Pyloric resection with Billroth 1 gastroduodenostomy versus bypass loop gastrojejunostomy for the management of post corrosive long segment pyloric stricture: a retrospective study

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Background
Accidental or suicidal ingestion of corrosive substances remains a major public health problem. Worldwide, the causative materials for corrosive injury are not the same. Surgery remains the mainstay of treatment with available different surgical options. The selected surgical technique should offer symptomatic treatment with less postoperative morbidity.

Patients and methods
Retrospective evaluation of 62 patients with long segment pyloric stricture postacid ingestion presenting with gastric outlet obstruction in the period between May 2017 and June 2022. They were divided into two groups, (group A (n=24), managed by Billroth 1 gastroduodenostomy; group B (n=38), managed by bypass loop gastrojejunostomy).

Results
In group A, the age ranged 3–59 years with a mean age of 25.7±22.62 years, while in group B, the age ranged 2.5–63 years with a mean age of 19.72±15.71 years. The type of ingested corrosive was hydrochloric acid in 60 patients (96.77%), while in two (3.23%) patients the exact acid was unknown. The interval between initial acid ingestion and symptoms of gastric outlet obstruction was 3 weeks to 18 months. In group A, the mean operative time was 64.125±7.023 min, while in group B, it was 61.763±7.837 min. Postoperative vomiting occurred in three (12.5%) patients in group A and in six (15.78%) patients in group B. In group A, the mean hospital stay was 5.5±0.932 days, while in group B, it was 4.921±1.806 days.

Conclusion
Billroth 1 gastroduodenostomy has the superiority of prophylaxis against long-term low risk of future malignancy. Bypass loop gastrojejunostomy is a safer alternative to Billroth 1 gastroduodenostomy, especially in the presence of severe perigastric adhesions and malnutrition. Future studies should include a large sample of patients with long periods of follow-up.

Keywords:
acid ingestion, Billroth 1 gastroduodenostomy, corrosive, gastric outlet obstruction, gastrojejunostomy, long segment, pyloric stricture

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Introduction

Ingestion of caustic substances whether accidental or with suicidal intent remains a major public health problem. The main contributors to these injuries are unrestricted sales with a lack of legal regulations [1]. These materials are easily accessible at home as it is used for toilet cleaning, soaps, bleaches, and disc batteries. The major risk factor for accidental ingestion by young children aged less than 5 years is the storage of these caustic materials in water or soft drink containers at accessible places at home [2,3]. Accidental ingestion by children accounts for 68–80% of cases worldwide. Suicidal intent, patients with psychiatric illness, and alcoholics account for the remainder of cases [4,5].

Worldwide, the causative materials for corrosive injury are not the same. Oral intoxication with caustic materials occurs after ingestion of alkalis (sodium and potassium, soaps, detergents), acids (sulfuric, hydrochloric, lactic, acetic, carbolic, oxalic), heavy metal salts, iodine tincture, formalin, and more other chemical caustic materials. The alkali sodium hypochlorite which is used in household bleach was the most frequently implicated caustic material according to the 2013 American Association of Poison Control Centers annual report with similar reports from European countries [6,7]. In developing countries like Egypt, Yemen, and India, caustic injury is caused by acids which they are cheaper

in comparison to alkalis and commonly used for toilet cleaning and disk batteries [8,9].

Caustic injury is followed by local reaction that may proceed to swelling, and necrosis up to perforation [10]. The injury mechanism is different for both acids and alkalis. Several factors judge the extent of injury like concentration, volume, corrosive type, duration of exposure, and viscosity of the corrosive substance. The total volume ingested is an important factor for evaluation of corrosive injury and it is considered massive ingestion when volume of 100 ml is ingested. Viscosity also has an important role in the degree of corrosive injury, as contact duration is increased with more viscid materials leading to more extensive trauma [7–9].

Acids are painful to the mouth than alkalis, so large volume of alkalis is ingested in comparison to acids. Acids are less viscous with lower specific gravity, so they are rapidly cleared from the esophagus to the stomach. Because of corrosive-induced pylorospasm, they stay at the prepyloric area with prolonged duration of contact with more extensive injury to the pylorus and for that reason the stomach is more frequently affected by acids than alkalis; however, gastric injury post alkali ingestion has also been reported. Acids, except for hydrofluoric acid, cause coagulative necrosis which decrease the incidence of perforation due to coagulum formation that limits further tissue penetration, while hydrofluoric acid leads to liquefactive necrosis with electrolyte imbalance due to fluoride absorption [7–9].

