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Case Series

Laparoscopic management of benign gastric outlet obstruction: a case series with review of literature

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ABSTRACT

Surgery for peptic ulcer disease has since long become a thing of the past, in the era of proton pump inhibitors. Today, it is difficult to believe that these were common operations about 6-7 decades ago. Such has been the impact of proton pump inhibitors (PPI), that, surgery for peptic ulcer has been relegated to the realms of history. However, even in present day, in some rare clinical situations, surgeons may have to resort to surgery for patients suffering from peptic ulcer disease. Herein, we present our experience of laparoscopic surgical management of one of those rare clinical scenarios related to severe peptic ulcer disease. The clinical condition in discussion is gastric outlet obstruction caused by severe peptic ulcer disease in the first part of duodenum. We identified 8 patients (7 males and 1 female) who underwent laparoscopic vagotomy with gastrojejunostomy for benign gastric outlet obstruction, at our institution, operated upon by a single surgeon, between 2004 to 2023. All patients had undergone oesophago-gastro-duodenoscopy (OGD scopy), and contrast enhanced computerised tomography (CECT) of the abdomen. The patients were analysed for age, sex, symptoms, pre-operative evaluation, morbidity/mortality and functional outcomes. They were also reviewed to examine the length of stay, length of procedure, complications and recurrent symptoms on follow-up.

Keywords: Duodenum, Gastric outlet obstruction, Gastrojejunostomy, Laparoscopic, Oesophagogastroduodenoscopy, Peptic ulcer disease, Truncal vagotomy

INTRODUCTION

One of the rare clinical scenarios related to peptic ulcer disease which warrants some endoscopic/surgical intervention, even in the modern era of proton pump inhibitors (PPI), is benign gastric outlet obstruction. Typically, this is caused by 'kissing' ulcers i.e. severe peptic ulcer disease involving both the anterior and posterior walls of the first part of duodenum (D1).[1] Over time, the diametrically opposite ulcers adhere together, thereby significantly narrowing the lumen of the D1. This is complemented by further narrowing caused by scarring and deformity of the D1, caused by the progressing and refractory disease.[1] This causes a 'bottlenecking' effect at the gastric outlet and eventually leads to delayed gastric emptying, accumulation of food residue in the stomach, gastric distension, regurgitation and vomiting.[2]

CASE SERIES

In this retrospective study, we identified 8 patients (1 female and 7 males) with a mean age of 36.75 years (range 22-74 years), who underwent laparoscopic truncal vagotomy and posterior gastro-jejunostomy at our institution, from 2004 to 2023; performed by a single surgeon. All patients had undergone OGD scopy and contrast enhanced computed tomography (CECT) abdomen. In all 8 patients, during OGD scopy, the performing gastroenterologist noted a dilated stomach containing food residue along with a scarred, deformed first part of duodenum (D1) with inability to enter and

negotiate it due to substantial resistance to its passage (Figure 1c and d). Also, multiple gentle attempts were made to pass in and across, a guide wire under fluoroscopic control. But they did not yield any positive result. This was obviously an attempt to negotiate the scarred strictured D1, which would then possibly enable the scope or the dilating balloon to pass over the guide wire, into D2. In all 8 patients, the CECT abdomen ruled out any mass and again confirmed a very scarred, deformed D1 and a distended stomach (Figure 1a and b). Thus, the diagnosis of benign gastric outlet obstruction was confirmed. The only symptom on presentation was vomiting of ingested food about 30-40 minutes after the meals. The average duration of the presenting symptom was 8 months (range 4-12 months). It had gradually worsened in frequency over the past 1-1.5 months, in all the patients. The patients were analysed for age, sex, symptoms, pre-operative evaluation, operative approach, morbidity/mortality, and functional outcomes, length of stay, length of procedure, complications and recurrent symptoms on follow-up. The information on patient demographics is summarised (Table 1).

