

## **Management Of Isolated Corrosive Antral Stricture- Experience Of A Tertiary Care Centre In South India.**

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### **I. Introduction**

Isolated corrosive antral strictures are less common than concomitant injury to the oesophagus. However, though the pathology due to corrosives is the same, the management of isolated antral corrosive strictures are different from oesophageal strictures.

We wish to present our experience in the management of Isolated gastric strictures from 2008-2017. 27 patients (14 M:13 F) were with isolated gastric corrosive strictures. 14 of these patients were taken for primary procedure without any enteral access in a median of 11 weeks (range 4-360 weeks). 13 patients had enteral access (11-FJ, 2-FJ+ Venting gastrostomy) done at a mean of 6.3 weeks (0.5-24 weeks). The time from EA (enteric access) to definite surgery was a mean of 16.6 weeks (4-42 weeks). The definite surgery included Antrectomy with handsewn Billroth 1 GDA (Gastro Duodenal Anastomosis) (where duodenal anastomosis done after stapling of the stomach distal to stricture was done in all cases) in 14 cases, Antrectomy with Billroth 1 (totally stapled) in 3 cases, antrectomy with GJ (3 cases), Anterior Gastrojejunostomy (GJ) alone (5 cases), Subtotal and Total Gastrectomy (1 case each). Subtotal and Total Gastrectomy was done as the stricture extended further into the proximal stomach. On subanalysis of the primary surgery group without enteral access, it was seen that GJ was done in 3 cases, Antrectomy with Billroth 1 (eight cases) and Antrectomy with Billroth 2 (2 cases) which included antrectomy with Roux en Y GJ (1 case) and Subtotal Gastrectomy (1 case). The outcomes of surgery for the primary surgery group and enteral access followed by surgery group were similar with regard to operative time (100 min vs 110 min), blood loss (30 vs 33 ml) and post operative stay (12 vs 11 days). One patient in the non enteral access group developed bile leak post operatively which required tube duodenostomy for successful management. There was also no significant differences between those going for stapled versus handsewn antrectomy and Billroth 1 anastomosis.

#### **Management of isolated corrosive antral stricture**

In general, acids with a  $\text{pH} < 2$  are considered corrosive. But pH is not the sole determinant of severity of corrosive action. In addition to pH, it is dependent on the molarity and complexing affinity of an acid. [1] The first case of corrosive induced antral stenosis was reported in 1828. [2] It is well known that corrosive acids affect the stomach more commonly than alkali which more commonly affect the oesophagus [3]. Acids by virtue of lower specific gravity and lesser viscosity, are cleared rapidly from the esophagus to the stomach and pool in the prepyloric area due to corrosive-induced pylorospasm [4,5]. They require a longer duration of contact [4] to cause mucosal damage by coagulation necrosis which lead to stricture formation and gastric deformities like pyloric stenosis, antral stricture, hour glass stomach, or small contracted stomach [6]. Strictures occur most commonly in antrum (1) or in the prepyloric area [7]. When the volume of the corrosive ingested is large or when the patient is on an empty stomach, the pylorus is involved or the entire stomach gets scarred leading to a diffusely contracted stomach. In postprandial state, the body of the stomach is mostly affected. [8] Hydrofluoric acid is an exception as it produces liquefactive necrosis and death may also occur rapidly from dyselectrolytemia as a result of fluoride absorption. [1] In India, the majority of the corrosive injuries are due to acids. The most common acids implicated are bathroom cleaning acid (concentrated hydrochloric acid) and—aquegia, a mixture of nitric acid and hydrochloric acid used as a solvent by goldsmiths [7]. The incidence of coexistent esophageal injury in the literature varies from 20% to as high as 62.5% [9-11]. Isolated injury to the stomach resulting in pyloric stenosis in the absence of oesophageal involvement is very rare, accounting to as little as 3.8% of all the cases of corrosive ingestion, as reported in literature [4]. One Indian study reported 33% of 109 injuries with isolated gastric injury only [7].