Corrosive alkalis cause tissue injury by progressive liquefactive necrosis which may extend over many days up to two weeks. In esophagus, this increases the incidence of perforation followed by periesophagitis with damage to nearby organs like the respiratory tract [7–9]. Ingestion of alkaline disk batteries mandates immediate removal as tissue damage occurs in just two hours while perforation will occur within 8-12 h as they contain a 45% solution of potassium hydroxide or sodium hydroxide [11,12]. The mucosa of the esophagus is more resistant to the injurious effects of acids than alkalis, as corrosive alkalis are often more thick with a longer contact time and also produce liquefactive rather than coagulative necrosis [13,14]. Following corrosive ingestion, the tissue injury goes into three phases; phase 1 lasts for 24–72 h and is characterized by cell necrosis, phase 2 lasts for 3–12 days and is characterized by mucosal breakdown and fibroblast colonization with granulation, and phase 3 that begins 3 weeks after the initial injury and extend up to 3–6 months and is characterized by stricture formation [15].

After initial caustic ingestion, esophageal repair usually begins on the 10^{th} day whereas esophageal ulcerations begin to epithelialize approximately 1 month after exposure. Esophageal stenosis may be seen as early as 3 weeks after ingestion and this usually presents with dysphagia and odynophagia [16,17]. It occurs in 10% to 20% of corrosive injuries, mostly at areas of anatomical narrowing of the esophagus commonly at the level of the cricoid cartilage, the aortic arch, below the left main bronchus, and at the lower esophagus at the esophageal hiatus [18]. Approximately 80% of strictures will cause dysphagia 8 weeks after their formation [19]. 30% of children suffering corrosive alkali ingestion will develop esophageal burns; of these, 50% will develop strictures [12].

Acute gastric corrosive injury may be manifested by upper abdominal pain, nausea, vomiting, and hematemesis which is usually self-limiting: though massive gastric or duodenal bleeding had been reported up to 2 weeks postcorrosive ingestion [10,20]. Late sequence of caustic gastric injury can be classified into the following five types; type I: short segment pyloric stenosis; type II: stenosis 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 [1].

Corrosive gastric injury management depends on many factors as time of presentation post injury, the patient general condition, concomitant esophageal injury, and type of gastric involvement. Surgery remains the mainstay of treatment for gastric corrosive strictures [21], even though good results have been reported with balloon dilatation [22]. The various treatment options include endoscopic balloon dilatation of strictures, feeding jejunostomy to improve patient general condition prior to surgery, gastrojejunostomy with or without vagotomy, pyloroplasty, and antrectomy with Billroth I anastomosis [23]. The selected surgical technique should offer symptomatic treatment with less postoperative morbidity. We conducted this study to present our experience and to compare Billroth 1 gastroduodenostomy and bypass gastrojejunostomy for the management of postcorrosive long segment pyloric stricture after acid ingestion.

Patients and methods

This retrospective study was conducted at Ain Shams University Hospitals in Egypt and Saudi Hospital in Hajja city, Yemen. After approval of the ethical committee, 62 patients with long segment pyloric stricture postacid ingestion in the period between May 2017 and June 2022 were retrospectively evaluated. They were divided into two groups, (group A (n=24), managed by Billroth 1 gastroduodenostomy; group B (n=38), managed by bypass loop gastrojejunostomy).

Inclusion criteria

All patients with long segment pyloric stricture postcorrosive acid ingestion presenting with gastric outlet obstruction.

