**Name of journal: *World Journal of Gastrointestinal Endoscopy***

**ESPS Manuscript NO: 26020**

**Manuscript Type: Review**

**Update on endoscopic management of gastric outlet obstruction in children**

Chao HC. Endoscopic management of pediatric gastric outlet obstruction

**Hsun-Chin Chao**

**Hsun-Chin Chao,** Division of Gastroenterology, Department of Pediatrics, Chang Gung Children’s Hospital, Chang Gung Memorial Hospital, Chang Gung University College of Medicine, Taoyuan 33305, Taiwan

**Author contributions:** Chao HC contributed to the manuscript.

**Conflict-of-interest** **statement:** No potential conflicts of interest relevant to this article were reported.

**Open-Access:** This article is an open-access article which was selected by an in-house editor and fully peer-reviewed by external reviewers. It is distributed in accordance with the Creative Commons Attribution Non Commercial (CC BY-NC 4.0) license, which permits others to distribute, remix, adapt, build upon this work non-commercially, and license their derivative works on different terms, provided the original work is properly cited and the use is non-commercial. See: http://creativecommons.org/licenses/by-nc/4.0/

**Manuscript source:** Invited manuscript

**Correspondence to: Hsun-Chin Chao,** **MD, Associate Professor,** Division of Gastroenterology, Department of Pediatrics, Chang Gung Children’s Hospital, Chang Gung Memorial Hospital, Chang Gung University College of Medicine, 5 Fu-Hsing Street, Gueishan District, Taoyuan City 33305, Taiwan. chaohero@yahoo.com

**Telephone**: +886-3-3281200

**Fax**: +886-3-3288957

**Received:** March 27, 2016

**Peer-review started:** March 28, 2016

**First decision:** May 23, 2016

**Revised:** June 18, 2016

**Accepted:** August 6, 2016

**Article in press:**

**Published online:**

**Abstract**

Endoscopic balloon dilatation (EBD) and surgical intervention are two most common and effective treatments for gastric outlet obstruction. Correction of gastric outlet obstruction without the need for surgery is an issue that has been tried to be resolved in these decades; this management has developed with EBD, advanced treatments like local steroid injection, electrocauterization, and stent have been added recently. The most common causes of pediatric gastric outlet obstruction are idiopathic hypertrophic pyloric stenosis, peptic ulcer disease followed by the ingestion of caustic substances, stenosis secondary to surgical anastomosis; antral web, duplication cyst, ectopic pancreas, and other rare conditions. A complete clinical, radiological and endoscopic evaluation of the patient is required to make the diagnosis, with complimentary histopathologic studies. EBD are used in exceptional cases, some with advantages over surgical intervention depending on each patient in particular and on the characteristics and etiology of the gastric outlet obstruction. Local steroid injection and electrocauterization can augment the effect of EBD. The future of endoscopic treatment seems to be aimed at the use of endoscopic electrocauterization and balloon dilatations.

**Key words:** Gastric outlet obstruction; Endoscopic balloon dilatation; Electrocauterization; Steroid injection; Children

**© The Author(s) 2016.** Published by Baishideng Publishing Group Inc. All rights reserved.

**Core tip:** Correction of gastric outlet obstruction without the need for surgery is an issue that has been tried to be resolved in these decades; this management has evolved with the development of pneumatic dilators and, more recently, local steroid injection and electrocauterization have been added. Endoscopic balloon dilatation (EBD) are used in exceptional cases, some with advantages over surgical intervention depending on each patient in particular and on the characteristics and etiology of the gastric outlet obstruction. Local steroid injection and electrocauterization can augment the effect of EBD.

Chao HC. Update on endoscopic management of gastric outlet obstruction in children. *World J Gastrointest Endosc* 2016; In press

**INTRODUCTION**

In the last few decades, upper gastrointestinal endoscopy is a technique widely employed for diagnostic and therapeutic purposes for evaluation of esophageal, gastric or duodenal diseases. Upper gastrointestinal endoscopy has become the common complementary test for investigation of gastric diseases due to its accessibility and safety assures extensive clinical utilization in patients with gastric or duodenal diseases. Recent technological advances in endoscopic imaging and tissue analysis obtained from the stomach aid to identify the characterization of diseases such as inflammation, infection and neoplasia. Recent technological advances have increased the capability of endoscopy in treating gastrointestinal diseases, including those affecting the stomach.

Diseases affecting the stomach have been described and eventually treated by endoscopy in routine clinical practice. Using endoscopy to elucidate gastric outlet obstruction (GOO) in children has been still a field of intensive and challenging research. This review provides an update on the role of endoscopy in the management of GOO, highlights the latest advances in the endoscopic management of GOO, and focuses on the efficacy of endoscopic balloon dilatation (EBD) in the pediatric population. We also point out recent evidence regarding the utility of magnifying endoscopy in the management of GOO.

***Data search***

The searches were limited to articles published in English as well as clinical articles or case studies to identify objective articles related to gastric outlet obstruction from January 1975 to June 2016. All articles considered eligible were evaluated, and finally selected on the basis of research and case series. The articles of gastric volvulus were searched, but this entity is excluded from this review, due to gastric volvulus is specific complex disease entity with varied causes (congenital, idiopathic, or acquired), and the gold standard treatment of pediatric gastric volvulus remains surgical intervention.

**ETIOLOGY**

GOO is an obstruction in the antrum, pylorus or bulbar duodenum. Unlike adult patients, most of the pediatric patients with GOO have benign disease. Peptic ulcer disease and corrosive ingestion are the leading causes of benign outlet obstruction in adults[1]; while idiopathic hypertrophic pyloric stenosis (IHPS) and peptic ulcer disease (PUD) remains the two most common cause of GOO in children[2-4]. Table 1 shows the etiology of GOO in children. IHPS are the most common cause of GOO in children. The typical presentation is increasing vomiting that becomes projectile between 2 and 8 wk in age. Gastric retention after prolonged obstruction contributes to gastric atony, while most cases are caused by antral, pyloric or duodenal ulceration. Scarring and tissue remodeling may cause GOO in chronic PUD. A significant decline of the incidence of PUD due to the discovery of *Helicobacter pylori (H. pylori)* and proton pump inhibitors (PPI**)**[5,6]. *H. pylori* infection participated a less significant role in children with GOO, compared to adults[3].

Caustic ingestion remains a major social and medical issue in children, especially for infants and young children. Case series of corrosive injury related gastric outlet obstruction have still been reported in these two decades[7-9]. GOO is a significant complication of corrosive ingestion[8]. Caustic ingestion (alkali or acid) can cause GOO as a result of antral/pyloric scarring. Other rare causes are gastric antral web[10], gastric duplication[11], ectopic pancreas[12], gastric volvulus[3], gastric polyps[3], idiopathic gastric outlet obstruction[13], foveolar cell hyperplasia[14], and bezoars[15,16]. Antral web, known as antral mucosal diaphragm or prepyloric web, is a rare etiology in pediatric GOO. Histologically the web is composed of normal, non-inflammed mucosal and submucosal gastric mural layers. Gastric duplication cyst are the least common of the alimentary duplications, they usually presented before 1 year of age with symptoms of obstruction, pain, bleeding or ulceration[11].Heterotopic pancreas is generally an asymptomatic lesion and is a rare cause of GOO. Gastric volvulus is characterized by a rotation of the stomach of more than 180° along its short or long axis causing variable extents of GOO. Acute gastric volvulus may create a closed-loop obstruction leading to incarceration and strangulation. In general, emergency surgery remains the standard treatment for acute gastric volvulus. Foveolar cell hyperplasia is a rare disease entity, described as a possible cause of for long-lasting GOO in patients with IHPS, it requires the excision to resolve the obstruction. Gastric polyps are often hyperplastic and asymptomatic. Gastric polyps are usually diagnosed at endoscopy incidentally. Lactobezoar is a condensed mass of undigested milk concretions to be found within the gastrointestinal tract[14]. Lactobezoar is often found in infants, it can precipitate GOO, resulting in medical or surgical conditions. The trichobezoar is another rare cause of obstruction of the gastrointestinal tract, and it is usually presented as GOO[15].

