy is indicated a traditional approach (laparotomy) or minimally invasive techniques (laparoscopy & thoracoscopy) maybe employed. Transhiatal and cervical mobilization of the esophagus followed by mobilization of the stomach with a proximal esophagostomy, followed by colonic or small bowel graft will be needed[13]. Novel techniques have been described in the acute caustic injury phase to repair the damaged esophagus. An animal dog model was constructed to employ a porcine-derived, xenogeneic extracellular matrix (ECM) from bladder submucosa or small intestine submucosal to repair induced esophageal injury. Surgical defects of approximately 5cm and 40-50% of the circumference were made and repaired with bio-graft and sutured circumferentially. By 60 days the graft was replaced with skeletal muscle, connective tissue, epithelium and was contiguous with the normal esophagus, however all showed stricturing at the graft site [24].

A study out of Argentina treated four patients treated with biologic scaffolds for various indications; one being caustic injury. All patients had a patch porcine urinary bladder ECM sutured to the healthy esophagus. All patients recovered appropriately however one patient developed a microleak that closed spontaneously 2 days after drainage. Follow up barium swallow and EGD showed appropriate emptying and complete mucosal remodeling at 2 months with normal squamous epithelium, however these results also showed stricture with

a 20% area of contraction at the patch level [25].

Chronic Caustic Injury and Pathophysiology- Chronic, or late complications after caustic injury remain a significant morbidity for patients who survive the acute phase. Acute care decisions such as NG tube placement, NPO, nutritional support and endoscopy have been studied to try to identify patients who are at risk for sequelae, hoping a combination of the previously mentioned may prevent long term complications[3,26]. If the caustic injury was secondary to a suicide attempt psychiatric treatment should have been initiated during the acute phase to prevent further attempts which would only exacerbate already existing pathology[13]. Patients who present with multiple signs and symptoms in the acute phase suggesting severe injury are more likely to develop strictures[26]. Stenosis, stricture, dysmotility, adenocarcinoma and rare pathologies such as trachoesophogeal fistulas have developed in caustic injury patients[3,9].

Operative reconstruction is performed to restore normal swallowing function, to treat chronic stenosis or resection in the case of malignancy. Even in the acute setting radical surgery has been attempted with promising results, mainly because of recent advancements in intensive care unit monitoring [27].

Stricture formation is common in the chronic setting and multiple studies have shown that endoscopic intervention with balloon dilation should usually precede operative intervention, including the pediatric population[6,28]. Strictures can be seen in as little as three weeks after the incident with an overall incidence of 10-20% following caustic ingestion in the adult population. These occur most commonly at points of anatomic esophageal narrowing (the level of the cricoid cartilage, aortic arch, distal to the left main stem bronchus and esophageal hiatus)[4,6] (Figure 1). A unique population are children who develop esophageal burns secondary to caustic injury as half of them will later develop structures[6]. Esophageal strictures usually manifest as dysphagia in only 3-4 weeks after the event[29]. Treating esophageal strictures is primarily performed with fluoroscopically-guided endoscopic dilatation with a reported perforation rate of 1.04%[15,28]. The success rate of endoluminal balloon dilatation in the adult and pediatric population (defined as an increased luminal diameter post-procedurally) is reported to be 91-100%, with clinical success rates in 60% of patients[28]. Non-caustic strictures have a lower recurrence rate (14-53%) compared to strictures from caustic ingestion which approach 91%. Dilation for both remains the most appropriate first-line treatment although recurrence rates are considerable. In Doo et al's article, they report primary patency rates (defined as the time from balloon dilatation to dysphagia recurrence) at 0.5, 1, 2, 3, 4, and 5 years were 36, 27, 14, 14, 14, and 14%, respectively[28]. Secondary patency (defined as the total time from the first balloon dilatation until the recurrence of dysphagia after additional endoscopic attempts of dilatation performed since the primary recurrence) rates at 0.5, 1, 2, 3, 4, and 5 years were 82, 82, 82, 56, 42, and 42%, respectively[28]. The secondary patency rate was higher than the primary patency rate with significant value ($p < 0.05$) [28].