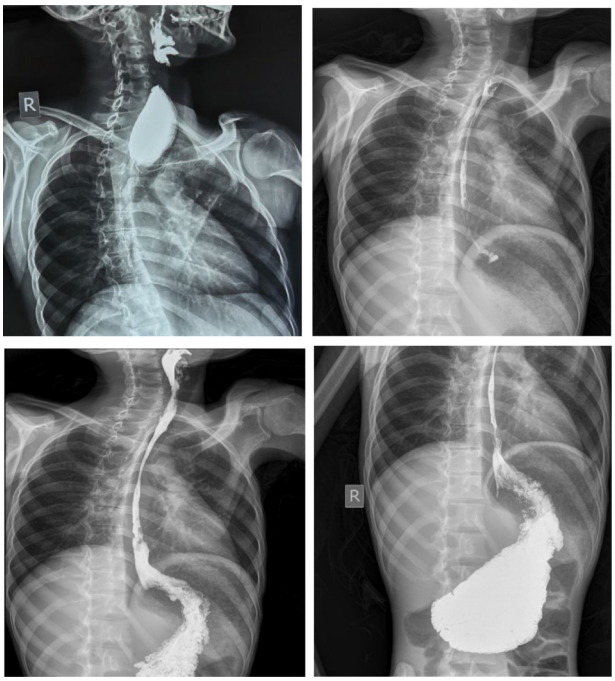
Exclusion criteria

Patients with associated esophageal strictures not responding to balloon dilatation, all other forms of corrosive gastric cicatrisation like short segment pyloric stenosis, mid gastric stenosis, hourglass deformity, proximal gastric stricture near the oesophagogastric junction, total gastric involvement, and gastric stricture associated with duodenal stricture.

History, physical examination and laboratory investigations were carried out for all patients. Diagnosis was made mainly by history of acidic corrosive ingestion, and it was confirmed by gastrograffin swallow (to detect associated esophageal stricture (Fig. 1)) and gastrograffin meal study to diagnose gastric injury and to delineate the extent of gastric cicatrisation (Fig. 2). Upper GI endoscopy was not routinely done in our unit as some patients were referred to us after failed endoscopic dilatation, while in the remaining patients the diagnosis was clear by contrast study which revealed complete gastric outlet obstruction, and endoscopy was spared only for patients with associated esophageal stricture who are in need for serial esophageal balloon dilatation.

Patients were either referred from pediatric or medical departments after failed endoscopic dilatation, or they were presented to emergency department and then they were admitted for resuscitation, rehydration and correction of abnormal electrolytes, low serum albumin, anemia if present, and preparation for surgery. Initial nutritional management was partial TPN for improvement of the nutritional status before surgery. No preliminary feeding jejunostomy was done in this study. We included only patients

Figure 1

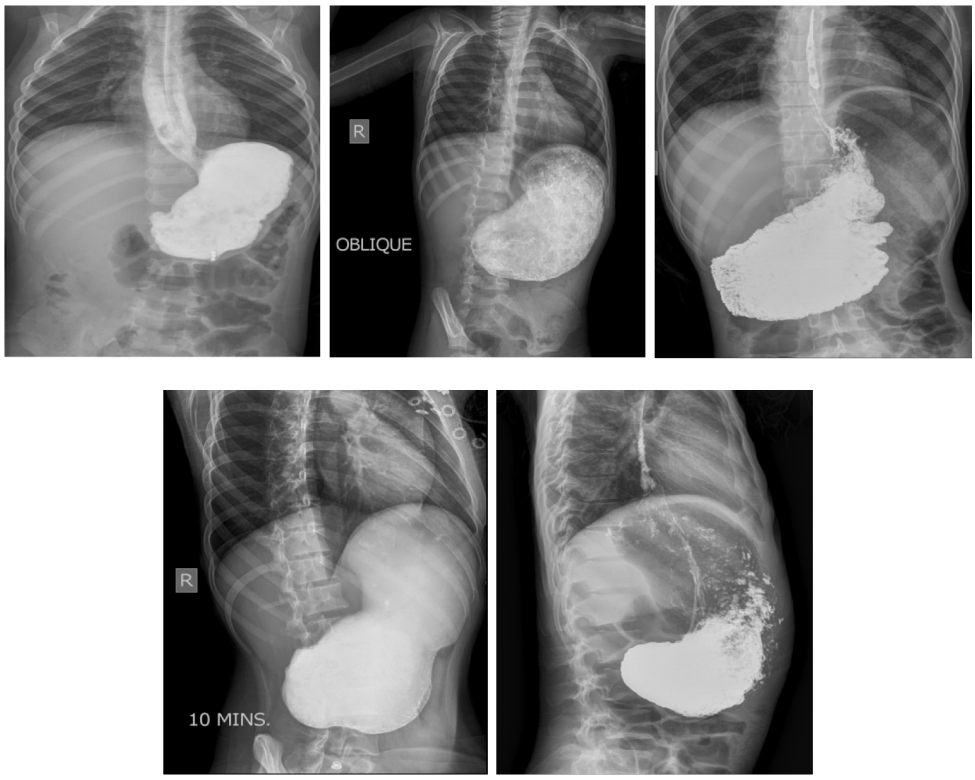


Contrast swallow and meal study for patient with esophageal stricture associated with pyloric stricture.