Inflammatory causes like Crohn’s disease and tuberculosis have been reported in adult patients with pyoric obstruction[17,18], these two disease entities are relatively rarely reported in pediatric patients. Isolated gastroduodenal Crohn’s disease is rare, occurring in fewer than 5% of patients. A continuity that involves the antrum, pylorus, and proximal duodenum have been reported in about 60% of patients[17]. In tuberculosis, involvement of stomach or duodenum occurs in 0.3 to 2.3% of patients, and 61 % of patients with gastroduodenal tuberculosis present as GOO[18]. Gastric polyps or neoplasms are rare in children but should always be considered as an etiology of GOO in children, especially in older patients[19].

**EVALUATION**

***Clinical manifestations***

The usual presentations were nausea, vomiting, epigastric pain, early satiety, abdominal distention, abdominal mass, visible peristalsis, weight loss and electrolyte imbalances. Epigastric pain, nausea and vomiting, abdominal distention, early satiety and weight loss are the most common presenting symptoms of GOO[20]. The onset of symptoms varies based on the etiology, symptoms usually occur rapidly with gastric volvulus, corrosive injury, food impaction (bezoar), prolapse of a large gastric polyp[3,8,15]. Other causes are inclined to follow a more slothful course. Malignant cause usually has a shorter duration of symptoms compared with benign causes. Patients with benign causes commonly presented with early satiety (53%) and bloating (50%) whereas in the patients with malignant causes presented more commonly with vomiting, pain, and weight loss[20].

Persistent succussion splash (a “splash” reflective of retained gastric material) detected by auscultation of for more than four after meals, suggesting GOO with a sensitivity of 50%[21].

***Investigations***

GOO patients with repeated vomiting may have electrolyte imbalances with hypokalemia or a hypochloremic metabolic alkalosis. Anemia, elevated inflammatory markers (C-reactive protein, ESR), and abnormal biochemical tests in hepatobiliary function, pancreatic function, or renal function may reflect the underlying disease. Elevation of serum gastrin concentration as a result of distention-induced gastrin release may occur in GOO patients and this condition can be confused with Zollinger-Ellison syndrome.

Plain radiographs may demonstrate an enlarged stomach. Small bowel may not be visualized because of air paucity. Calcified gall stone and/or pancreatic calcification may be revealed. Diagnosis was easy clinically and confirmed by barium studies and/or upper gastrointestinal endoscopy. Contrast studies with barium or water soluble contrast aid in providing diagnostic clues to the underlying diseases. Failure of contrast passing into the small bowel is highly suggestive of complete GOO. Barium studies are helpful in delineating the site of obstruction as well its extent. Adequate gastric decompression should be initiated before performing barium or water soluble contrast to reduce the risk of aspiration. CT scan may disclose additional anatomical details, specially the wall thickness in the stomach, pylorus and duodenum, biliary lesions, pancreatic abnormalities or lesions, and enlarged lymph nodes not visualized on regular imaging studies[21].

Endoscopy is often required to ascertain the diagnosis of GOO and identify a specific etiology in company with a therapeutic assistance. Patients should fast for at least four hours before the endoscopic procedure. Nasogastric tube suction is recommended before endoscopic procedure to reduce the risk of aspiration. Endoscopic biopsy provides histological diagnosis in specific diseases and aid in confirming or excluding malignancy.

**MANAGEMENT**

All patients with symptoms of persistent GOO need hospitalization. Intravenous volume resuscitation with normal saline; replacement of electrolytes; and measurements of electrolytes and arterial pH are the principles of fluid and electrolyte resuscitation for GOO[21]. Hypokalemia and metabolic alkalosis should be looked for and treated.

Gastric decompression by nasogastric tube should be done at admission, this procedure is useful in relieving pain and discomfort of distention in patients who have edema and spasm due to active ulceration. Nasogastric decompensation also helps to clear the stomach for endoscopic procedures and reduce gastric capacity which is essential for endoscopic examination and further surgery or endoscopic treatment. Administration of antacid (H2-receptor antagonists or proton pump inhibitors) is usually required.

**ENDOSCOPIC INTERVENTION**

A definite treatment is required if the GOO is persistent, secondary to fibrotic scarring, or an irreversible condition. In place of traditional surgical intervention or rigid esophagoscopy, therapeutic fibreoptic endoscopy is concluded to be an effective and safe treatment modality in pediatric patients[22]. Endoscopic treatment offered depended on the cause of GOO. Unless the etiology of GOO is evident from the antecedent history, such as PUD, caustic ingestion, or prior surgery, one must exclude rare diseases like Crohn disease, tuberculosis or malignancy by endoscopy and biopsy.

***EBD***

The through-the-scope (TTS) balloon catheter with variable diameter balloons are available from 6 mm to 20 mm which are inflated with a hydrostatic device attached to a pressure gauge. A radio-opaque wire can guide the stricture segment and augment the efficacy of the EBD in difficult strictures. A single and short stricture provides the best result with EBD; hence patients should be evaluated with proper imaging studies (barium studies/CT scan) before EBD procedure.

Although the devices and techniques used in the EBD procedure for pediatric patients are basically identical to those procedures in adult patients, several anatomic limitations must be addressed. Endoscopic procedure is difficult in newborns due to remarkable anatomic limitations. The length and diameter of esophagus is 8-10 cm, and around 5 mm in neonates, causing compression of trachea during endoscopic intervention. TTS balloon dilations are possibly performed in toddlers and children, but this technique is limited in neonates or small infants as there are no balloon catheters that fit through a 2-mm channel.Balloon dilatation can be performed in neonates or infants who cannot tolerate a standard-size endoscopy by using biliary dilation balloons in size of 4-10 mm and length of 2-8 cm. These balloons can be applied endoscopically with 0.035-inch guide-wires.Balloon sizes are usually increased by 2 mm for subsequent EBD sessions. The balloon catheter upon removal may reveal blood clots or bloods if an effective dilatation had been reached.

***Experiences of EBD in recent decades***

Most reports regarding EBD management of GOO were found in adult population, the technique of EBD in GOO is less performed in pediatric population. A series of case studies observed that surgical treatment can give definitive therapy in PUD related GOO without *H. pylori* infection[2]. Until the advent of EBD, surgery was the only treatment for these patients. Vagotomy with antrectomy or pyloroplasty was performed in PUD patients with GOO who were refractory to medical therapy[4].