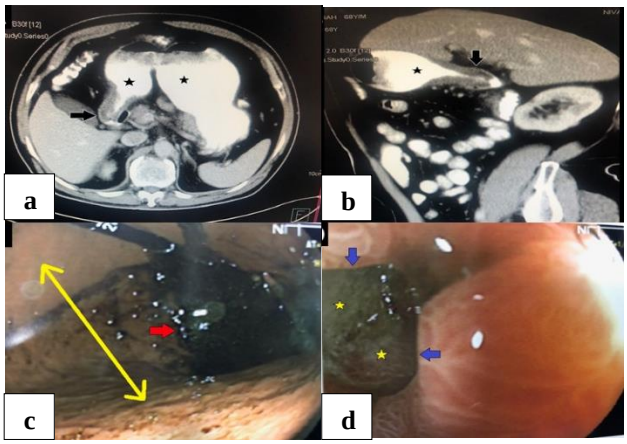

Figure 1 (a and b): CECT abdo axial and sagittal views: show distended contrast filled stomach (black asterisks) with narrowing of D1 (black arrow), (c) OGD scopy pic showing hyper distension of stomach (double headed yellow arrow) and accumulated food residue in stomach (red arrow), and (d) OGD scope at edge of pylorus (blue arrows) unable to enter D1, but taking a peek at D1 stricture (yellow asterisks).

All 8 patients were on proton pump inhibitors (PPIs) pre-operatively, for variable periods ranging from 4 to 6 months. Duration of symptoms ranged from 4 months to 1 year. A gastroenterology referral was advised to all 8 patients for endotherapy vis-a-vis balloon dilatation and/or self-expandable stent insertion. But even a basic endoscopic access into D1 could not be achieved. The patients then underwent a full investigational workup and were then taken up for surgery – laparoscopic truncal vagotomy with drainage procedure (gastro-jejunostomy).

The patients were followed up at 1-, 4- and 6-weeks post-surgery. Further evaluation was carried out after 6 months and then yearly for 2 years. Those patients who failed to physically follow up at 6 months, 1 year and/or 2 years (long term follow up) were telephonically interviewed with a standard questionnaire. At the time of writing this paper, all the patients who were past their last post-operative out patients department (OPD) follow-up visit, were interviewed telephonically with the same questionnaire. Patients were asked for the presence of nausea and vomiting following meals. Ours was a retrospective study of hospital inpatient records, OPD data and information obtained from the telephonic questionnaire.

Table 1: Patient demographics.

Variables	N
Total no. of patients	8
M:F	7:1
Age (in years) (average)	22-74 (36.75)
Mean BMI	21 (18-23)
Average duration (months) of pre-op symptoms	8 (4-12)
Pre-op. OGD scopy	No passage of scope/guide wire into D1
Pre-op. CECT abdomen	No tumor/mass, scarred and deformed D1

Pre-operative anaesthesia check-up was carried out in all the patients. Antibiotics prophylaxis of Ceftriaxone (1-1.5 gm intravenous) and Metronidazole (500 mg intravenous), as per the hospital's antibiotic policy, were administered just before the induction of the anaesthesia. The patient was placed in a supine reverse Trendelenburg position with legs split up with the operating surgeon standing between the legs. The monitor was placed at the head end of the patient on the patient's left side facing the operating surgeon. Pneumoperitoneum was established by closed technique through the Veress's needle. A standard five trocar technique was used. The first optic trocar (10 mm) was inserted at the level of the junction of the upper two-thirds and lower one-third of the xiphisternum to the umbilicus line, slightly to the left of the midline. Four additional ports, right midclavicular (5 mm), subxiphoid (5 mm), left midclavicular (10 mm) and left anterior axillary (5 mm), were inserted under vision. The procedure began with an incision of the gastro-hepatic omentum and entry into the lesser sac. The dissection was then extended cephalad towards the right crus of the diaphragm. The posterior parietal peritoneum over the right crus was incised and the oesophagus dissected away from it. The posterior vagus nerve was then identified and skeletonised (Figure 2b). A 2 cm stretch was then excised between Liga clips. The dissection was then directed to the anterior aspect of the esophagus. The phreno-esophageal membrane was divided and mediastinal space anterior to esophagus, entered. The anterior vagus nerve was then identified, skeletonised and it's 2 cm stretch excised between clips (Figure 2a). Both the specimens were sent