with long segment pyloric stricture (Fig. 3), and all other forms of corrosive gastric cicatrisation were excluded. All patients underwent an open surgery via upper midline incision followed by evaluation of the stenotic segment and the remnant gastric volume and perigastric adhesions if present. In group A, resection of the stenotic segment with Billroth 1 gastroduodenostomy with hand sewn reconstruction was done (Fig. 4). In group B, patients underwent hand sewn retro colic isoperistaltic loop gastro-jejunostomy starting 25–30 cm after duodenojejunal flexure followed by side to side anastomosis between the afferent and efferent jejunal loops to guard against reflux biliary gastritis (Fig. 5).

Medical records were retrospectively evaluated for patients with gastric outlet obstruction operated for long segment pyloric stenosis post corrosive acid ingestion. We reviewed patient related information like age, sex, intent of ingestion whether accidental or suicidal, nature of corrosive ingested, duration between time of ingestion and symptoms of gastric outlet obstruction, pre-operative management and resuscitation given to the patient, presenting symptoms, nutritional status, indications for surgery and type of surgical procedure, intraoperative findings, operative time, intraoperative difficulties, and postoperative course and complications. Patients were followed-up for at least 1.5 years postoperative, for early and late complications.

Figure 2

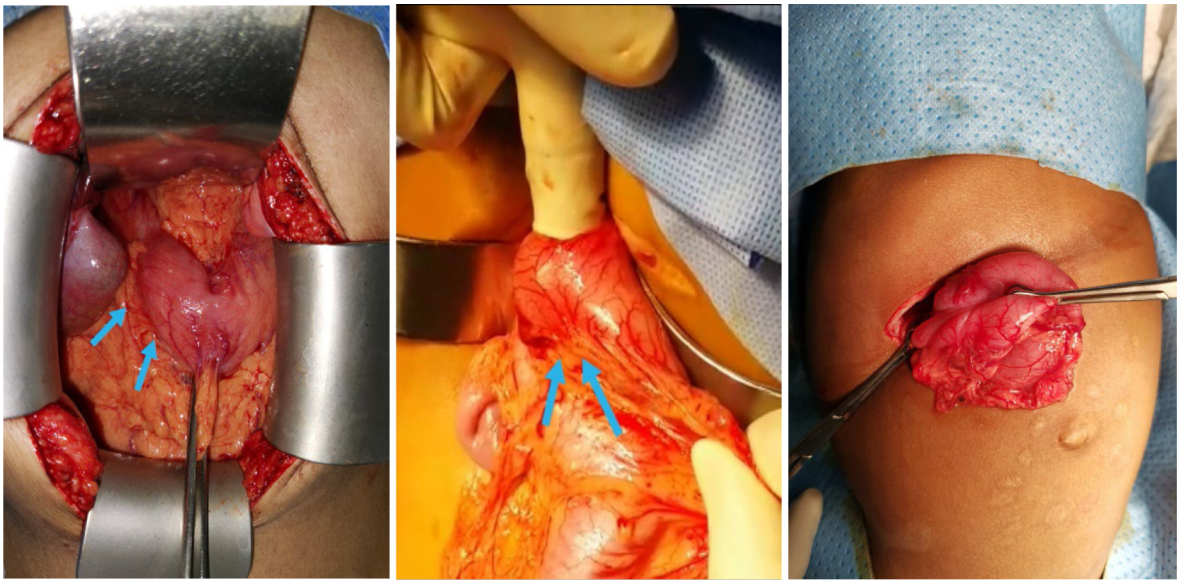


Contrast meal study for different patients with postcorrosive gastric outlet obstruction.