Surgical options must be explored for failed endoscopic management of chronic tight/lengthy strictures or the presence of malignant degeneration[30]. For this reason, if initially a surgical feeding tube is placed for nutritional support, a jejunostomy feeding tube is preferred to preserve a gastric conduit. Restoring gastrointestinal continuity is the goal of any operative approach, whether it is from esophagoplasty or resection and grafting which can be from a variety of organs such as the colon, stomach or free-jejunal grafts [13]. Some of the earliest successes to restore food passage was from a right colon interposition anastomosed to the pyriform sinus or a mobilized stomach anastomosed to the pharynx[13]. Though mortality is low from these operations significant morbidity such as graft necrosis, stenosis, empyema, vocal cord paralysis and fistulae have been reported. For these reasons, endoscopic treatment has become the first line therapy in most instances in the

hopes to elude operative morbidity[13,30]. Minimally invasive techniques have become an important part of treatment and a major paradigm shift in care[30]. Though many procedures maybe attempted minimally invasively which can lead to a lower morbidity to the patient, the patient must be fit enough for the hemodynamic changes associated with laparoscopy.

Resection versus bypass has been debated and each has their own risks and benefits whether they are done laparoscopically or with laparotomy. The blind- ended bypassed esophagus can have sequelae of mucocele or abscesses[13]. For this reason a roux limb from the jejunum has been described to allowed esophageal drainage into the GI tract, or destruction of the mucosa with the use of sclerosing agents[13]. If esophagoplasty is indicated to widen a stricture it maybe attempted laparoscopically however the main morbidity is an esophageal leak[13]. If there is a concern for carcinoma, which is present in up to sixteen percent of corrosive injury patients, a formal resection should ensue to avoid malignant degeneration[13]. Considering the risk of malignancy in acute versus chronic injury is paramount as a bypass for a strictured esophagus maintains less morbidity secondary to avoiding the substernal and thoracic dissection necessary for conduits.

If resection is indicated, a number of different grafts or conduits can be used.

Usually an esophagectomy with a gastric conduit is an attractive option because of only one anastomosis, however because of the unique nature of caustic injuries, often times the stomach is also damaged and not suitable as the conduit[13]. Jejunal grafts have been described and often have a robust blood supply however limited length. They do have an inherently higher risk for anastomotic leak compared to a gastric conduit as there are multiple anastomotic sites[13]. Colonic grafts are the most widely used, either from the left or right side of the colon and depend mostly on the most robust blood supply, as length is not usually an issue[13]. The swallowing function after colonic bypass is favorable with up to 92% of patients having adequate swallowing function[13].

Endoluminal Techniques: Endoluminal techniques have been developed to decrease esophageal stricture ratesfrom injury from caustic exposure or after submucosal resection for Barrett's esophagus. Treatment of esophageal strictures in the pediatric population has seen advancements in nonsurgical therapy. Mitocycin-C has been shown to decrease collagen synthesis and scar formation when applied topically and has been used endoluminally for esophageal strictures[31]. Delivery of topical mitomycin-C soaked pledget via combined endoscopic/fluoroscopy has been used in 2 pediatric patients who suffered caustic injury resulting in stricture. Its novel application with pledgets and endoluminal sheaths limited the exposure of mitomycin-C to healthy tissue. Their results showed resolution of the strictures after 4 applications (6-8 weeks apart) with mitomycin-C, however in one patient a long segment in the distal esophagus was excised with gastric pull-up after no resolution with topical treatment[31].

Minimally Invasive Procedures: Minimally invasive esophagectomy with colonic conduit, transhiatal esophago-gastrectomy and thoracoscopic procedures are now being performed after corrosive injury [22,23,32].

A staged total gastrectomy with cervical anastomosis was performed for gastric necrosis in a middle-aged male after hydrocholoric acid ingestion. This was accomplished with laparoscopic exploration with feeding jejunostomy tube, subtotal gastrectomy and partial esophagectomy on initial presentation followed by esophagocoloplasty and cervicotomy six months later[22]. The authors further claim that laparoscopy is a useful diagnostic tool in the acute or chronic stage to assess the extent of intra-abdominal damage[22].