for a histopathological confirmation. The right sided trocar was now converted to 10 mm and the camera was shifted into it. The operating surgeon then shifted to the patient's right side to perform the rest of the operation. The umbilical 10 mm trocar is then converted to 12 mm to facilitate stapling. The gastro-colic omentum was then incised using the harmonic scalpel and posterior wall of the stomach was identified (Figure 2c). A loop of jejunum, approximately 15-20 cm from the duodeno-jejunal flexure, was identified. A posterior, short loop, retrocolic, iso-peristaltic stapled-cum-sutured gastro-jejunostomy was then fashioned using the harmonic scalpel and endo-GIA linear cutter loaded with green cartridge (Figures 2d, 3a-d, and 4a-c). A no. 12 Ryle's naso-gastric tube (RT) was passed across the anastomosis. On either side of the anastomosis, 2 extra sero-muscular approximating stitches were taken so as to prevent torsion (Figure 4d). A peritoneal toilet was given and a 32 French tube drain inserted from the left side, before concluding the operation.

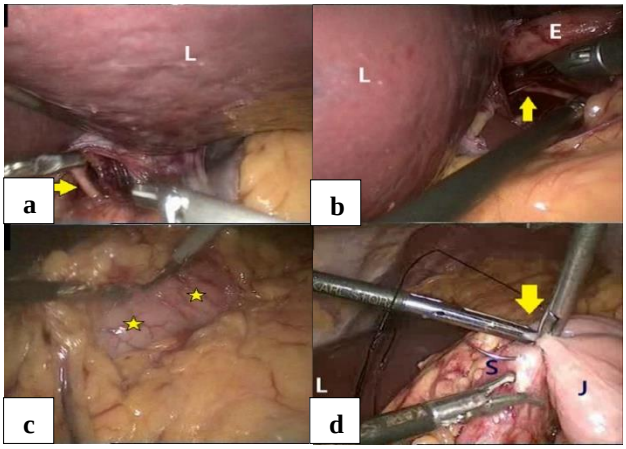

Figure 2: Operative pictures (a) anterior vagus nerve (yellow arrow) being skeletonised before division, (b) posterior vagus nerve (yellow arrow) being clipped prior to division, also seen are the liver and esophagus, (c) window created in gastro-colic, through which posterior wall of stomach yellow asterisks) is visualised, and (d) stay stitch (yellow arrow) being taken through posterior wall of stomach and proximal jejunum.

All 8 patients were kept nil per oral for 3 days. They were started on liquids per orally on post-operative day (POD) 4 and semi solid diet on POD 5 after removal of the RT. The abdominal drains were removed on POD 5 in all the patients. Seven patients were discharged on POD 6 and 1 was discharged on POD 7, after first bowel movement. On their POD 10 OPD follow up visits, all their operative wounds had healed well and all were asymptomatic.

The average operating time was 130 minutes (range 120-140 minutes). The mean hospital stay was 6.13 days (range: 6-7 days). One patient had surgical emphysema which settled without any further intervention, over 24 hours. Two patients had minor port site bleeding which was stopped with external pressure. At post-operative follow-up, 100% (n=8) of the patients reported marked improvement in their preoperative symptoms, while 1 (12.5%) patient described heartburn (grade I, mild, daily) at their 1st, 4th and 6th week follow up visits. There was no conversion to open and we observed no peri-operative mortality. There were no anastomotic leaks in this series. There were no patients who underwent re-do surgeries, in the series. Histopathological evaluation confirmed nerve tissue in all 16 specimens and there were no negative reports. The patient who reported mild reflux related symptoms was managed effectively to his satisfaction, with a proton pump inhibitor course for 1 month. At the time of writing this paper, a telephonic interview was conducted with the patients. Over an average follow up period of 207.75 months (range 145-235 months), all were asymptomatic. None of the patients of this series developed dumping syndrome. The intra and post-operative details are summarised (Table 2).

Table 2: Intra and post-operative details.