Statistical analysis

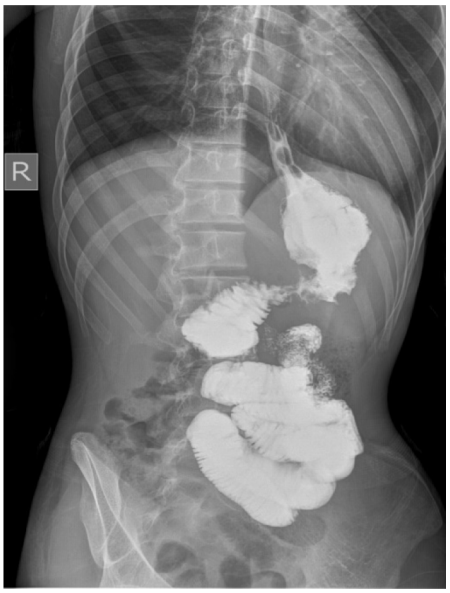
Continuous data were presented as mean±standard deviation (SD). Categorical data were presented as percentages. Analysis of variance (ANOVA) and the rank-sum test were used to analyze continuous data. The results were regarded significant (S) with P less than 0.05 and highly significant (HS) with P less than 0.01. P greater than or equal to 0.05 was regarded nonsignificant (NS). χ^2 test (with Yates correction and Fisher's exact test) and the Student t or

Figure 3



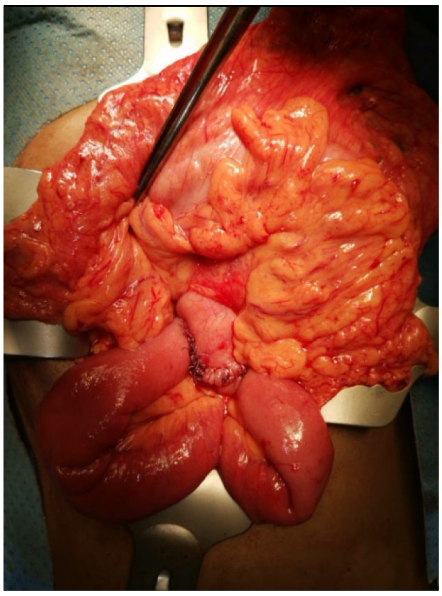
Long segment corrosive pyloric strictures.

Figure 4



Contrast study postpyloric resection with Billroth gastroduodenostomy.

Figure 5



Retro colic isoperistaltic loop gastro-jejunostomy.

Mann–Whitney *U* tests were used according to the characteristics of the study variables and the conditions of applicability.

Results

In this study, 62 patients with long segment pyloric stricture post acid ingestion in the period between May 2017 and June 2022 were retrospectively evaluated. They were divided into two groups, (group A (n=24), managed by Billroth 1 gastroduodenostomy; group B (n=38), managed by bypass loop gastrojejunostomy). The study included 46 (74.2%) females and 16 (25.8%) males with no statistically significant difference between both groups regarding sex (Table 1). In group A, the age ranged 3–59 years with a mean age of 25.7±22.62 years, while in group B, the age ranged 2.5–63 years with a mean age of 19.72±15.71 years, (P=0.131) (Table 2).

Table 1 Patient sex, corrosive type, intent of ingestion, and interval between ingestion and presentation with gastric outlet obstruction

Item	Distribution	Number (Percentage)
Sex	Female	46 (74.2%)
	Male	16 (25.8%)
Corrosive type	Hydrochloric acid	60 (96.77%)
	Unknown	2 (3.23%)
Intent of ingestion	Suicidal	19 (30.65%)
	Accidental	43 (69.35%)
Interval between ingestion and gastric obstruction	3–6 weeks	56 (90.32%)
	12 months	4 (6.45%)
	18 months	2 (3.23%)

The type of ingested corrosive was identified in 60 (96.77%) patients and it was hydrochloric acid used as bathroom cleaner, while in two (3.23%) patients the exact acid was not identified. The intent of acid ingestion was suicidal in 19 (30.65%) patients, while in 43 (69.35%) patients, it was accidental especially in pediatric ages, and those with mental and psychiatric illness.

In this study, the commonest presenting symptoms in patients with gastric outlet obstruction were nausea, postprandial abdominal distension, epigastric pain, non-bilious persistent vomiting, dehydration, constipation, progressive weight loss, and hematemesis with anemia in some patients. In one (1.61%) patient with associated esophageal stricture, there was also dysphagia, chest pain, odynophagia, and vomiting. The interval between initial acid ingestion and symptoms of gastric outlet obstruction was 3–6 weeks in 56 (90.32%) patients, four (6.45%) patients were presented after 1 year, and 2 (3.23%) patients were presented after 18 months.