Many evidences prove that EBD is an effective alternative in the management of GOO[17,23-39]. The EBD management combined endoscopic placement of guidewire with fluoroscopically-guided balloon dilation to treat GOO. A study of EBD of upper digestive tract stricture in 23 adult patients had gastric or pyloric strictures were evaluated. Percent of 92 were successfully dilated, with a complication rate of 3%[23]. EBD has become the first-line therapy in most of the patients with benign causes of GOO[24]. Subsequently, a number of reports appeared highlighting the safety and efficacy of the procedure[24,25,27-36].

Compared to adult patients, endoscopic management in pediatric patients is limited by their anatomical limitation with difficulty in passing endoscopy through pharyngeal inlet and difficulty to perform EBD because of smaller size of pylorus and duodenal bulb, especially in neonates or infants. Besides, a relatively higher risk of complications was noted in pediatric population. In pediatric patients, the technique of EBD treatment in esophageal stricture is relatively more mature than EBD treatment in pyloric stricture. We reviewed the endoscopic management both in adult and pediatric patients with GOO, and further point to recent evidence regarding the utility of magnifying endoscopic treatment in the management of pediatric diseases related GOO.

***PUD***

Fibrotic scarring in PUD may cause irreversible GOO which requires intervention. Traditionally, surgery has been the standard mode of treatment for PUD-related GOO. In adult series of patients with PUD-related GOO, 80%-90%s underwent surgery[40], about 60% received surgery in the first hospitalization and 20% in the subsequent hospitalizations[41]**.** Recently, many case series studies indicate that balloon dilatation is an effective alternative to surgery in adult patients with PUD-related GOO[28-35]. A major surgery and associated morbidity can be obviated with the development of TTS EBD. EBD has emerged as an effective alternative to surgery in selected groups of patients. EBD has been shown to be effective in ulcer related GOO. The performance of EBD is preferred with balloon catheters of incremental diameter gradually to achieve the end-point of 15 mm. Fluoroscopic study is not routinely used by most endoscopists although fluoroscopic evaluation it is recommended for EBD. Previous experiences indicated that the requirement of EBD varied from once a week to once in three weeks in PUD-related GOO. EBD procedure related complications are uncommon, massive bleeding is rare, perforation occurs more frequent with balloon sizes larger than 15 mm. A Review of cumulative experience in 30 patients who underwent EBD for peptic ulcer-induced GOO and had follow-up of a mean of 15 mo (range 4-28 mo), 6-18-mm (median 15-mm) balloons were inflated a median of 2 times (range 1-4 times) for a median of 60 s (range 30-180), 20 (67%) patients had one treatment and 10 (33%) had multiple treatments, 24 (80%) patients achieved sustained symptom relief. The authors concluded that EBD is safe and effective for most patients with ulcer-induced GOO[16].

In view of the confounding factors of *H. pylori* eradication, chronic usage of NSAID, use of PPI in patients with PUD-related GOO, immediate response is prominent, but with variable long-term results. EBD must be combined with eradication of *H. pylori*. Studies specify that the eradication of *H. pylori* contributed to a good long-term response in 70%-80% of patients during a follow-up period of 9-98 mo[29,321,34,37]. In an adult series, 25 patients with proven GOO secondary to PUD were managed with EBD using TTS balloons, 80 % of patients remained asymptomatic (follow-up: median 9 mo, range 2-24 mo)[39].

Literatures regarding to the use of EBD in pediatric GOO patients are limited in recent decades.Chan *et al*[42] reported 3 children (ages 2, 4, and 8 years) suffering from peptic pyloric stenosis with EBD followed by H2-receptor antagonist therapy. There were no complications due to the procedures, and no recurrences of symptoms over a follow-up of 5-30 mo (mean, 17 mo). The authors suggest that EBD is an option for the initial nonoperative treatment of pediatric peptic pyloric stenosis.

***IHPS***

IHPS is typically treated with surgical pyloromyotomy. If the child is well-hydrated with normal electrolytes, and if surgeons with expertise in the procedure are available, surgery usually takes place on the day of diagnosis. EBD had been used to treat IHPS in recent decades. EBD was considered as a safe procedure for treating IHPS infants and was recommend to be as an initial approach before pyloroplasty in such presentations[43-45].Recurrent pyloric stenosis is rare in IHPS patients after balloon dilatation[43]. However, because balloon dilatationdoes not consistently destroy the muscular ring[46], EBD is preferably reserved for patients in whom with a significant risk for general anesthesia or in whom with difficulty for surgical intervention.

***Caustic injury***

GOO is one of the most common gastric complications of caustic agent ingestion that may require surgical treatment[7-9].Sodium hydroxide and potassium hydroxide, and hydrochloric acid were the common ingested caustic agents for GOO[7].

The severity of mucosal injury at the antrum and pylorus decided the variety of surgical treatment. Moderate mucosal injury (superficial ulcerations with intact mucosa) may induce partial pyloric obstruction; severe mucosal injury (deep ulcerations, hemorrhagic erosions, eschar formation) may cause complete pyloric obstruction. For adult patients with caustic-induced GOO, surgery had been the only option available as well[47]. The standard treatment of pediatric caustic-induced GOO is surgery, gastrojejunostomy provides good long-term results with minimal morbidity particularly in patients without severe gastric injury[9]. An early surgical intervention has decreased the morbidity and mortality[7].

Adult experiences specify more difficulty of using EBD to dilate caustic-induced GOO than PUD-related GOO, besides; those patients with caustic-induced GOO have more recurrences and requiring more sessions of EBD. The mean number of sessions (range, 2-13) for caustic GOO is significantly more than the number of sessions for peptic GOO which required only 1-3 sessions[32].

Successful results of a caustic stricture by EBD management offers guidelines for the use of this procedure[37,48]. Kochhar *et al*[36] reviewed 41 patients with EBD management, 39 (95.1 %) underwent successful responses after repeated dilations with a mean (SD) of 5.8 (2.6) sessions (range, 2-13) to reach the end point of 15 mm. The mean (SD) size of the initial dilatation was 8.2 (0.6) mm (range, 8-10). Finally, 2 patients received surgery including one with perforation and the other with intractable pain every time he received EBD. Other complications included minor self-limiting pain (*n* = 8) or bleeding (*n* = 7). The authors concluded that EBD is a safe, effective, and long-term alternative to surgery for caustic GOO[37]. Another adult report (*n* = 31) of caustic GOO found all patients successfully respond to repeated dilations to reach the end point of 15 mm with a range of 3-18 (median, 9) sessions of dilations during a mean period of 7 wk (range, 1.5-16) of follow-up[49].

There are only few reports of EBD management in pediatric cases with caustic injury related GOO. A pediatric study enrolled 8 cases caustic ingestion indicated that caustic injury related GOO could be successfully treated through EBD in suitable patients, surgery can be avoided[50].

A pediatric case series of 6 children (mean age was 2.9 years, range 1.5-3 years) with caustic injury related GOO (2 ingested acid corrosives, 4 ingested alkali corrosives). Balloon dilatation of the pylorus was performed in 1 patient successfully, the others received pyloroplasty (3 patients), and Billroth I procedures (2 patients). The authors recommended early definitive surgical intervention in cases with severe pyolric stricture[51].