Parameters	N
Average operating time (minutes)	130 (120–140)
Average length of stay (days)	6.13 (6–7)
Average follow up period (months)	207.75 (range 145–234)
Post op mortality	0
Intra op morbidity	
Port site bleed	2
Surgical emphysema	1
Post op morbidity	
Surgical emphysema	1
Mild reflux	1
Anastomotic leak	0
Recurrent post meal vomiting	0
Dumping syndrome	0
Conversion to open	0

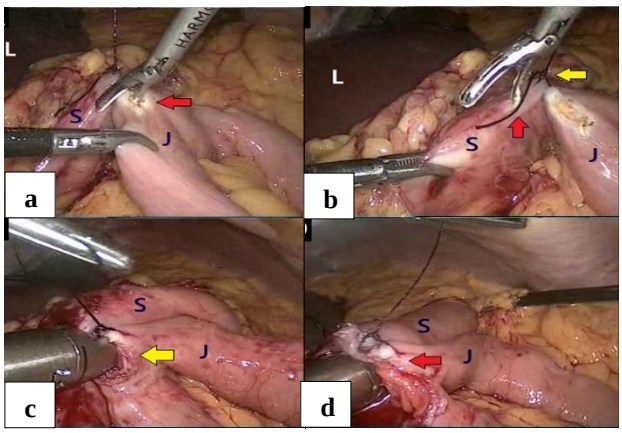

Figure 3: (a) Jejunotomy (red arrow) being made with harmonic scalpel, (b) gastrotomy (red arrow) in progress using harmonic scalpel (yellow arrow), (c) and (d) laparoscopic stapler - linear cutter inserted (yellow arrow) and fired (red arrow).

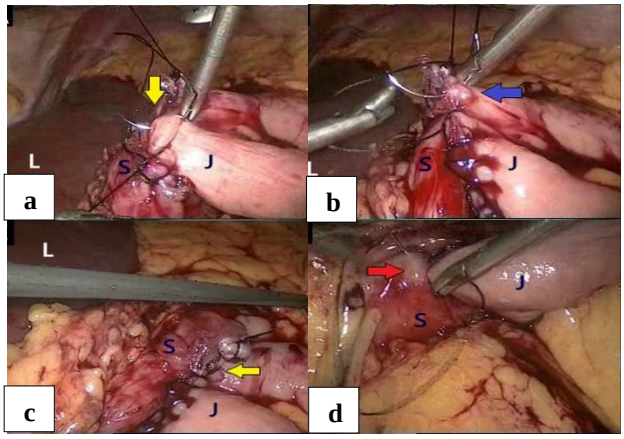

Figure 4 (a and b): The opening caused by the stapler being suture closed (yellow and blue arrows), (c) completed suture line (yellow arrow), and (d) additional sero-muscular stitches (red arrow) being taken on either side of anastomosis to prevent twist/volvulus.