In group A, the mean operative time was 64.125 ±7.023 min, ranging from 61 to 95 min. In group B, the mean operative time was 61.763±7.837 min, ranging from 49 to 79 min without statistical significant difference between both groups (P= 0.111) (Table 2). Neither mortality nor

Table 2 Age, operative time, hospital stay, and postoperative complications

Patient characteristics	Group A Billroth 1 gastroduodenostomy *N*=24	Group B Loop gastrojejunostomy *N*=38	*P* value
Age			
Mean	25.7±22.62 years	19.72±15.71 years	0.131
Range	3–59 years	2.5–63 years	
Operative time			
Mean	64.125±7.023 min	61.763±7.837 min	0.111
Range	61–95 min	49–79 min	
Hospital stay			
Mean	5.5±0.932 days	4.921±1.806 days	0.051
Range	4 to 7 days	3 to 13 days	
Chest infection	2 (8.33%)	3 (7.89%)	0.476
Postoperative vomiting	3 (12.5%)	6 (15.78%)	0.360
Surgical site infection	1 (4.16%)	2 (5.26%)	0.422
Postoperative thrombocytopenia	2 (8.33%)	4 (10.52%)	0.387
Fever of unknown origin	1 (4.16%)	3 (7.89%)	0.271

thrombotic complications were encountered in both groups. Pulmonary complications occurred in two (8.33%) patients in group A and in three (7.89%) patients in group B ($P=0.476$), in the form of chest infection with minimal pleural effusion which responded well to chest physiotherapy, antibiotics, mucolytic, and bronchodilators without any need for ventilatory support.

Postoperative vomiting either because of paralytic ileus or anastomotic edema occurred in three (12.5%) patients in group A and in six (15.7%) patients in group B ($P=0.360$). Vomiting delayed starting postoperative oral feeding; it stopped with conservative treatments without any need for surgery or endoscopy. One (4.16%) patient in group A and two (5.26%) patients in group B ($P=0.422$) had wound infection which caused slight increase in the hospital stay. It was managed by removal of some stitches plus antibiotics according to result of culture and sensitivity in addition to regular dressing.

Two (8.33%) pediatric patients in group A and four (10.52%) pediatric patients in group B ($P=0.387$) developed postoperative thrombocytopenia with a platelet count less than 100×10^9 cells/l with wound and anterior abdominal wall ecchymosis, it required platelet transfusion and patients were discharged home after increase of the platelet count. One (4.16%) patient in group A and three (7.89%) patients in group B ($P=0.271$) were presented after discharge with fever of unknown origin which subsided with antibiotics and antipyretics. In group A, the mean hospital stay was 5.5 ±0.932 days, ranging from 4 to 7 days. In group B, the mean hospital stay was 4.921±1.806 days, ranging from 3 to 13 days without statistical significant difference between both groups ($P=0.051$).

All patients gained weight, and hypoproteinaemia was corrected following surgery. None of our patients developed anastomotic leakage or intra-abdominal collection or adhesive bowel obstruction or incisional hernia, or had clinical recurrence of gastric outlet obstruction in the follow-up period. Patients who had bypass gastrojejunostomy had upper GI endoscopy with biopsy from the stenotic segment every 6 months for the first year and then every year to assess the stricture to rule out any malignant change. All biopsies were benign during the follow-up period. Three (7.89%) patients in group B, who had bypass gastrojejunostomy, had stomal ulceration which healed with medications. None of the patients showed any manifestations of anastomotic stenosis during the period of follow-up. One patient with associated esophageal stricture was under frequent endoscopic dilatation, he was presented later to ER with food bolus impaction (piece of meat), which was successfully removed via upper endoscopy, this patient underwent esophageal resection with stomach pull up later with smooth postoperative course.

Discussion

Ingestion of caustic materials has a serious public health impact worldwide. In low income countries, its incidence is more frequent than other countries and also it is less frequently reported [10]. Caustic injuries of the stomach and esophagus are classified pathologically similar to thermal skin burn degrees, where erythema and minimal tissue damage are the features of the first degree followed by mucosal regeneration without scar or stricture manifestations. In the second degree, the corrosive substance penetrates deeply through the submucosal layer into the muscular layer followed later by granulation tissue

formation and fibroblast reaction and contraction resulting in strictures which are common at areas with anatomical narrowing in the esophagus and at the antrum and pylorus of the stomach [17]. Deep penetration with perforation of the viscus characterizes the third degree of injury.

