

## Case Series

# Pyloric stricture after corrosive ingestion in children: an attempt to address the optimum management of this rarity

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## ABSTRACT

Corrosive ingestion in pediatric population can have devastating consequences. Pyloric stricture which is a rarer complication has not been discussed in details in existing literature. Whereas the presentation is more or less similar, a “case specific” approach may be required for the best outcome. We analyzed our series of eight patients to formulate a suitable approach to its management. This was a prospective observational study in the department of Pediatric Surgery in a tertiary health care centre in central India. Eight (n=8) patients with corrosive injuries exclusive to the pyloric antrum were analyzed with respect to the corrosive ingested, symptomatology, nutritional status, investigation findings, surgery undertaken and follow up. Total number of patients in our study were eight. Male -7 and female-1, mean age of 6.8 years, ranging from 4 and 10 years, most common agent was acid, ingested accidentally. Period of development of gastric outlet obstruction was 23 days, range between 11 days and 33 days. Initially presented with odynophagia but later developed features of gastric outlet obstruction. Procedure performed were Heineke Mickulicz pyloroplasty and Billroth I gastroduodenostomy with FJ depending on the intra operative findings. No significant post operative complications were encountered on follow up, all the patients had improved general condition and gained adequate weight. No re do surgeries were performed. Corrosive injury of the UGI tract is not uncommon in children. Pyloric stricture as a complication is relatively rare. Parents may seek consultation late only after the child has lost reasonable amount of weight. UGI Endoscopy and UGI contrast study are indispensable to evaluate the severity of damage and formulate the optimum plan of surgery. Early surgical intervention gives excellent result. Both Pyloroplasty and Billroth I anastomosis are safe with low morbidity and excellent long term outcome.

**Keywords:** Corrosive pyloric stricture, corrosive ingestion, Heineke Mickulicz pyloroplasty, Billroth I anastomosis.

## INTRODUCTION

Ingestion of corrosive agent is a frequent cause of benign strictures of the upper aero-digestive tract in India. Accidental ingestion cause serious injuries in children and mostly seen in children less than 5 years, most of the times accidental exposure occurs due to improper storage and lack of parental supervision.<sup>1,2</sup> Easy availability of corrosives as toilet cleaners or as wall coats is responsible for majority of accidental injuries While most injuries are

limited to the oesophagus, some can cause distal injuries to stomach and duodenum. The mechanism of injury is different for acid and alkali but both start initially with regional reaction that may proceed to oedema, eschar formation or even perforation.<sup>3</sup> The extent of damage depends on multiple factors like nature and concentration of the agent, amount consumed, gastric residue and gastric emptying.<sup>4</sup> Although extensive literature is available on corrosive injuries of the oesophagus, streamlining its management, the same for gastric or pyloric injuries are far

less, more so for pediatric population. Hence, a consensus on its optimal management is lacking and a lot is left to the concerned surgeon to decide. In this study, we prospectively analysed a series of pyloric strictures with regards to the agent involved, its type, the time of presentation following ingestion to the development of features of Gastric Outlet Obstruction (GOO) and attempted to draw a conclusion on the most suitable management.

### CASE SERIES

This was a prospective observational study conducted from January 2017 to August 2020. Informed written consent was taken from all and the study was cleared by the institutional ethics committee. Eight (n=8) patients of GOO (complete or partial) following corrosive ingestion were included. Patient profile was noted in terms of age and sex. Nature of the corrosive ingested, its approximate amount and time to presentation (till development of GOO) were recorded. Nutritional assessment was based on initial weight (before corrosive ingestion), weight at presentation, haemoglobin level and serum albumin. An esophagogastroduodenoscopy (EGD) done in most cases around 2-3 weeks after corrosive ingestion and upper GI contrast study using non-ionic contrast agent was done in all preoperatively. Patients who had only oesophageal injury without involvement of the pylorus were excluded.