***Gastric antral web***

Treatment of antral web usually consisted of incision of the web and construction of a patulous gastric outlet by surgery, and most patients remained asymptomatic after operation[10]. Endoscopic treatment had been used to treat antral web, endoscopic diathermy and EBD successfully resolved the pyloric web[10,21,51]. Lu *et al*[52] successfully treated antral web with EBD in an young infant, the EBD was attempted sequentially using different sized water-inflated balloons (8, 10 and 12 mm). The stenosis was dilated with balloons incrementally to 12 mm diameter.

***Post-operative GOO***

EBD resolved the in nearly 70% of GOO patients with postvagotomy gastric outlet stenosis[30]. Lanuti *et al*[53] evaluated therole of pyloromyotomy and management with endoscopic pyloric dilatation in the patients with Post-esophagectomy GOO, the results showed that post-operative GOO could be effectively managed with endoscopic pyloric dilatation, the authors concluded that routine pyloromyotomy for the prevention of post-esophagectomy GOO may be unwarranted. Swanson *et al*[54]affirmed that EBD could obviate the requirement of pyloroplasty at esophagectomy.

The application of EBD as treatment of post-operative GOO was described in an 11-year-old boy with surgical injury to the vagus and two infants after insufficient pyloromyotomy, EBD achieved successful results and was considered a good alternative to surgery in these conditions[55].

EBD has successfully dilated the anastomotic strictures following gastric bypass surgery or vertical band gastroplasty in the patients with morbid obesity[56].

***Late-onset primary GOO***

Late-onset primary GOO in childhood is a rare condition. A series of 8 pediatric cases received succeeded in treating late-onset primary GOO by using EBD, there is no recurrence for one year[57]. Another experience successfully used EBD to treat 5 pediatric cases with late-onset pyloric stenosis, 3 cases need repeated EBD[58].

***NSAID related GOO***

NSAIDs are among the most frequently prescribed medications. Although NSAID related GOO is a rare condition, chronic NSAID consumption could cause GOO[58-60].Duodenal web-like strictures associated with long-term NSAID use has been described[61].

The literature about the role of EBD management in NSAID-induced GOO is still scarce. A case series (*n* = 10) with endoscopic management for NSAID-induced pyloroduodenal obstruction found that duodenum was the most common site of involvement (50%), followed by both pylorus and duodenum (40%) and pylorus (10%). The strictures in a majority of patients were web-like, 90% of cases were successfully treated with repeated EBD. Among these successful cases, a 15-mm balloon diameter was achieved after a mean (SD) of 2 (1.6) sessions, and a mean (SD) of 5.3 (2.7) sessions was required to during a mean period of 4.5 mo (range, 2–15). There were no complications or mortality. The literature in relation to EBD management of pediatric NSAID related GOO is scant, EBD was used to treat NSAID related GOO successfully in a child[62].

***Inflammatory conditions***

A definite treatment for the antecedent disease is crucial and may avert the need for EBD or surgery in inflammatory diseases (eosinophilic gastritis, Crohn’s disease, *etc.*) or infectious disease like tuberculosis related GOO, Crohn’s disease or tuberculosis related GOO may respond to balloon dilatation, however, multiple recurrences usually occurs if the underlying disease is not effectively treated. Gastroduodenal Crohn’s disease and tuberculosis had been successfully treated with EBD procedure[31,63].

***Complications of EBD***

In general, EBD is relatively a safe procedure with infrequent complications. Perforation and bleeding are rarely reported for balloon dilatation smaller than 15 mm. Two of 30 patients (6.7%) dilated to 18 mm suffered perforation[6]. Both recovered uneventfully after surgery.A large case series of 23 patients with PUD related stenosis encountered only one perforation with EBD management[35]. Another large series of 54 cases by Lau *et al*[28] reported 4 perforations with EBD management,2 of 16 patients who underwent EBD with a 16-mm diameter balloon encountered perforation while 2 of 3 patient with 20-mm diameter balloon had perforation. It therefore appears that EBD management with balloon diameter greater than 15 mm is more prone to be complicated with perforation.

Pain during EBD is not uncommon, but is often self-limited. A recent study observed that 19.5% of patients with caustic GOO had self-limiting pain during EBD[37]. The complications with EBD procedure observed in a case series (*n* = 31) with caustic GOO included self-limiting pain (*n* = 10), bleeding at the time of the procedure (*n* = 9), and one perforation (3.2%) who required surgery.[41] Kochhar *et al*[37] reviewed 41 corrosive injury patients with GOO could be successfully taken for EBD, and self-limiting pain (*n* = 8) or bleeding (*n* = 7), perforation (*n* =1) were noted among these patients.

***Outcome of EBD intervention***

A good result can be anticipated in the majority of patients with PUD- and corrosive-related GOO after EBD intervention[32]. EBD for benign GOO in adults is a generally accepted method of treatment. Previous literatures advocate that more than 75 % of patients with PUD-related GOO respond to EBD and the long-term use of proton pump inhibitor is needed to obviate recurrences after *H. pylori* eradication.The results of EBD for PUD-related GOO is variable because not all studies consider the confounding factors, such as *H. pylori* infection, use of NSAIDs, practice and compliance of proton pump inhibitor. Immediate relief of obstruction with EBD has been commonly found in the majority of patients, but achieved varied long-term response from 16%[36] to 100%[32]. The eradication of *H. pylori* have reported a good long-term response in 70%-80% of patients over a period of 9-98 mo[6,29,30,35,39].

Lam *et al*[38] compared the response rates of EBD between 14 patients with positive *H. pylori* infection and 11 *H. pylori*-negative, EBD management was responsive in 78.6% of *H. pylori-*positive, while only 45.4% in *H. pylori* negative patients.Eradication of *H. pylori* combined with EBD had a lower rate of ulcer complications such as bleeding or obstruction compared to *H. pylori* negative group (21% *vs* 55%) over a follow-up of 24 mo. A case series (n = 11) indicated that eradication of *H. pylori* with 1-3 sessions of EBD successfully resolved obstruction in all of the patients[32].A study by Cherian *et al*[34] indicated comparable results in long-term follow-up of their Peptic-GOO patients with EBD and drug therapy.

Patients with young age, continuous use of NSAIDs, or long-lasting symptoms requiring repeated EBD had unfavorable outcomes with the need for multiple dilations or surgery for GOO[40]. DiSario *et al*[6] observed that a long-length stricture was associated with poor outcome for GOO. The majority of studies did not describe the duration of proton pump inhibitors making comparisons incomparable between studies.

Need of more than 2 sessions of dilations is a risk factor for EBD failure and requirement for surgery. Rapid recurrence of symptoms is found in patients with malignant GOO. As many benign GOO patients had underlying PUD, eradication of *H. pylori* at the time of balloon dilation will guarantee higher long-term successful rates[25].

An adult study of 45 patients with pyloric stenos did a follow-up of mean 32 mo (range, 4-126) indicated that immediate response rate of the EBD treatment was observed in 43 cases (95.6%), and clinical remission was observed in 38 cases (84.4%)[64]. Over a period of 30 mo, no recurrence was noted in 55.8% of patients with clinical remission, relapse was observed in 39.5% of patients over a mean period of 22.9 mo. Three patients (6.7%) had complications (one bleeding and 2 perforations). Thirteen patients (29%) underwent surgery. *H. pylori* was positive in 97.7% of the patients, and 78.4% of them had successful eradication of *H. pylori*. This study further found that unsuccessful eradication of *H. pylori* and smoking were two risk factors for the recurrence of pyloric stenosis[64].