DISCUSSION

Gastric outlet obstruction (GOO) is a clinical syndrome which usually manifests as abdominal pain, post prandial vomiting, early satiety and weight loss. The presenting symptom of GOO is usually vomiting and is described as non-bilious and characteristically containing undigested food particles.[3] In the early stages of obstruction, vomiting may be intermittent and usually occurs within 1 hour of a meal.[3] Patients with GOO resulting from a duodenal ulcer or incomplete obstruction typically present with symptoms of gastric retention, including early satiety, bloating or epigastric fullness, indigestion, anorexia, nausea, vomiting, epigastric pain, and weight loss.[3] They are frequently malnourished, dehydrated and have a metabolic insufficiency.[4] Once GOO is suspected, timely investigations to determine the underlying etiology of the obstruction should be performed. It can be either due to a mechanical cause or a motility disorder. Mechanical obstruction can be at the level of distal stomach, pylorus or first part of duodenum. It can be extrinsic or intrinsic. Etiology of GOO can also be categorised as benign and malignant. Benign causes which lead to mechanical obstruction include peptic ulcer disease, non-steroidal anti-inflammatory drug (NSAID) use, *Helicobacter pylori* infection, polyps, corrosive substance ingestion, gastric tuberculosis, gastric bezoars, gastric volvulus, Crohn's disease, eosinophilic gastroenteritis, Bouveret syndrome, annular pancreas, pancreatitis-acute/chronic and anastomotic strictures.[5] Malignant cause of mechanical GOO includes distal gastric cancer, pancreatic adenocarcinoma, gastric lymphoma, metastatic or primary duodenal malignancy, gastric carcinoid and locally advanced gall bladder carcinoma or cholangiocarcinoma.[5] Gastroparesis is the most common motility disorder that causes GOO and may be due to diabetes mellitus (leading cause of gastroparesis), medications such as opiates or anticholinergics, injury to vagus nerve due to fundoplication procedure or bariatric surgeries.[6] Before the advent of PPIs, peptic ulcer disease (PUD) was the major cause of GOO. With the availability of better treatment of *H. pylori* infections and the use of PPIs, the incidence of GOO by PUD has declined to just 5%.[4] More recently, 50 to 80% of cases of GOO are related to underlying cancers.[4] The incidence of peri-pancreatic malignancy causing GOO is 15% to 20%.[4] The patients often present with nausea and vomiting as their chief complaint. Acute onset of symptoms may lead a health care provider to suspect gallstones, pancreatitis, peptic ulcer disease, volvulus, or migration of PEG-tubes in specific cases. Benign causes of GOO most commonly present with early satiety (53%) and bloating (50%), while symptoms of the malignant causes include pain, vomiting, weight loss, and malnutrition.[4] *Hypertrophic pyloric* stenosis (HPS) is a frequent cause of GOO in neonates. It occurs in 1.5 to 3 per 1000 live births and more common in males (1:150 males and 1:750 females).[7] Its occurrence is rare in older children and adolescents. HPS is caused by diffuse hypertrophy and hyperplasia of the pyloric smooth muscles which leads to narrowing of the antrum, resulting in GOO. HPS is one of the most common indications for surgery during the first six months of life. Abdominal distension and a succussion splash occur in about 25% of patients and is a reflection of retained gastric material.[4,7] If a succussion splash is noted more than four hours after a meal, it is usually suggestive of a GOO with a sensitivity of 50%.[4,7] Plain radiography shows distended gastric air bubbles which do not cross the midline.[4,7] If the obstruction is large, the small bowel may not be visualized. Studies with barium or water-soluble contrast can provide more information on the underlying cause of the blockage. If the contrast fails to pass into the small bowel, it is suggestive of complete obstruction. The CT scan may also give additional details such as the thickness of the pylorus or gastric wall, and it can also reveal if lymph nodes or pancreatic lesions are present. Endoscopy is generally needed to confirm and establish the specific cause of the obstruction. A naso-gastric tube should be inserted and suction should be done before endoscopy to reduce the risk of aspiration. Following gastric decompression, to further evaluate mechanical outlet obstruction, a saline load test can be helpful. The saline load (750 ml) is emptied into a patient's stomach through a nasogastric tube. If more than 400 ml gastric contents are aspirated after 30 minutes, it is considered a positive test.[8] Biopsies taken during endoscopy can confirm if there is malignant cause for the GOO. In benign GOO caused by PUD, conservative management should be tried first. This includes acid suppression, NSAID avoidance, testing for and treating *Helicobacter pylori*. If conservative management fails, dilation via endoscopy and then SOS surgery should be attempted.[9] Endoscopic balloon dilation (EBD) was introduced in the 1980s, and prior to this, GOO was managed primarily with a surgical approach. First, endoscopy is done to visualize the ulcer in the narrowed portion of the stomach or duodenum.[10] Once the stricture is visualized by endoscopy, endoscopic balloon dilation is attempted. A balloon dilator is inserted through the