A Ryle’s tube (RT) was initially inserted in all and trial of feeds given through that. All the patients were started on total Parenteral nutrition (TPN). Those who tolerated RT feeds were continued to be given so. In those with poor nutritional status with persistent vomiting and dyselectrolytemia, a feeding jejunostomy (FJ) was done. A definitive procedure like pyloroplasty (with or without FJ), Billroth I (with or without FJ) was done after 6-8 weeks from ingestion. Per operative findings were recorded. Post surgery, oral feeds were started when normal bowel sounds returned. Patients were discharged when satisfactory. They were called for follow up at 2 weeks, 3 months, 9 months and 18-24 months. History of vomiting (persistent or recurrent GOO due to anastomotic narrowing) was taken and improvement in nutritional status judged by the same parameters as mentioned before. We did not repeat an UGI endoscopy or dye study in follow up visits and relied mostly on the clinical presentation. The FJ was removed after 8-10 weeks of the definitive surgery.

### Outcomes

We had 7 (87.5%) male and 1 female (12.5%) in our study. Age of the children varied between 4 years and 10 years with a mean age of 6.8 years. Most common corrosive agent was acid (toilet cleaner) which was in 7(87.5%) of the patients, 1(12.5%) patient had ingested alkali (glass cleaner). The mean amount ingested was 6.25 ml. In all, the corrosive was ingested accidentally.

**Table 1: Demographic details of patients.**

Age in years	Sex	Hb (gm%)	Albumin (mg/dl)	Nature of substance /Approx volume (ml)	Period of development of GOO (days)	Weight prior to ingestion (kgs)	Weight after ingestion (kgs)	%weight loss(approx)
6	M	11.5	3.0	Acid/5ml	25	25	22.5	10
6	M	10.1	3.3	Alkali/10ml	15	30	26.5	15
4	M	10.2	3.6	Acid/5ml	28	19	17.1	10
6	F	12.0	3.9	Acid/5ml	11	30	27	10
8	M	10.9	2.9	Acid/5ml	32	30	27	10
6	M	9.8	2.8	Acid/10ml	20	26	22.1	15
9	M	11.0	3.1	Acid/5ml	20	28	25.2	10
10	M	11.8	3.2	Acid/5ml	33	29	26.1	10

**Table 2: Investigations and surgical procedures.**

UGIE Findings	UGI contrast study findings	Operation	Hb% Post surgery (9 months)	Albumin Follow up in mg/dl (9 months)
<b>Pyloric cicatrisation+, scope not negotiable into D1</b>	Dilated stomach with narrowed pyloric lumen	Billroth I+FJ	12.5	3.5
<b>Complete Pyloric cicatrisation +, scope not negotiable into D1</b>	Dilated stomach with narrowed pyloric lumen	Billroth I+FJ	11.3	3.8

Continued.

UGIE Findings	UGI contrast study findings	Operation	Hb% Post surgery (9 months)	Albumin Follow up in mg/dl (9 months)
<b>Complete Pyloric cicatrization+, scope negotiable into D1 with difficulty</b>	Dilated stomach with narrowed pyloric lumen	Billroth I+FJ	11	3.9
<b>Incomplete pyloric stricture + , scope negotiated beyond into Duodenum</b>	Dilated stomach with narrowed pyloric lumen	Heineke Mickulicz pyloroplasty	12.8	4.1
<b>Incomplete pyloric stricture+, scope negotiated beyond into Duodenum</b>	Dilated stomach with narrowed pyloric lumen	Heineke Mickulicz pyloroplasty	12.6	3.3
<b>Incomplete pyloric stricture+, , scope negotiated beyond into Duodenum</b>	Dilated stomach with narrowed pyloric lumen	Heineke Mickulicz pyloroplasty	10.8	3.3
<b>Complete Pyloric cicatrization +, scope not negotiable into D1</b>	Dilated stomach with narrowed pyloric lumen	Billroth I+FJ	12.1	3.6
<b>Incomplete pyloric stricture+, scope negotiated into Duodenum</b>	Dilated stomach with narrowed pyloric lumen	Heineke Mickulicz pyloroplasty	13.0	3.7

UGIE- Upper Gastrointestinal Endoscopy; UGI contrast - Upper Gastrointestinal contrast study; FJ- Feeding Jejunostomy; D1- First part of duodenum

All the 8 patients were referred to our centre when they developed features of gastric outlet obstruction (GOO), initial conservative treatment being provided in most hospitals in peripheral centres.



