Surgical Management of Gastric Outlet Obstruction Due to Corrosive Injury

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Abstract Corrosive gastric injury is common in India due to easily available acids which are ingested mostly for suicide attempt. Resulting gastric outlet strictures need operation in majority of the patients. We review our experience of surgical management of these cases. Thirty-seven consecutive patients over the last 4 years with corrosive gastric strictures were reviewed. Extent of gastric cicatrization was assessed by endoscopy and barium study. Nutrition was maintained, if necessary by feeding jejunostomy. Early definitive operation was preferred. Outcome of surgery was assessed. Patients presented with vomiting, weight loss, and dysphagia. Twenty patients (54 %) had predominant gastric corrosive injury. The oesophageal stricture in other 17 patients (46 %) could be managed easily by endoscopic dilatation. Prepyloric short gastric stricture was found in 19 patients (51 %). Surgical procedures included stricturoplasty for short strictures and gastrojejunostomy for more extensive distal gastric cicatrization. Complications included wound infection in six (16 %) patients and pneumonitis in four (11 %) patients. All patients gained weight within 6 to 8 weeks. Management of corrosive gastric injury depends on the extent of gastric involvement, associated oesophageal stricture, and general condition of the patient. Early definitive operation and avoiding gastric resection can give satisfactory outcome.

Keywords Corrosive injury · Gastric outlet obstruction · Stricturoplasty · Pyloroplasty

Introduction

Corrosive injuries of the upper GI tract are common in developing countries like India. The spectrum of problem is different from developed Western countries where it is more common in paediatric age group and is mostly accidental. But in India, it is more common in adults and usually due to suicidal attempts. Also, the corrosive agents differ as alkali abuse like lye, battery, etc. is rare and commonly available acids like toilet-cleaning muriatic and hydrochloric acids are ingested more frequently in India [1, 2]. The extent of damage in the oesophagus and stomach depends on an agent, amount ingested and duration of contact. Alkalis are more viscous, tend to adhere to oesophageal mucosa and cause liquefaction necrosis leading to severe oesophageal injuries. Acids are cleared rapidly from the oesophagus to the stomach and, due to corrosive-induced pylorospasm, have long duration of contact with the gastric mucosa and cause damage by coagulative necrosis [1]. The patients present with dysphagia, pain, vomiting and weight loss. Management of corrosive gastric injury depends on the general condition of patient, associated oesophageal injury and type of cicatrization of stomach. In this article, we report our experience of the management of gastric outlet obstruction.

Materials and Method

Over the last 4 years (2009–2012), 37 patients with predominant gastric injury due to corrosive ingestion have been treated in our department which is a tertiary referral centre in Eastern India. The ages of the patients varied from 17 to 52, but 24 (65 %) patients were between 20 and 40 years old. Out of 37 patients, 21 (57 %) were female (Fig. 1). The presenting symptoms were recurrent vomiting, weight loss, malnutrition, dysphagia and abdominal pain. All patients had both upper GI

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Fig. 1 Profile of patients with corrosive gastric outlet obstruction

endoscopy and contrast study (barium swallow) to assess the extent of injury and cicatrization.

In all patients, nutrition was maintained with calorie-rich enteric formulations orally or through jejunostomy, and anaemia and dyselectrolytaemia were corrected before surgery. Patients with severe corrosive injury to the oesophagus and stomach presenting as perforation or gangrene and those with severe oesophagogastric cicatrization which needed replacement of both were excluded from this study.

Results

The study included 21 female patients and 16 male patients with median age of 27 years (Fig. 1). The cause, corrosive agents, type of injury and the symptoms are given in Table 1. Twenty patients (54 %) had predominant gastric injury sparing

the oesophagus. Twenty patients (54 %) had prepyloric short stricture (Table 2). The interval between corrosive injury and presentation is 2 weeks to 8 months (median 8 weeks). Seventeen patients (46 %) presented with concomitant oesophageal stenosis which could be managed easily by endoscopic dilatation. Nutrition was maintained with enteral feeding orally or through a nasogastric tube. Fifteen patients (40 %) had feeding jejunostomy prior to definitive surgery.

Surgical Management

The choice of definitive surgery for corrosive gastric outlet obstruction was determined by the extent of cicatrization of the stomach and the general condition of the patient.

Patients with short-segment stricture involving the pylorus and/or antrum were treated by stricturoplasty anastomosing healthy gastric mucosa to duodenal mucosa. In patients with long-segment stricture involving the distal stomach or extending to the duodenum, either loop or Roux-en-Y gastrojejunostomy was done. In one patient with delayed presentation and doubt of mucosal dysplasia, Billroth I gastrectomy was done. One patient with proximal gastric stricture near the oesophagogastric junction was managed by successful endoscopic dilatation. The timing and nature of definitive surgery after corrosive injury are given in Table 3.

Morbidity/Mortality

Out of 36 patients who had operation for corrosive gastric stricture, four (11 %) patients developed pneumonitis, and six (16 %) patients had wound infection. Post-operative paralytic ileus developed in two (5 %) patients, but none had anastomotic leak or intra-abdominal collection. There was no mortality, except that one patient who had a long-standing psychiatric problem committed suicide 3 months after recovery.