working channel of the scope, or a balloon is placed over a guidewire under fluoroscopic guidance. The extent of the stricture dictates the amount of dilation required. Generally, narrow strictures will require dilation in a stepwise manner, which is carried out over multiple sessions. EBD turns out to be successful in the short term and almost instant symptom improvement is seen. It may be beneficial to postpone dilation beyond 15 mm until after a period with medical management. After a sufficient dilation is achieved, a sustained clinical response is seen in only around 70% to 80% of patients. If the stricture is refractory to dilation, stent placement with or without surgery should be considered.[11] Stent placement with self-expandable metal stents (SEMSs) has been used as an alternative to surgery. Surgical management is also one option for treating benign GOO if the pylorus cannot be safely dilated due to obstruction or if the obstruction remains despite of endoscopic and medical management. When the extrinsic compression of GOO is caused by chronic pancreatitis, is less likely to respond to EBD and should be considered for early surgery.[7] For malignant obstruction, resection, decompressive gastrostomy, bypass surgery, endoscopic stenting and endoscopic ultrasound-guided gastroenterostomy are some of the treatment options. Diagnostic laparoscopy or exploratory laparotomy can be done to evaluate the degree of disease before a surgical bypass is performed, which is usually done as a palliative measure. Those with several areas of obstruction should undergo decompressive gastrostomy, and enteral or parenteral feeding options may be considered. Endoscopic deployment of SEMS can be done as a palliative measure for malignant GOO to provide relief from obstructive symptoms and to improve patients' quality of life. Surgery, including gastrojejunostomy, can also be considered for GOO. When malignant GOO is not amenable to surgery or SEMS placement, a percutaneous decompressive gastrostomy (PDG) can be used. A PDG with jejunal extension allows for decompression and access for enteral nutrition.[12] Another treatment modality is endoscopic ultrasound (EUS) guided gastro-jejunostomy using lumen-apposing metal stents (LAMS) where a bypass is created by inserting a stent from the stomach to the small bowel distal to the obstruction under EUS and fluoroscopic guidance. This approach is useful for both malignant and benign GOO. Patients undergoing endoscopic treatment with either balloon dilatation or stenting are at risk for perforation. Although endoscopic balloon dilatation (EBD) associated perforation rates in benign peptic stenosis are 3% to 6%, higher rates are seen with a balloon diameter greater than 15 mm.[9] A brief overview of various therapeutic options for benign GOO is summarised (Table 3).

Table 3: Various therapeutic modalities for GOO, in current use.

Therapy	Originator-pioneer/year of origin/indication	Salient features	Results	Advanta-ges	Disadvantages
Endoscopic balloon dilation (EBD)	Benjamin et al/1984/GOO[19]	Through-the-scope 5-mm balloon used over guide wire	*70-80% of patients benefitted, *only short term <6 months	Minimally invasive	*Poor long term results, *no large volume long term follow up studies
Endoscopic placement of self-expanding metal stent (SEMS) deployment	Dohmoto et al/1990/large bowel malignancy, indications then expanded to gastroduodenal and biliary pathologies[20]	Palliative treatment for malignant GOO, also has a limited role in benign GOO	86-89% short term (<6 months) success rates	Minimally invasive	*Poor long term results, *no large volume long term follow up studies
Endoscopic ultrasound-guided gastroenterostomy (EUS-GE)	*Binmoeller et al/2012/in a porcine model; *Barthet, Binmoeller et al/2015/3 humans[21,22]	*Study in 5 pigs, good results, mature tract noted even after stent removal after 5 months; * 3 patients with benign and malignant GOO underwent EUS-GE, 1st reported humans	Good early results reported in animal model and first 3 patients/no major complications	Minimally invasive	*No large cross-sectional human studies, *no long term follow up studies
Percutaneous decompressive gastrostomy (PDG)	Gauderer/1980[23]	Tube insertion, placement and fixation into hyper-distended stomach on emergency basis to de-compress – a	Effective procedure for deflation of stomach with assured results	Easy to perform, fast and effective	Not a curative definitive procedure, cannot be standalone therapy

Continued.