EBD can make surgery unnecessary for postoperative GOO and later for peptic, corrosive and postvagotomy gastric outlet stenosis in nearly 70% of patients with benign GOO[30]. Kochhar *et al*[37] performed EBD in 31 patients with caustic-induced gastric injury,30 (96.8%) did not have recurrence of stenosis over a mean follow-up of 21 mo (range, 3-72).

There is less experience of evaluating outcome of EBD for GOO in children. A pediatric case series (*n* = 14) evaluated the effect of endoscopic balloon dilatation and surgical treatment in children’s pyloric stricture, surgical correction is still the most common treatment in the majority of cases of pyloric stenosis[50]. In this series, the authors stated that benign GOO can be effectively and successfully treated through EBD in suitable patients, surgery can be avoided in patients with successful pyloric balloon dilatation[50]. There are two long-term studies on EBD management for children with benign pyloric stenosis**,** response rates were varied between 16% and 80%[6,36].

***Advanced techniques augmenting EBD***

A number of practitioners have used supplementary techniques to augment the efficacy of EBD. EBD could be augmented with local (intralesional) steroid injections and endoscopic incision with electrocauterization.

***Intralesional steroid injections***

Intralesional steroid injections augmented the effect of balloon dilation had been reported in patients with caustic GOO[65,66], the GOO responded with 1-2 sessions of steroid injections. Intralesional steroid injections have been illustrated to inhibit stricture formation by impeding the synthesis of collagen, chronic scarring, and fibrosis[66].Ketchum LD *et al*[67] specify that steroid (Triamcinolone) offers cross linking of collagen leading to scar contracture; the contracture will not occur if stretch of scar occurs with steroid injection. Steroids may diminish scar formation by reduction of fibrotic healing that appears after balloon dilation[68]. Efficacy of steroids augmenting EBD in GOO has been also demonstrated in the other two studies by Kochhar *et al*[69] and Lee *et al*[70] Successfully cases treated with steroids and balloon dilations included three patients with caustic GOO, one peptic, and another post-pyloroplasty.

***Endoscopic incision***
EBD with additional endoscopic incision achieved successful results in caustic-induced GOO. Boron *et al*[71] successfully used electrocauterization endoscopically to incise the stenotic segment with standard sphincterotomy in a patient with refractory pyloric stenosis. Hagiwara *et al*[72] also successfully resolved the stenosis by using combined EBD with electrosurgical incisions in the patients with refractory post-operative pyloric stenosis. I have also successfully used this technique in a young infant with refractory pyloric stenosis secondary to surgical excision of gastric antral web, a satisfactory result after 2 sessions of combined endoscopic electrocauterization and balloon dilatation was achieved[73]

**END POINT OF EBD**

No consensus has been reached on the issue of end point of EBD for GOO, especially in pediatric cases. Most experts[27,29,32-73] have used 15 mm balloons as the end point for GOO while some of them have only dilated to 10-12 mm[30,35]. Balloons of 16 mm, 18 mm and 20 mm are uncommonly used[28,30,35]. The size of balloon catheters for adult GOO was recommended to be used with step-wise manner, from 10-12mm to 12-15 mm[31,72] The EBD should be more cautiously performed on pediatric patients than on adult ones if with peptic, caustic or post-operative causes induced GOO. I usually dilate with step-wise manner of catheter balloons inflated with the use of a pressure gauge system for 60-120 s in pediatric patients. Balloon catheter sizes were increased by 2 mm for subsequent EBD sessions, from 6-8 mm to 10-12 mm in infants and toddlers, from 8-10 mm to 10-12 mm in younger children, and from 10-12 mm to 12-15 mm subsequently in older children.

**OTHER INTERVENTIONS**

***Gastric Peroral Endoscopic Pyloromyotomy***

Gastric Peroral Endoscopic Pyloromyotomy (G-POEM) is performed with similar techniques to esophageal per-oral endoscopic myotomy. Replacing traditional laparotomy and laparoscopic approaches, G-POEM provides a natural orifice procedure to incise and divide the pyloric sphincter.

Surgical pyloromyotomy has shown to be effective in reducing pyloric stenosis or gastroparesis symptoms, but it requires advanced skills for laparoscopic suturingand carries a risk of leakage and potential further narrowing of gastric outlet. Therefore, G-POEM as a less invasive treatment, is used to deal with gastroparesis recently.

Although laparoscopic pyloromyotomy is still considered as a simple, and safe treatment for pediatric IHPS, G-POEM technique is similarly simple, safe, but less invasiveness, and this procedure can be performed at outpatient department[74]**.** A case series of 10 IHPS infants (7 boys, 3 girls; aged 3-7 wk) underwent endoscopic pyloromyotomy with an electrosurgical needle knife to incise the pylorus from antral to duodenal side, most (90%) of the patients were done at outpatient department.All patients did not encounter any complications and tolerated regular feedings as they recovered from sedation.All of them were discharged on the same day of endoscopic procedure and doing well during follow-up (range, 6 mo-2 yr)[74].

A growing body of evidence suggests that G-POEM may be a salvage therapy improves gastric emptying in patients with different types of refractory gastroparesis. Those patients with refractory gastroparesis may respond to endoscopic pyloromyotomy. An adult case series of G-POEM using selective circular myotomy for patients with refractory gastroparesis symptoms due to varied cause (post-infectious, post-surgical, or idiopathic) were successfully performed without any complications. All cases experienced obvious success after G-POEM[75,76].

***Endoscopic stent***

Endoscopic stent was usually used to manage malignant GOO. As gastric or duodenal malignancy is very rare in children, there is no pediatric literature about the use of endoscopic stent for malignant GOO. Palliation of the obstructive symptoms is the primary aim of treatment in the cancer related GOO. Self-expandable metal stents have emerged as a promising treatment option[77]. Topazian *et al*[78] firstly reported endoscopic treatment of GOO with endoluminal self-expanding metallic stents (SEMSs) in 1992. In recent two decades, experiences of the use of endoscopic stents have gradually increased. Several studies have reported that patients who are having high risk for long-term GOO should undergo endoscopic stents, given its safety, minimal invasiveness, and cost-effectiveness[79,80].

***Endoscopic Mucosal Resection for gastric polyps***

Although most pediatric gastric polyps are considered benign lesions, removal of symptomatic polyps are necessary for symptom relief, histological diagnosis, and avoidance of malignant potential. A standard-size polypectomy snare can be accessed through a 2.8-mm channel endoscopically to do polypectomy in the majority of children. Pontone *et al*[81] did the endoscopic mucosal resection in the patients with multiple large antral hyperplastic polyps causing GOO with the use of a submucosal cushion under the lesion allowing a steady positioning of the polyp in the gastric lumen without further infiltration[81]. The authors concluded that endoscopic mucosal resection provides tissue for histopathology to diagnose the nature of the polyp and achieves symptomatic resolution.

***Endoscopic fragmentation for bezoars***

Surgery is the treatment of choice for tricobezoar. However, endoscopic treatments have been described, such as endoscopic fragmentation, extracorporeal lithotripsy and laparoscopic extraction[16].

