Corrosive acid injury of the stomach

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Abstract

Ingestion of corrosives with accidental or suicidal intent is a common problem in Sri Lanka. Management options and outcomes of corrosive injuries on stomach are not well documented in our setting. The clinical presentation, complications and management outcomes of nine patients with corrosive injury to stomach are presented. Gastric outlet obstruction seen in majority, was managed with bypass procedure (n=5) or resection (n=4). The outcomes of management were successful with both methods.

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Introduction

Corrosive substances either ingested accidentally or with the intent of suicide cause devastating injuries to the upper gastrointestinal tract (GI) and presents as a major management problem to the clinician. The majority of victims are young and in their productive age group. Unlike in the West where most of these injuries are due to alkali, severe upper GI injuries due to acids are seen in countries like Sri Lanka, India, and Turkey [1-4]. They are less well documented. The extent and degree of corrosive acid injury would depend on the nature of the acid, its concentration, the volume ingested and the presence or absence of food in the stomach at the time.

Several theories have been proposed to explain the cause for this peculiar nature of acid injuries. The relative resistance of the squamous epithelium of the oesophageal mucosa to acids and rapid transit through oesophagus to stomach with minimal contact time of acids with oesophageal mucosa reducing the degree of oesophageal injury is one such theory [1]. It is thought that the reservoir function of stomach causes accumulation of significant amount of acid especially in the distal part of the stomach increasing the contact time and hence the increased degree of burns. Further the reflex pyloro-spasms caused by acids delay gastric empyting, further aggravating the degree of acid burn [1]. This theory is supported by the fact that almost all living survivors of gastric acid injuries develop gastric outlet obstruction due to severe antro-pyloric scarring, sometimes with minimal or no injury to the proximal stomach.

Methods

Nine patients with corrosive acid injury were admitted to the University Surgical Unit at Peradeniya Hospital over the period 1989-2006. Their clinical records, operation notes and follow up data were retrospectively perused. The patterns of presentation, the diagnostic and investigative data, the early and late management problems and procedures were recorded.

Results

Ages of our patients ranged from 18-42 years (mean=29 years) and 5 (55%) were females. Substances ingested were sulphuric acid (n=5), acetic acid (n=1), nitric acid (n=1) and in two patients the ingested acid was unknown. Predominant clinical presentation was gastric outlet obstruction (GOO) seen in all patients except the one patient who presented immediately after ingestion with gastric perforation and peritonitis. In all except one patient, the acute stage was managed at a different hospital.

Discussion

Most authors agree that upper GI endoscopy is an important diagnostic tool to demonstrate the anatomical extent and the degree of burn injury [2, 5]. Endoscopic findings provide a basis for grading these injuries. Early endoscopy can demonstrate the full thickness injuries which usually needs surgical intervention. Barium swallows or barium meal with screening was performed on eight of our patients as a means of demonstrating the anatomy of the lesion. Most of the surgical decisions were taken based on this investigation.

Many have demonstrated good long term success rates in adults with corrosive gastric acid strictures with either resection of the involved segment and anastomosis of a jejunal loop to maintain continuity or a jejunal loop bypass anastomosis made to a relatively spared area of stomach leaving the strictured part of stomach in situ.

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Table 1. Presentation and management outcomes of corrosive acid burns to stomach