Therapy	Originator-pioneer/year of origin/indication	Salient features	Results	Advanta-ges	Disadvantages
		bridging procedure to definitive therapy			
Laparoscopic/ open GJ	Anton Wolfler-1st open GJ/1881/antral CA	Wide sutured or stapled anastomosis	Leaks, anastomotic ulcers, jejuno-gastric intussusception	Good long term results	More invasive than endoscopy

Peptic ulcer disease (PUD) includes both gastric ulcer and ulcer in the first part of duodenum. PUD occurs due to imbalance between protective and destructive factors which ultimately lead to damage to the superficial mucosal layer exposing the inner submucosa to the gastric acid.[13] Among the various causes of PUD, Helicobacter pylori and NSAID use are common. Recurrent use of NSAIDs and concomitant *H. pylori* infection lead to low levels of prostaglandins and persistent inflammation leads to increased susceptibility of mucosal layer to gastric acid leading to ulcer formation. Ulcer may form on the anterior surface of the inner lining of duodenum or the posterior surface of duodenum. But when both anterior and posterior ulcers co-exist, they may adhere to each other and form what is known as 'kissing' duodenal ulcer. When the ulcer apposing surfaces are large enough, narrowing of the luminal space occurs and leads to gastric outlet obstruction. Esophago-gastro-duodenoscopy can be attempted and ballon dilatation of the segment may be tried. If this fails a bypass path eg. Gastro-jejunostomy for the gastric content has to be made so as to avoid acidic content reaching the ulcerated portion and also for allowing the flow of nutrition into distal parts of the intestines. Acidic secretion can be reduced either with proton pump inhibitors (like pantoprazole, rabeprazole, omeprazole or esomeprazole) or vagotomy procedure like truncal vagotomy, selective vagotomy and highly selective vagotomy. The indications for vagotomy are only few due to advancements in medical therapy and it is reserved for complicated ulcer disease which has failed medical therapy. The type of surgery depends on type of ulcer, complications (bleeding, perforation, obstruction, intractability) and location of ulcer (type I to V gastric ulcer – modified Johnson classification).[14] The treatment strategies widely vary depending on the etiology. Sequelae of vagotomy in peptic ulcer are dysphagia, dumping syndrome, gastric emptying disturbances, diarrhoea, and functional disturbances of liver, bile ducts and pancreas. Dumping syndrome is characterized by rapid gastric emptying which leads to excess gut hormone release. There are two subtypes to this syndrome i.e. early dumping syndrome and late dumping syndrome. Early dumping syndrome is characterised by symptoms occurring within 30 min of meal. Early dumping symptoms are divided into abdominal and systemic symptoms. Abdominal symptoms consist of borborygmi, fullness, abdominal distension, pain, nausea and diarrhoea. Systemic (vasomotor) symptoms consist of palpitations, desire to lie or sit down, feeling of warmth, sweating, flushing, dizziness, fatigue and exhaustion. Early dumping is the result of rapid nutrient delivery into the small intestine. Late dumping syndrome occurs after 1 to 3 hours after a meal and the symptoms are caused due to hypoglycaemia which include fatigue, weakness, confusion, hunger, syncope, and loss of consciousness, and autonomic and/or adrenergic reactivity.[15] All these disturbances, except dysphagia, are less pronounced after high selective vagotomy.[16] Post vagotomy syndrome following the operative section of the vagus nerves is characterized by diarrhoea. The incidence of diarrhoea after truncal vagotomy is about 25% and 15% after selective vagotomy.[17] The complications of truncal vagotomy are well documented. Resection of the vagal nerve trunks above the celiac and hepatic branches leads to parasympathetic denervation of the pylorus, liver, biliary tree, pancreas, and small and large intestines. Complications include delayed gastric emptying, post vagotomy diarrhoea, post vagotomy hypergastrinemia, dumping syndrome and ulcer recurrence.[18] In benign GOO caused by PUD, lifestyle modifications are the cornerstone for management including acid suppression, NSAID avoidance, smoking cessation, testing for and treating *H. pylori*.

CONCLUSION

As seen in this case series, surgery is very rarely required for peptic ulcer disease in the era of PPIs. The 'kissing ulcers' of peptic origin in D1 are known causative factors for benign GOO. Also, this study underscores the fact that laparoscopic therapy for benign GOO is feasible in an advanced setup complemented by essential advanced laparoscopic surgical skills.

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