**SURGERY**

Benign GOO may, however, still require operative intervention when non-operative treatment fails[82]. Peptic ulcer-induced gastric outlet obstruction can be treated safely with EBD. About 65% of patients have sustained symptom relief, but many require more than one dilation session. Outcomes may be improved with effective ulcer therapy with acid reduction and eradication of *H. pylori*[82]. Compared to endoscopic access, surgical approach is more associated with morbidity and mortality, surgery is considered to be reserved for failure of endoscopic treatment[83]. Surgeries for peptic GOO include antrectomy with vagotomy, pyloroplasty with vagotomy, gastrojejunostomy with truncal vagotomy, and pyloroplasty. In peptic GOO gastrojejunostomy can be combined with truncal vagotomy and antrectomy, gastrojejunostomy (Billroth II reconstruction) was considered in peptic GOO with altered anatomy. Laparoscopic gastrojejunostomy become a favorable modality of surgery in peptic GOO for its shorter hospitalization due to quick postoperative recovery compared with conventional laparotomy surgery [84].

**FUTURE DIRECTIONS**

With further development of technologies in therapeutic endoscopy, EBD could become the worldwide treatment of choice for pediatric GOO. The future of endoscopic treatment seems to be aimed at the combined use of endoscopic electrocauterization with balloon dilatations in intractable pyloric stricture, and G-POEM appears to be technically feasible and effective in IHPS or gastroparesis patients.

**CONCLUSION**

Correction of gastric outlet obstruction without the need for surgery is an issue that has been tried to be resolved in these decades. With the development of therapeutic endoscopy in pediatric patients, the therapeutic endoscopy becomes an integral part of the management of pediatric patients with GOO.

In recent decades; the endoscopic management of GOO has developed with EBD and additional advanced devices and techniques like local steroid injection, electrocauterization, G-POEM, and stent have been added to augment the efficacy of EBD.

With improvements in techniques and devices, therapeutic results of EBD have been achieved in pediatric patients with peptic pyloric stricture, IHPS, caustic injury related pyloric stricture, congenital antral web, post-operative GOO, and NSAID related GOO despite the inherent technical difficulties of this procedure in children. Local steroid injection and electrocauterization can augment the effect of EBD. Gastric peroral endoscopic pyloromyotomy (G-POEM) appears to be technically feasible in IHPS patients. Clinical applications of G-POEM in pediatric patients with gastroparesis can be considered after confirmation of its efficacy and safety in additional pediatric studies.**REFERENCES**

1 **Kochhar R**, Kochhar S. Endoscopic balloon dilation for benign gastric outlet obstruction in adults. *World J Gastrointest Endosc* 2010; **2**: 29-35 [PMID: 21160676 DOI: 10.4253/wjge.v2.i1.29]

2 **Patel RA**, Baker SS, Sayej WN, Baker RD. Two Cases of Helicobacter pylori-Negative Gastric Outlet Obstruction in Children. *Case Rep Gastrointest Med* 2011; **2011**: 749850 [PMID: 22606426 DOI: 10.1155/2011/749850]

3 **Yen JB**, Kong MS. Gastric outlet obstruction in pediatric patients. *Chang Gung Med J* 2006; **29**: 401-405 [PMID: 17051838]

4 **Edwards MJ**, Kollenberg SJ, Brandt ML, Wesson DE, Nuchtern JG, Minifee PK, Cass DL. Surgery for peptic ulcer disease in children in the post-histamine2-blocker era. *J Pediatr Surg* 2005; **40**: 850-854 [PMID: 15937829 DOI: 10.1016/j.jpedsurg.2005.01.056]

5 **Graham DY.** Ulcer complications and their nonoperativetreatment. In: Sleisenger M, Fordtran J (eds). Gastrointestinal Disease 1993: 698

6 **DiSario JA**, Fennerty MB, Tietze CC, Hutson WR, Burt RW. Endoscopic balloon dilation for ulcer-induced gastric outlet obstruction. *Am J Gastroenterol* 1994; **89**: 868-871 [PMID: 8198096]

7 **Ciftci AO**, Senocak ME, Büyükpamukçu N, Hiçsönmez A. Gastric outlet obstruction due to corrosive ingestion: incidence and outcome. *Pediatr Surg Int* 1999; **15**: 88-91 [PMID: 10079337 DOI: 10.1007/s003830050523]

8 **Ozokutan BH**, Ceylan H, Ertaşkin I, Yapici S. Pediatric gastric outlet obstruction following corrosive ingestion. *Pediatr Surg Int* 2010; **26**: 615-618 [PMID: 20443118 DOI: 10.1007/s00383-010-2613-6]

9 **Ozcan C**, Ergün O, Sen T, Mutaf O. Gastric outlet obstruction secondary to acid ingestion in children. *J Pediatr Surg* 2004; **39**: 1651-1653 [PMID: 15547828 DOI: 10.1016/j.jpedsurg.2004.07.008]

10 **Bell MJ**, Ternberg JL, McAlister W, Keating JP, Tedesco FJ. Antral diaphragm--a cause of gastric outlet obstruction in infants and children. *J Pediatr* 1977; **90**: 196-202 [PMID: 830910 DOI: 10.1016/s0022-3476(77)80629-x]

11 **Macpherson RI**. Gastrointestinal tract duplications: clinical, pathologic, etiologic, and radiologic considerations. *Radiographics* 1993; **13**: 1063-1080 [PMID: 8210590 DOI: 10.1148/radiographics.13.5.8210590]

12 **Ozcan C**, Celik A, Güçlü C, Balik E. A rare cause of gastric outlet obstruction in the newborn: Pyloric ectopic pancreas. *J Pediatr Surg* 2002; **37**: 119-120 [PMID: 11782002 DOI: 10.1053/jpsu.2002.29443]

13 **Sharma KK**, Ranka P, Goyal P, Dabi DR. Gastric outlet obstruction in children: an overview with report of Jodhpur disease and Sharma's classification. *J Pediatr Surg* 2008; **43**: 1891-1897 [PMID: 18926227 DOI: 10.1016/j.jpedsurg.2008.07.001]

14 **Morinville V**, Bernard C, Forget S. Foveolar hyperplasia secondary to cow's milk protein hypersensitivity presenting with clinical features of pyloric stenosis. *J Pediatr Surg* 2004; **39**: E29-E31 [PMID: 14694404 DOI: 10.1016/j.jpedsurg.2003.09.040]

15 **DuBose TM**, Southgate WM, Hill JG. Lactobezoars: a patient series and literature review. *Clin Pediatr* (Phila) 2001; **40**: 603-606 [PMID: 11758960 DOI: 10.1177/000992280104001104]

16 **Ruiz HD**, Palermo M, Ritondale O, Pest E, Pest P, Villafañe V, Bruno M, Tarsitano FJ. [Gastro-duodenal trichobezoars: a rare cause of obstruction of the gastrointestinal tract]. *Acta Gastroenterol Latinoam* 2005; **35**: 24-27 [PMID: 15954733]

17 **Nugent FW**, Roy MA. Duodenal Crohn's disease: an analysis of 89 cases. *Am J Gastroenterol* 1989; **84**: 249-254 [PMID: 2919581]