Case No.	*Upper Gastro-intestinal Endoscopy*	*Barium meal*	*Surgery*	*Outcome*
1	Acute presentation. UGIE or Barium not done. Immediate surgery. showed a perforation in the distal portion of an empty stomach with punctate burns of several loops of bowel.		Freshening and repair of the stomach and damaged small bowel loops.	Despite resuscitation patient succumbed to the injuries.
2	Oesophagus-mucosal erythema, no stricture. Stomach -antro-pyloric scarring and stricture.	Gastric outlet obstruction with proximal distension.	Retro-colic, iso-perisaltic gastro-jejunostomy.	Complete relief of symptoms with good weight gain.
3	Normal oesophagus. Stomach non distensible, only proximal stomach seen.	Extensive scarred and shrunken distal stomach with gastric outlet obstruction.	Gastric resection sparing a limited uninvolved area around fundus. Isoperistaltic jejunal loop reconstruction.	Complete relief of symptoms.
4	Not done.	Oesophagus-stricture at GO-junction. Extra gastric collection, antro-pyloric narrowing with gastric outlet obstruction.	Repair of gastric outlet junction injury with closure of fistula. Resection of distal stomach, proximal gastro-jejunostomy.	1.Leak from GO repair 2.Leak from colo-oesophageal anastomosis. Persistent sepsis.
			Right colonic transposition when first surgery failed.	Death of patient.
5	Erythematous mucosa, no strictures. Strictured pylorus with ulceration.	Distended proximal stomach with mid / distal stomach scarring. ‘‘Tea pot stomach’’	Polya type gastrectomy with retro-colic jejunal anastomosis.	Complete relief of symptoms.
6	Dilated proximal oesophagus gastro-oesophageal junction stricture dilated successfully.	Scarring of lesser curve and antro-pyloric canal with gastric outlet obstruction.	Resection of strictures in distal stomach, Retrocolic loop gastrojejunostomy i.e.gastrectomy	Relief of symptoms after oesophagus was dilated.
7	Oesophagus-normal. Entire stomach poor distensibility, pyloric stricture.	Reduced distensibility of entire stomach, distal scarring, free flow of barium across narrowed pylorus.	Ante-colic, isoperistaltic proximal gastrojejunostomy.	Complete relief of symptoms.
8	Three months after ingestion oesophagitis with ulceration and antro-pyloric stricture.	Oesophagial stricture at 15 cm and partial pyloric obstruction.	Oesophagial dilation Pyloroplasty.	Dysphagia less and no vomiting.
	Four years later admitted with vomiting, no loss of appetite or loss of weight.	Partial pyloric obstruction with abnormality of duodenal cap (oesophagial pouch proximal oesophagus).	Proximal gastrojejunostomy.	Complete relief of symptoms.
9	Lower oesophagial partial stricture from 20cms, small contracted stomach with mucosal scarring.	Small contracted stomach and narrowed antrum scarred lining.	Oesophagial balloon dilatation.	Prolonged post-operative hypotension, Died cause unknown probably hypokalaemia.
			Proximal anticolic gastrojejunostomy.	

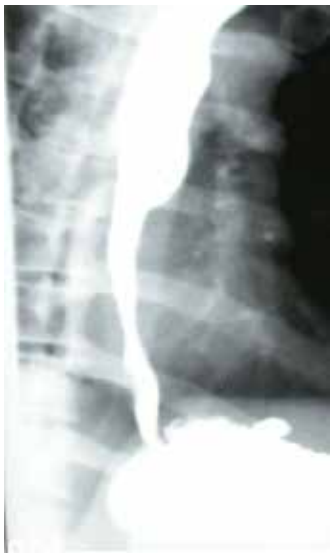

Figure 1. **Corrosive oesophageal stricture with gastric stricture 1B (above).**

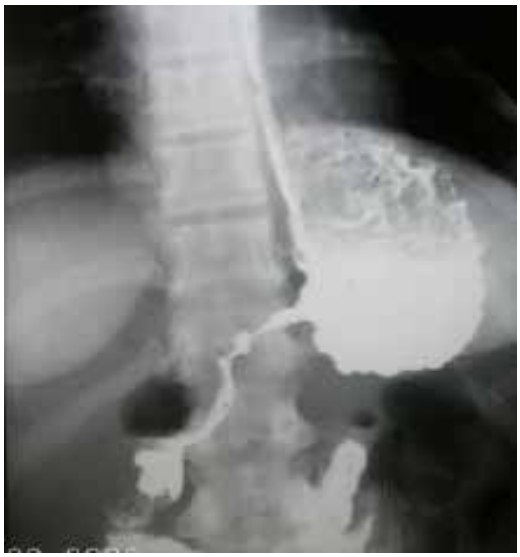

Figure 2. **Corrosive gastric stricture.**

These two were the methods most favoured by us as well, with advantages and disadvantages noted in both methods.

Bypass surgeries using a roux loop of jejunum to a spared area of stomach without a resectional surgery is a relatively simpler operation with relatively low perioperative complications. Many authors have demonstrated a satisfactory long term patency rate for such anastomoses [1,2,6]. This becomes the only available option where near full thickness gastric wall burns in posterior stomach cause complete inflammatory obliteration of lesser sac with pancreas getting densely adherent to stomach. On the other hand all types of bypass procedures on stomach are associated with an increased long term risk of gastric malignancy [7].

However leaving behind the grossly diseased distal part of stomach in situ, which is a potential site for a future malignancy, is the main drawback of this procedure. Two cases of squamous carcinoma of the stomach occurring 12 and 27 years after corrosive acid ingestion have been reported from Sri Lanka [7]. There are no long term studies with an adequate number of patients to prove or disprove this fact. We need to follow up our patients to answer this question.

Declaration of Interest

There are no conflicts of interest.

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