Table 1 Pattern of corrosive injury

	n (%)
Cause	
Suicidal	30 (81)
Accidental	6
Assault	1
Agent	
Toilet-cleaning agents/muriatic acid, etc.	30 (81)
Other acids like sulphuric/nitric	7
Injury	
Gastric	20 (54)
Oesophagogastric	17
Symptoms	
Vomiting	37 (100)
Weight loss	37 (100)
Abdominal pain	24 (65)
Dysphagia	20 (54)

Table 2 Distribution of gastric strictures

Type of strictures	No. of patients (<i>n</i> =37)
Type I	20
Type II	12
Type III	5
Type IV	–
Type V	–

Follow-up

All patients gained weight, and hypoproteinaemia was corrected within 6 to 8 weeks. Seventeen patients with associated oesophageal injury are under periodic dilatation programme. Patients who had stricturoplasty have upper GI endoscopy every 3 months for the first year and then every 6 months to assess gastric outlet which remained satisfactory in a follow-up over 2 years (Fig. 2). Four patients had long-standing corrosive gastropathy.

Out of 16 patients who had gastrojejunostomy, two patients had stomal ulceration which healed with medications. Patient who had gastrectomy did not have any dysplasia or carcinoma in histopathology.

All patients of corrosive injury of the oesophagus and stomach should have lifelong surveillance.

Discussion

Corrosive injury leading to cicatrization of the oesophagus and stomach is common in India. Corrosive injury in developed Western countries involves accidental ingestion in children. In contrast, in India, young adults consume different acids available easily for housecleaning with suicidal intent.

Gastric damage commonly occurs in the antrum or distal stomach because of pooling of a corrosive agent in this area due to pylorospasm leading to longer duration of contact. It

Table 3 Surgical management in corrosive gastric stricture (*n*=35)

Time interval of definitive surgery	Stricturoplasty	Gastrojejunostomy
Within 2 months	10	5
Between 2 and 3 months	3	10
Between 3 and 6 months	5	1
After 6 months	1 (failed endoscopic dilatation)	
	19	16

One patient had Billroth I gastrectomy, and another patient has dilatation

Fig. 2 a Two months after corrosive gastric injury involving pylorus. b The same patient 2 years after stricturoplasty

causes severe fibrosis which lead to stricture and gastric outlet obstruction [1]. Common symptoms are persistent vomiting and weight loss. Most of the patients develop features of gastric outlet obstruction within 1 to 3 months, though delayed presentation even after years was also seen [1, 3]. Dysphagia, a cardinal symptom of concomitant oesophageal stricture, may mask the gastric outlet obstruction till patients can eat after endoscopic dilatation [1].

Both upper GI endoscopy and barium study is important to assess the extent of gastric injury. Based on those, it has been classified into five types by Ananthakrishnan et al. [1]:

Type I Short ring stricture of the stomach within 1 to 2 cm of the pylorus.

Type II Stricture extending proximally up to the antrum.

- Type III Mid-gastric stricture involving the body and sparing the proximal and distal parts of the stomach.
- Type IV Diffuse gastric involvement like linitis plastica.
- Type V Gastric stricture associated with a stricture in the first part of the duodenum.

Nonsurgical management of corrosive gastric injury by endoscopic dilatation is unsatisfactory in most of the cases. Short-segment gastric stricture can be managed by endoscopic dilatation [3], but majority of the patients need surgery [1, 4–6]. Surgical management of corrosive gastric stricture is usually delayed due to poor general health, anaemia and time taken for gastric inflammation to settle so that the extents of cicatrization become obvious [1, 4]. Many patients need feeding jejunostomy, initially. Unfortunately, for poor patients, adequate nutrition is difficult to maintain through jejunostomy feeding, and they need to return to their jobs as early as possible.

In this context, early definitive surgery for gastric outlet obstruction is considered. Early definitive surgery has been reported by Tseng et al. in a series of 31 patients within 2 months of injury [7]. Hwang et al. compared early definitive surgery within 1 to 4 months after injury with delayed surgery and concluded that early definitive treatment can give better quality of life to the patients [8]. In this series, 28 patients (78 %) had early definitive surgery for gastric outlet obstruction within 3 months without any mortality or significant morbidity.

The main aim of surgery in corrosive GOO is the relief of obstruction with reasonable gastric volume. For short-segment stricture in distal stomach stricturoplasty or pyloroplasty, either Heineke–Mikulicz type or Y-V flap can be done [1]. In this series, we did Heineke–Mikulicz-type stricturoplasty with satisfactory outcome. Concern about subsequent fibrosis causing further narrowing of gastric outlet is valid, but we have not encountered any in more than 2 years of follow-up. Though there are many reports about future malignancy developing in corrosive oesophageal stricture [9, 10], there is hardly any report of malignancy developing in corrosive gastric stricture [1]. So the role of gastric resection to prevent malignancy in future is unproven.

In cases of severe stricture and loss of gastric volume, gastrojejunostomy was preferred. To prevent bile reflux, Roux-en-Y gastrojejunostomy was preferred. As some of these patients may develop a significant oesophageal stricture needing surgery later, anticollic route seemed more appropriate for gastrojejunostomy. If the patient had feeding jejunostomy, it was kept for early feeding.

Conclusion

Corrosive injury predominantly causing gastric outlet obstruction is common. Conventionally, patients have two operations, initial feeding jejunostomy followed by definitive operation. For poor patients, maintenance of nutrition through jejunostomy feeding is difficult, and they need to resume their jobs. Early definitive operation within 3 months of injury in the form of stricturoplasty or gastrojejunostomy should be considered. In cases of localised stricture near the pylorus, stricturoplasty is a more physiological operation than gastrojejunostomy, and mortality and morbidity of gastric resections can be avoided.

Conflict of interest None.

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