**Figure 1: UGI contrast study showing partial pyloric stricture.**



**Figure 2: UGI contrast study showing complete stricture.**

The mean duration to development of GOO was 23 days, range varying between 11 and 33 days and standard deviation being 7.3. The mean initial weight (before ingestion) was 27 kgs, weight at presentation was 24 kgs, patients had lost a mean weight of 11.25% since ingestion. The mean value of haemoglobin and albumin were 10.9 gm% and 3.2 mg/dl respectively. Demographic details are shown in Table 1.

The most consistent finding on EGD was a scarred pyloric antrum and a partially cicatrised gastric outlet not satisfactorily relaxing with air insufflations making the scope non-negotiable into the duodenum and Partially cicaterised pylorus in few (Figure 3). A dilated stomach with or without narrow stream of contrast flow to the duodenum was observed on contrast radiographs (Figure 1,2).



**Figure 3: EGD showing partial pyloric stricture.**

We did FJ in 3 (37.5%) including the one who had ingested alkali and 5 (62.5%) tolerated semisolid RT feeds. Of the

5 who tolerated RT feeds, a Heineke Mickulicz pyloroplasty was done in 4 (80%) as the outlet obstruction was partial. All 3 from FJ group and 1 from RT group (n=4) underwent resection of strictured pyloric segment due to complete stricture and Billroth I anastomosis (Figure 3). Investigation findings and surgery done are depicted in Table 2. Post operatively, all children were kept NPO for mean duration of 5 days in pyloroplasty group and 6 days in Billroth I group. In 4 of the pts who underwent Billroth I and FJ, FJ feeds were started on day 3. At 9 months, the mean weight gain was 4.4 kgs. The mean gain in haemoglobin and albumin was 0.9 gm% and 0.45 gm/dl respectively. None of the patients showed any clinical sign of anastomotic narrowing till 24 months of follow up.



**Figure 4: Intraoperative resected pylorus prepared for Billroth I anastomosis.**

## DISCUSSION

Accidental ingestion of corrosive substances by children is not an infrequent pediatric emergency. Rampant use in household works and easy availability is the most important reason behind. Incidence is more common in low socioeconomic status population and when both parents are working. Acid which is used as toilet cleaner is the most common offending agent.<sup>5</sup> At times, children with unclear history may present late beyond a few weeks when the symptoms became obvious.

The oesophagus and stomach carry the major brunt of injury and one may be considered mutually exclusive of the other. Whereas esophageal injuries may present early due to odynophagia, injuries of the pyloric antrum may present relatively late when substantial cauterization of the gastric outlet has set in. However, this is comparatively rarer accounting for about one third of all cases.<sup>6</sup>

The tendency of acids “to lick the oesophagus and bite the pyloric antrum” is well known. Viscidity and specific gravity of corrosive acids are lower than that of alkalis, so acids are associated with rapid transit through the oesophagus and the damage primarily occurs in the antrum and pyloric region of stomach due to stasis.<sup>7</sup> Also the tendency of acid to cause pyloric spasm increases the

contact time thereby further aggravating the insult. Alkalis cause deeper injuries and perforation due to absence of eschar formation. However, irrespective of the nature of the corrosive, multiple factors influence the severity of the damage. These include chemical nature of the agent, the amount of gastric residue at the time of ingestion and immediate vomiting.<sup>8</sup> Long term complications like stricture formation are more likely associated with acid. This corroborated well even in our study as acid was the most common agent and pylorus the most common site of injury. (Most of the pts with pyloric stricture present within three months of corrosive ingestion, symptoms may develop as late as one year after injury.<sup>9</sup> In our study the mean time to symptoms due to GOO was 3 weeks (23 days).