18**.** [**Padussis J**](http://www.ncbi.nlm.nih.gov/pubmed/?term=Padussis%20J%5BAuthor%5D&cauthor=true&cauthor_uid=16217956), [Loffredo B](http://www.ncbi.nlm.nih.gov/pubmed/?term=Loffredo%20B%5BAuthor%5D&cauthor=true&cauthor_uid=16217956), [McAneny D](http://www.ncbi.nlm.nih.gov/pubmed/?term=McAneny%20D%5BAuthor%5D&cauthor=true&cauthor_uid=16217956). Minimally invasive management of obstructive gastroduodenal tuberculosis. *Am Surg* 2005; **71**: 698-700 [PMID: 16217956]

19 **Miner PB**, Harri JE, McPhee MS. Intermittent gastric outlet obstruction from a pedunculated gastric polyp. *Gastrointest Endosc* 1982; **28**: 219-220 [PMID: 7129059 DOI: 10.1016/S0016-5107(82)73075-5]

20 **Jaka H**, Mchembe MD, Rambau PF, Chalya PL. Gastric outlet obstruction at Bugando Medical Centre in Northwestern Tanzania: a prospective review of 184 cases. *BMC Surg* 2013; **13**: 41 [PMID: 24067148 DOI: 10.1186/1471-2482-13-41]

21 **Ferzoco SJ,** Soybel DI. Gastric outlet obstruction, perforation and other complications of gastroduodenal ulcer. In: Wolfe HM, editor. Therapy of digestive disorders. 2007: 357-375

22 **Goenka AS**, Dasilva MS, Cleghorn GJ, Patrick MK, Shepherd RW. Therapeutic upper gastrointestinal endoscopy in children: an audit of 443 procedures and literature review. *J Gastroenterol Hepatol* 1993; **8**: 44-51 [PMID: 8439662 DOI: 10.1111/j.1440-1746.1993.tb01174.x]

23 **Lindor KD**, Ott BJ, Hughes RW. Balloon dilatation of upper digestive tract strictures. *Gastroenterology* 1985; **89**: 545-548 [PMID: 4018500 DOI: 10.1016/0016-5085(85)90449-4]

24 **Rana SS**, Bhasin DK, Chandail VS, Gupta R, Nada R, Kang M, Nagi B, Singh R, Singh K. Endoscopic balloon dilatation without fluoroscopy for treating gastric outlet obstruction because of benign etiologies. *Surg Endosc* 2011; **25**: 1579-1584 [PMID: 21052720 DOI: 10.1007/s00464-010-1442-y]

25 **Yusuf TE**, Brugge WR. Endoscopic therapy of benign pyloric stenosis and gastric outlet obstruction. *Curr Opin Gastroenterol* 2006; **22**: 570-573 [PMID: 16891891 DOI: 10.1097/01.mog.0000239874.13867.41]

26 **Benjamin SB**, Cattau EL, Glass RL. Balloon dilation of the pylorus: therapy for gastric outlet obstruction. *Gastrointest Endosc* 1982; **28**: 253-254 [PMID: 7173580 DOI: 10.1016/S0016-5107(82)73105-0]

27 **Benjamin SB**, Glass RL, Cattau EL, Miller WB. Preliminary experience with balloon dilation of the pylorus. *Gastrointest Endosc* 1984; **30**: 93-95 [PMID: 6714610 DOI: 10.1016/S0016-5107(84)72329-7]

28 **Lau JY**, Chung SC, Sung JJ, Chan AC, Ng EK, Suen RC, Li AK. Through-the-scope balloon dilation for pyloric stenosis: long-term results. *Gastrointest Endosc* 1996; **43**: 98-101 [PMID: 8635729 DOI: 10.1016/S0016-5107(06)80107-0]

29 **Boylan JJ**, Gradzka MI. Long-term results of endoscopic balloon dilatation for gastric outlet obstruction. *Dig Dis Sci* 1999; **44**: 1883-1886 [PMID: 10505729 DOI: 10.1023/A: 1018807125952]

30 **Solt J**, Bajor J, Szabó M, Horváth OP. Long-term results of balloon catheter dilation for benign gastric outlet stenosis. *Endoscopy* 2003; **35**: 490-495 [PMID: 12783346 DOI: 10.1055/s-2003-39664]

31 **Misra SP**, Dwivedi M. Long-term follow-up of patients undergoing ballon dilation for benign pyloric stenoses. *Endoscopy* 1996; **28**: 552-554 [PMID: 8911802 DOI: 10.1055/s-2007-1005553]

32 **Kochhar R**, Sethy PK, Nagi B, Wig JD. Endoscopic balloon dilatation of benign gastric outlet obstruction. *J Gastroenterol Hepatol* 2004; **19**: 418-422 [PMID: 15012779 DOI: 10.1111/j.1440-1746.2003.03283.x]

33 **Perng CL**, Lin HJ, Lo WC, Lai CR, Guo WS, Lee SD. Characteristics of patients with benign gastric outlet obstruction requiring surgery after endoscopic balloon dilation. *Am J Gastroenterol* 1996; **91**: 987-990 [PMID: 8633593]

34 **Cherian PT**, Cherian S, Singh P. Long-term follow-up of patients with gastric outlet obstruction related to peptic ulcer disease treated with endoscopic balloon dilatation and drug therapy. *Gastrointest Endosc* 2007; **66**: 491-497 [PMID: 17640640 DOI: 10.1016/j.gie.2006.11.016]

35 **Kozarek RA**, Botoman VA, Patterson DJ. Long-term follow-up in patients who have undergone balloon dilation for gastric outlet obstruction. *Gastrointest Endosc* 1990; **36**: 558-561 [PMID: 2279642 DOI: 10.1016/S0016-5107(90)71163-7]

36 **Kuwada SK**, Alexander GL. Long-term outcome of endoscopic dilation of nonmalignant pyloric stenosis. *Gastrointest Endosc* 1995; **41**: 15-17 [PMID: 7698619 DOI: 10.1016/S0016-5107(95)70270-9]

37 **Kochhar R**, Dutta U, Sethy PK, Singh G, Sinha SK, Nagi B, Wig JD, Singh K. Endoscopic balloon dilation in caustic-induced chronic gastric outlet obstruction. *Gastrointest Endosc* 2009; **69**: 800-805 [PMID: 19136104 DOI: 10.1016/j.gie.2008.05.056]

38 **Lam YH**, Lau JY, Fung TM, Ng EK, Wong SK, Sung JJ, Chung SS. Endoscopic balloon dilation for benign gastric outlet obstruction with or without Helicobacter pylori infection. *Gastrointest Endosc* 2004; **60**: 229-233 [DOI: 10.1016/S0016-5107(04)01569-X]

39 **Griffin SM**, Chung SC, Leung JW, Li AK. Peptic pyloric stenosis treated by endoscopic balloon dilatation. *Br J Surg* 1989; **76**: 1147-1148 [PMID: 2597970 DOI: 10.1002/bjs.1800761112]

40 **Weiland D**, Dunn DH, Humphrey EW, Schwartz ML. Gastric outlet obstruction in peptic ulcer disease: an indication for surgery. *Am J Surg* 1982; **143**: 90-93 [PMID: 7053661 DOI: 10.1016/0002-9610(82)90135-0]

41 **Jaffin BW**, Kaye MD. The prognosis of gastric outlet obstruction. *Ann Surg* 1985; **201**: 176-179 [PMID: 3970597 DOI: 10.1097/00000658-198502000-00007]