### **Pathophysiology and classification**

The spectrum of gastric injury due to corrosives vary from acute injury ( partial or total gastric mucosal or transmural necrosis) to chronic injury. In the acute phase ,oral hyperemia were seen most commonly followed by ulceration and necrosis.[7]

Chronic corrosive gastric injury was classified into the following five types with variations in treatment:[12]

- type I: short ring stricture of the stomach within one or two centimeters of the pylorus; (most common-90%).They are treated most commonly by antrectomy and reconstruction(Billroth-1) orGastrojejunostomy
- type II: stricture extending proximally up to the antrum,which is treated bygastrojejunostomy
- type III: mid gastric stricture involving the body of the stomach and sparing the proximal and distal parts of the stomach.They are usually treated by a distal gastric resection and polyareconstruction
- typeIV:diffusegastricinvolvementproducinga–linitisplasticalikeappearance–.Theyaretreatedby colojejunal bypass with or withoutgastrectomy
- type V: gastric stricture associated with a stricture of the first part of the duodenum

### **Clinical features**

The most common presentation in the acute phase is with abdominal pain, vomiting, and hematemesis [9,13,14]. Rarely ,acute corrosive injury can cause gastric perforation with delayed presentation .However rare, hematemesis following corrosive ingestion is usually self-limiting. However, there are some isolated reports of subacute massive bleeding from the stomach or duodenum following corrosive ingestion [15].

In the late phase, corrosive injury may produce Gastric Outlet Obstruction (GOO) with characteristic feeling of fullness of stomach, nausea, and vomiting and weight loss including severe dehydration and dyselectrolytemia .On clinical examination ,dilated stomach.with succussion splash and ausculto percussion which are classic signs of GOO may be seen.[16]

### **Investigations**

#### **• Endoscopy**

The most useful investigation in the evaluation of an acute corrosive gastric injury is an upper gastrointestinal endoscopy as its vital to evaluate the mucosa of stomach and assessing the degree of injury. it may be done between 3-4 [7] days as the risk of perforation is lowest at this point[17] and the findings will help plan further intervention.In the late phase, endoscopy can delineate any strictures-their location,extent and may even offer venue for therapeutic intervention.

#### **• CT**

CT is non invasive and is more useful in detecting even minor perforation .It can be done in equivocal cases when suspecting perforation in the acute phase . In the late phase , it may provide information regarding the type of stricture involving the stomach .

#### **• Laparoscopy**

In acute injury, Laparoscopy is a useful adjunct to assess patients who have a high risk of gastric perforation as seen on endoscopy or in patients with severe esophageal injury in whom an upper gastrointestinal endoscopy to assess the stomach is not feasible. Some authors advocate routine laparoscopic examination in all injuries of seconddegreeorgreater[1].However thisapproachisnotcommonplace[7].

#### **• Barium Meal**

In the chronic phase, Barium meal may showcatrization, predominantly involving the antrum with an overtly distended stomach and a narrowedpyloric lumen associated with delayed emptying or , linitisplastica type deformity with multiple pseudodiverticula. The radiological findings do not vary as to the type of corrosive ingested .[19]. The results of barium meal study and endoscopy are complementary and help determine the appropriate management

### **Treatment**

#### *Acute phase*

#### **• Conservativemanagement**

All patients who are hemodynamically stable with no evidence of perforation are started on conservative management which includes nil per oral (NPO), placement of an indwelling nasogastric tube, starting of parenteral feeding which may be transitioned to oral feeding subject to patient tolerance.Avoiding a gastric lavage neutralizing the acids or alkali is to be followed.[7] All patients with second degree or greatercorrosive are given parenteral broad spectrum antibiotics and intravenous Proton Pump Inhibitors. Two meta-analyses [20, 21] have described no benefit in the use of systemic corticosteroids to prevent stricture formation.