A thorascopic-assisted esophagectomy with laparoscopic gastric pull-up was performed for a young female and on a child after esophageal stricture from caustic injury[23]. A VATS was performed first to expose the thoracic esophagus and circumferentially mobilize it.

The laparoscopic abdominal portion followed by first stapling across a previous gastrostomy site. This was followed by mobilizing the stomach being sure to keep the gastroepiploic arcade intact, and a pyloroplasty laparoscopically. After a Kocher procedure, a gastric tube was formed and a small neck incision was made to pull the stomach caudal. The proximal esophagus was anastomosed with the stomach[23]. Javed et al reported on 39 patients treated with either an open or minimally invasive procedure consisting of gastric or colonic pull-through with the laparoscopic group having requiring significantly less analgesic medication. The colonic mobilization, retrosternal tunnel and delivery of colon into the neck were all accomplished laparoscopically, followed by a cervical pharyngo/esophagocolic anastomosis[32].

Conclusion: Caustic injuries caused by chemical ingestion are rare but require meticulous acute and chronic management. Their incidence is decreasing in the United States but remains a significant treatment challenge with high financial costs to the healthcare system. Burns with acid or alkali cause a different pathogenesis of disease but both can cause mild to severe injury with GI perforation, sepsis and death[3]. Acute treatment involves fluid resuscitation, antibiotic administration and radiographic imaging with or without the use of flexible endoscopy. Mild injury can be treated with careful follow up but assessment at some point with endoscopy is recommended even though the optimal timing is still under debate[13]. Contained perforations can be treated conservatively in stable patients however surgical intervention maybe needed for source control, the placement of feeding tubes or even resection. Strictures, outlet obstruction and dysmotility are all long-term complications of caustic ingestion and are first treated with endoscopy. If endoscopy fails or if carcinoma is feared, resection is indicated whether in the adult or pediatric population. A variety of conduits have been used with success in esophagectomy patients and recently more of these procedures have been performed in a minimally invasive manner with laparoscopy and thorascopy.

Laparoscopic intervention has been demonstrated to be feasible and safe for both acute injury to further diagnose intra-abdominal disease and chronic injury for a minimally invasive approach for esophagoplasty or resection. Although new, laparoscopy has documented success and may become part of future algorithms in treating caustic ingestion.

Table 1-Common Acidic & Alkali Solutions

Acids	Common Acidic Products	Alkalis	Common Basic Products
Sulfuric Acid	Surface household cleaners	**Hypochlorite**	Oven & liquid drain cleaner
Hydrochloric Acid	Rust remover	**Sodium hydroxide**	Disk battery
Nitric Oxide	Swimming pool cleaner	**Ammonia**	Dishwater detergent
Salicylic Acid	Aspirin	**Calcium hydroxide**	Ammonia cleaning product

Table 1 showing common acidic and alkali solutions and their commercial products that contain the caustic solutions.

Table 2- Endoscopic Grading System for Corrosive Injuries (14)

Grade	Findings
0	Normal
1	Mucosal edema and hyperemia
2a	Friability, erosions, hemorrhage, blisters, white-membranes, exudates, superficial ulcers
2b	Grade 2a + deep discrete or circumferential ulceration
3a	Small areas of scattered necrosis
3b	Extensive necrosis

Table 2 showing a grading system for corrosive injuries that range from normal (0) to extensive necrosis (3b) of the organ involved.

Figure 1

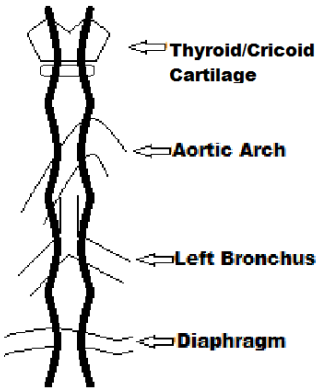

Figure 1- Showing the multiple sites of anatomic narrowing of the esophagus which correlate to the most common locations of esophageal stricture after caustic injury.

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