Literatures pertaining to corrosive injury of the oesophagus are abundant. But relative rarity of gastric outlet obstruction as a complication of corrosive injury has not yet led us to form a uniform protocol. In our study the commonest symptom was GOO was a relatively late presenting child with classical signs of foul smelling vomitus containing semi digested food particles a few hours after meal and signs of malnutrition in the form of weight loss, anemia and hypoproteinemia. Such symptoms may develop within three months of ingestion but may be as delayed as 1 year after the injury. We found this duration to be 3 weeks in our series.

An immediate EGD may provide useful details regarding the extent of the damage. But it is better to be avoided unless an experienced endoscopist and a pediatric surgeon are not readily available. The same may be done quite safely after an interval of 3-4 weeks. The healing is usually optimum by that time and fibrosis mature to delineate the final magnitude of the scarring. This may practically be a more rational approach as far as definitive surgical intervention is concerned. Though multiple grades of injuries of the oesophagus well known, the same grading system has not been consensually adapted for pyloric injury. Our finding of varying degrees of pyloric cauterization with partial or total occlusion of the gastric outlet is similar to the existing literature. UGI contrast study though not mandatory, may give invaluable details regarding distal passage thereby helping in deciding about the most appropriate surgical approach. It usually shows an over distended stomach with narrowed pyloric lumen associated with delayed emptying which was evident in all of our patients.

Such patients are usually malnourished due to poor satiety and damage to acid secreting cells of the stomach. Hence, prior nutritional stabilization with TPN is of paramount importance for a good final outcome. At times, a feeding jejunostomy may even be considered for this purpose as it was done in 3 of our patients.

The definitive surgery should only be undertaken when the nutritional profile has considerably improved. Varied opinions exist regarding the most suitable operation.



Arguments are there if a drainage procedure like pyloroplasty would be adequate or a more extensive surgery like resection of scarred antrum with gastroduodenostomy/jejunostomy should be done. For practical purpose, however, this should be guided by intraoperative findings in addition to EGD and contrast study reports. We did only a pyloroplasty in those cases with partial stricture. We felt that the potential risks of a gastroenteric anastomosis in such situation would outweigh the benefits. On the other hand, intractable strictures were excised and continuity established by Billroth I anastomosis. We also fashioned a Feeding Jejunostomy alongside. When we reviewed literature, we found that similar approach was followed by many.<sup>10</sup>

There are various other modes of non operative treatment of pyloric strictures which has been reported in adult population which include endoscopy and balloon dilatation, insertion of stents, but reports in pediatric population have only been anecdotal. No such interventions could be planned in our study due to logistics constraints.<sup>11</sup>

Mild to moderate vomiting after any surgical procedure is expected. This may be secondary to prolonged gastric dilatation and gastritis after stasis. It uneventfully resolves in the first week. Gradual gain in weight is observed in a few weeks time followed by improvement in nutritional parameters. Anastomotic stricture is potentially serious long term sequelae which may develop even after a decade. In our series, we did not encounter such in our limited follow up tenure. However, for definitive conclusion, prolonged follow up is warranted.

## CONCLUSION

Corrosive injury of the UGI tract is not rare in children. Pyloric stricture as a complication is relatively rare. Parents may seek consultation late only after the child has lost reasonable weight accompanied by vomiting and electrolyte disturbances. EGD and UGI contrast study are indispensable to know about the severity of damage and formulate the optimum surgery plan. A pyloroplasty for a relatively preserved antrum and resection of one that is grossly scarred promises good outcome. Prior nutritional correction by TPN or FJ may ensure rewarding outcome. However, a long series with more patients and longer follow up would further enhance the credibility of our observation.

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