- **Exploratory laparotomy**

Emergency surgical intervention may be needed mostly in Grade III injuries with necrosis, where patients develop signs of esophageal perforation, peritonitis, or uncontrolled massive hematemesis [11]. There should be a low threshold for consideration of laparotomy at the earliest suspicion. If a laparotomy is undertaken, the stomach is assessed intraoperatively and feeding access in the form of a feeding jejunostomy is performed along with resection (total or subtotal or distal gastrectomy), depending on the extent of necrosis with duodenal closure and cervical oesophagostomy. Reconstruction is best performed later after improving the nutrition status and tiding over of the acute crisis [7]. It is to be noted that any surgical procedure in the acute phase is associated with high morbidity and mortality and all procedures should be kept to a minimum.

*Chronic phase*

Healing process after corrosive injury begins three weeks after the injury, leading to fibrosis and narrowing of lumen, ultimately resulting in stricture. [22] Corrosive injury most often causes pyloric or antral stenosis though shortening and irregularity of lesser curvature and hour glass deformity of stomach may also occur. Gastric outlet obstruction due to antral or pyloric stenosis is however, less common as compared to esophageal stricture. Most of the patients with pyloric stenosis present within three months of ingestion. However, symptoms are known to develop as late as one year after injury [23]. The timing of definitive procedure is controversial but it suffices to state that it can be done when the full extent of cicatrization is visible and patient is nutritionally optimized which may take from 3 to 24 months. [10,24,25]. Partial strictures can be managed by endoscopic treatment or pyloroplasty.

- **Endoscopic Treatment**

**Balloon dilation**

Successful endoscopic balloon dilation has been reported [26]. However successful endoscopic dilation of isolated gastric corrosive strictures are less common and are associated with more complications than oesophageal and concomitant (oesophageal and gastric) corrosive strictures. [27]

The antrum in corrosive strictures are often angulated and strictures are located in curved areas such as the pylorus or duodenal bulb making endoscopic dilations hazardous. [28]. Also corrosive strictures may not be amenable to balloon dilation in the long term [29,30]

**Endoscopic Intralesional steroid injection**

Intralesional steroids to treat corrosive pyloric strictures has been described [31]. However, they are not common place.

- **Surgery**

**Resection or bypass procedures**

Complete stricture resulting in GOO are usually managed by surgery. The surgery procedures offered may depend on the length of the stricture and the general condition of the patient.

Procedures are classified into resection procedures like Antrectomy with reconstruction (bilroth 1 or bilroth 2 or roux limb) [32] for localised strictures or subtotal or distal total gastrectomy in case of diffuse strictures.

Bypass procedures include Gastrojejunostomy (with or without vagotomy).

Most centers prefer some form of gastric resection considering the possibility of malignant potential of scarred stomach (10, 33). However, Gastrojejunostomy is reasonable alternative [34] as the risk of malignant transformation is overrated [23] and can be done with minimal risk of stomal ulceration due to histamine released achlorhydria (physiological antrectomy) (10) especially in children. [35]

Complications encountered in resection procedures include pneumonitis, wound infection, intra-abdominal infection, anastomotic breakdown, reactivation of pulmonary tuberculosis and dumping syndrome. [36]

**Pyloroplasty (Finneys)**

Partial obstruction with moderate mucosal injury usually responds to pyloroplasty. [36]

**Other procedures**

Pedicled flap antropyloroplasty [37] is an alternative in patients with corrosive gastric injury especially for asymptomatic esophageal stricture with gastric outlet obstruction. It helps to widen the narrowed antrum and restore gastric tube length span.

VY advancement antro-pyloroplasty [38] requires little dissection and could be done early in the evolution of the stricture. It is commonly tried in children

## **II. Conclusion**

Isolated corrosive gastric involvement, though rare in the overall presentation of corrosive acid injuries of the GIT, does occur causing serious injury in the acute phase. Once the acute phase is tided over, the definite phase for treating the resulting stricture is undertaken. Surgery is the primary modality and resection of the strictures with reconstruction can be undertaken with excellent short term results including minimal morbidity and mortality in an optimally optimized patient. Endoscopic dilation is another treatment option but is

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