Acids, except for hydrofluoric acid, tend to form a coagulum after mucosal injury which limits the further injury into the deeper tissues of the affected organ. Gastric injury due to acids is more common than alkalis, however, it had been reported that alkalis also may cause gastric injury. Deep penetration with perforation characterizes alkalis as they are thicker with prolonged mucosal contact time leading to progressive liquefactive necrosis. The volume and consistency of the ingested corrosive determine the extent, location, and the degree of injury [15]. The lower pharynx and upper esophagus are more injured by powder or crystal form as they adhere to the mucosa with prolonged contact time in comparison to liquid form which is more injurious to the body and lower end of the esophagus and stomach. Corrosive damage is more when the caustic substance is swallowed with suicidal intent especially with an empty stomach [1].

After ingestion of caustic material, patients may present with oral and throat pain and ulcers with drooling of saliva, nausea, vomiting, chest pain, dysphagia, odynophagia, hematemesis, and signs of perforation with mediastinis or peritonitis [24]. The need for an emergent intervention has a persistent long-term negative impact both on morbidity and mortality [10].

After resolution of the acute injury, patients may present later with dysphagia or persistent vomiting with weight loss due to stenosis at the esophagus or at the stomach resulting in gastric outlet obstruction. Patients may have also symptoms of gastroesophageal reflux, and esophageal or gastric carcinoma manifesting many years later after caustic ingestion (esophageal carcinoma occurs in perhaps 3% of patients; gastric carcinoma is very rare) [25,26]. The risk for development of esophageal stricture is increased with strong caustic material [27]. Chronic gastric caustic injury can be in the form of short or long segment pyloric or antral stricture up to diffuse gastric injury. Results of endoscopic dilatation are mostly unsatisfactory in the majority of patients and they will need surgery later [1]. After resuscitation and improvement of the patient bad parameters, the choice of definitive surgical procedure regarding resection and reconstruction should be tailored according to the general condition of patient, associated esophageal injury, and the pattern of gastric involvement [21].

In this study, 62 patients with long segment pyloric stricture were divided into two groups, (group A (n=24), managed by Billroth 1 gastroduodenostomy; group B (n=38), managed by bypass loop gastrojejunostomy). The study included 46 (74.2%) females and 16 (25.8%) males with more female predominance similar to the study conducted by Chibishev and colleagues with 75.76% female predominance [28], and by Ray and Chattopadhyay who reported that 57% of patients were females [29], unlike male predominance in the study conducted by Thomas *et al.* (61 males; 17 females) [30].

In group A, the age ranged 3–59 years with a mean age of 25.7±22.62 years, while in group B, the age ranged 2.5–63 years with a mean age of 19.72±15.71 years, (P=0.131). In the study conducted by Turner and Robinson, patients had a median age of 2.6 years (range: 11 months–18 years) [31]. Gupta and colleagues reported ages ranged from 16 to 72 years [32]. Ray and Chattopadhyay reported age incidence between 17 and 52 years with age range 20–40 years in 65% of patients [29]. Arévalo-Silva and colleagues documented that there was a biphasic age distribution with half of patients were children less than 5 years old and the remaining were adults (range: 5 months–71 years) [20].

The type of ingested corrosive was identified in 60 (96.77%) patients and it was hydrochloric acid used as bathroom cleaner, while in two (3.23%) patients the exact acid was not identified. In a descriptive-analytical study by Balderas and colleagues, they reported that the caustic material was acid in 41% (hydrochloric acid 36.8%) and alkaline in 59% of patients (sodium hydroxide 41.4%) [33]. Chibishev and colleagues noted in their report that the caustic material was hydrochloric acid in 63.64%, NaOH in 27.27%, and CH3COOH in 9.09% of patients [28]. Havanond and Havanond reported that a toilet disinfectant containing a strong acid was the substance ingested in 62% of cases [34]. Rodriguez Vargas and colleagues reported that ingested caustic substances were the following: bleach (sodium hypochlorite) in 78% of cases, hydrochloric acid in 20% of cases, and caustic soda in 2% of cases [35].

The intent of acid ingestion was suicidal in 19 (30.65%) patients, while in 43 (69.35%) patients, it was accidental especially in pediatric ages, and those with

mental and psychiatric illness. Adedeji and colleagues published the results of a retrospective study and documented that suicidal ingestion was the cause in 71.4% of patients, especially among adults, while accidental ingestion was the cause in 28.6% [36]. Struck and colleagues reported a case series of 28 patients with caustic material ingestion with suicidal intent in 64% of cases; 50% of them had a past history of psychiatric problems (depression, schizophrenia) and 33.3% of them had a past history of suicidal attitude [37]. Thomas and colleagues noted that the ingestion was accidental in 62 of 78 (79%) patients [30]. Chibishev and colleagues noted in their report that the caustic material ingestion was suicidal in 87.87%, and accidental in 12.12% of cases [28].

In this study, the presenting symptoms were nausea, postprandial abdominal distension, epigastric pain, nonbilious persistent vomiting, progressive weight loss, and hematemesis with anemia in some patients. In one (1.61%) patient with associated esophageal stricture, there was also dysphagia, chest pain, odynophagia, and vomiting. Ray and Chattopadhyay reported that all patients suffered from vomiting and weight loss while abdominal pain and dysphagia were present in 65%, 54% of patients, respectively [29]. They also noted that 46% of cases presented with associated esophageal stricture which was managed easily by endoscopic dilatation. Honar and colleagues noted in their study that drooling of saliva, vomiting, and dysphagia were the most common presenting symptoms [38]. Gupta and colleagues reported that repeated vomiting and upper abdominal pain were the main symptoms in 75% of patients in addition to early satiety with failure to thrive in 25% of patients [32].

The interval between initial acid ingestion and symptoms of gastric outlet obstruction was 3–6 weeks in 56 (90.32%) patients, four (6.45%) patients were presented after 1 year, and 2 (3.23%) patients were presented after 18 months. Ray and Chattopadhyay noted in their study that 2 months was the median period between ingestion and symptoms (range: 2 weeks–8 months) [29]. The study published by Gupta and colleagues reported that the interval between caustic material ingestion and presentation with gastric obstruction was 6 weeks–26 months (median 5months) [32].

In our study, 2 (8.33%) patients in group A and 3 (7.89%) patients in group B had chest infection. Postoperative vomiting occurred in 3 (12.5%) patients in group A and in 6 (15.78%) patients in group B. One (4.16%) patient in group A and two (5.26%) patients in group B had wound infection. None of our patients developed post-operative bleeding or anastomotic leakage or stenosis or adhesive bowel obstruction or there was any mortality. Ray and Chattopadhyay noted in their study that 11% of cases developed pneumonia, surgical site infection was encountered in 16% of patients, while 5% of cases had paralytic ileus, but none had anastomotic leakage or intra-abdominal collection [29]. They also reported that there was no mortality, except one patient died post suicide 12 weeks after surgery [29]. Venugopal and colleagues reported at their study of antrectomy and Billroth 1 reconstruction that postoperative complications were primary hemorrhage in one patient which necessitates urgent re-exploration, anastomotic leakage in one patient who was successfully responded to conservative treatment, wound dehiscence in one case underwent secondary suturing, and revision surgery for 2 patients with anastomotic stenosis. There was no death reported at their study [39].

Patients who had bypass gastrojejunostomy had upper GI endoscopy with biopsy from the stenotic segment every 6 months for the first year and then every year to assess the stricture to rule out any malignant change. All biopsies had benign histopathology during the follow-up period. It is reported that post corrosive gastric and esophageal cancers may have a latent period up to 40–50 years [40]. According to some reports, 3% of esophageal cancers had past history of corrosive ingestion [40]. The most common location is the level of carina with better prognosis than esophageal cancers of other etiology. Gastric cancer is a very rare complication following corrosive ingestion [41].

Conclusion

The health organizations should give more attention for prevention of the corrosive injury rather than spending more cost for treatment. For the management of postcorrosive long segment pyloric stricture, both Billroth 1 gastroduodenostomy and bypass loop gastrojejunostomy are safe and effective. Billroth 1 gastroduodenostomy has the superiority of prophylaxis against long-term low risk of future malignancy. Bypass loop gastrojejunostomy is a safer alternative to Billroth 1 gastroduodenostomy especially in the presence of severe perigastric adhesions, and malnutrition; marginal ulceration is rare may be due to physiologic antrectomy resulting from mucosal damage. All patients of caustic injuries to the upper gastrointestinal tract should undergo lifelong

surveillance with endoscopy. Future studies should include large sample of patients with long periods of follow-up.

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Conflicts of interest
There are no conflicts of interest.

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