42 **Chan KL**, Saing H. Balloon catheter dilatation of peptic pyloric stenosis in children. *J Pediatr Gastroenterol Nutr* 1994; **18**: 465-468 [PMID: 7915308 DOI: 10.1097/00005176-199405000-00011]

43 **Nasr A,** Ein SH, Connolly B. Recurrent pyloric stenosis: to dilate or operate? A preliminary report. *J Pediatr Surg* 2008; **43**: E17-20 [PMID: 18280264 DOI: 10.1016/j.jpedsurg.2007.10.039]

44 **Karnsakul W**, Cannon ML, Gillespie S, Vaughan R. Idiopathic non-hypertrophic pyloric stenosis in an infant successfully treated via endoscopic approach. *World J Gastrointest Endosc* 2010; **2**: 413-416 [PMID: 21191516 DOI: 10.4253/wjge.v2.i12.413]

45 **Ogawa Y**, Higashimoto Y, Nishijima E, Muraji T, Yamazato M, Tsugawa C, Matsumoto Y. Successful endoscopic balloon dilatation for hypertrophic pyloric stenosis. *J Pediatr Surg* 1996; **31**: 1712-1714 [PMID: 8986998 DOI: 10.1016/S0022-3468(96)90059-7]

46 **Hayashi AH**, Giacomantonio JM, Lau HY, Gillis DA. Balloon catheter dilatation for hypertrophic pyloric stenosis. *J Pediatr Surg* 1990; **25**: 1119-1121 [PMID: 2273424 DOI: 10.1016/0022-3468(90)90744-T]

47 **Chaudhary A**, Puri AS, Dhar P, Reddy P, Sachdev A, Lahoti D, Kumar N, Broor SL. Elective surgery for corrosive-induced gastric injury. *World J Surg* 1996; **20**: 703-76; discussion 706 [PMID: 8662156 DOI: 10.1007/s002689900107]

48 **Treem WR**, Long WR, Friedman D, Watkins JB. Successful management of an acquired gastric outlet obstruction with endoscopy guided balloon dilatation. *J Pediatr Gastroenterol Nutr* 1987; **6**: 992-996 [PMID: 3681588 DOI: 10.1097/00005176-198711000-00031]

49 **Kochhar R**, Poornachandra KS, Dutta U, Agrawal A, Singh K. Early endoscopic balloon dilation in caustic-induced gastric injury. *Gastrointest Endosc* 2010; **71**: 737-744 [PMID: 20363415 DOI: 10.1016/j.gie.2009.11.038]

50 **Temiz A**, Oguzkurt P, Ezer SS, Ince E, Gezer HO, Hicsonmez A. Management of pyloric stricture in children: endoscopic balloon dilatation and surgery. *Surg Endosc* 2012; **26**: 1903-1908 [PMID: 22234589 DOI: 10.1007/s00464-011-2124-0]

51 **Tekant G**, Eroğlu E, Erdoğan E, Yeşildağ E, Emir H, Büyükünal C, Yeker D. Corrosive injury-induced gastric outlet obstruction: a changing spectrum of agents and treatment. *J Pediatr Surg* 2001; **36**: 1004-1007 [PMID: 11431765 DOI: [10.1053/jpsu.2001.24725](http://dx.doi.org/10.1053/jpsu.2001.24725)]

52 **Lu JP**, Huang Y, Wu J, Chen SY. Uncommon congenital antral web misdiagnosed twice as a pyloric ulcer: successful treatment with endoscopic balloon dilatation. *Turk J Pediatr* 2014; **56**: 100-102 [PMID: 24827957]

53 **Lanuti M**, de Delva PE, Wright CD, Gaissert HA, Wain JC, Donahue DM, Allan JS, Mathisen DJ. Post-esophagectomy gastric outlet obstruction: role of pyloromyotomy and management with endoscopic pyloric dilatation. *Eur J Cardiothorac Surg* 2007; **31**: 149-153 [PMID: 17166733 DOI: 10.1016/j.ejcts.2006.11.010]

54 **Swanson EW**, Swanson SJ, Swanson RS. Endoscopic pyloric balloon dilatation obviates the need for pyloroplasty at esophagectomy. *Surg Endosc* 2012; **26**: 2023-2028 [PMID: 22398960 DOI: 10.1007/s00464-012-2151-5]

55 **Heymans HS**, Bartelsman JW, Herweijer TJ. Endoscopic balloon dilatation as treatment of gastric outlet obstruction in infancy and childhood. *J Pediatr Surg* 1988; **23**: 139-140 [PMID: 3343648 DOI: 10.1016/S0022-3468(88)80142-8]

56 **Sataloff DM**, Lieber CP, Seinige UL. Strictures following gastric stapling for morbid obesity. Results of endoscopic dilatation. *Am Surg* 1990; **56**: 167-174 [PMID: 2316938]

57 **Boybeyi O**, Karnak I, Ekinci S, Ciftci AO, Akçören Z, Tanyel FC, Senocak ME. Late-onset hypertrophic pyloric stenosis: definition of diagnostic criteria and algorithm for the management. *J Pediatr Surg* 2010; **45**: 1777-1783 [PMID: 20850620 DOI: 10.1016/j.jpedsurg.2010.04.014]

58 **Geraghty RJ**, Black D, Bruce SA. The successful medical management of gastric outflow obstruction associated with the use of non-steroidal anti-inflammatory drugs in the elderly. *Postgrad Med J* 1991; **67**: 1004-1007 [PMID: 1775405 DOI: 10.1136/pgmj.67.793.1004]

59 **Weaver GA**, Harper RL, Storey JA, Jenkins PL, Merrell NB. Nonsteroidal antiinflammatory drugs are associated with gastric outlet obstruction. *J Clin Gastroenterol* 1995; **20**: 196-198 [PMID: 7797825 DOI: 10.1097/00004836-199504000-00006]

60 **Kannan S**, McGreevy PS, Fullerton TE. Nonsteroidal anti-inflammatory drug induced duodenal web. *S D J Med* 1997; **50**: 393-394 [PMID: 9401436]

61 **Puri AS**, Monga R, Garg S, Sharma BC, Satapathy S, Sarin SK. Diaphragm disease of duodenum following long-term NSAIDs use: endoscopic management. *Indian J Gastroenterol* 2004; **23**: 189-190 [PMID: 15599008]

62 **Gobbi D**, Billi P, Fascetti Leon F, Alvisi P, Lambertini A, Lima M. Pneumatic pyloric dilatation for the treatment of gastric outlet obstruction in a child. *Pediatr Int* 2013; **55**: 382-385 [PMID: 23782371 DOI: 10.1111/ped.12022]

63 **Kim JH**, Shin JH, Di ZH, Ko GY, Yoon HK, Sung KB, Song HY. Benign duodenal strictures: treatment by means of fluoroscopically guided balloon dilation. *J Vasc Interv Radiol* 2005; **16**: 543-548 [PMID: 15802456 DOI: 10.1097/01.RVI.0000150033.13928.D4]

64 **Hamzaoui L**, Bouassida M, Ben Mansour I, Medhioub M, Ezzine H, Touinsi H, Azouz MM. Balloon dilatation in patients with gastric outlet obstruction related to peptic ulcer disease. *Arab J Gastroenterol* 2015; **16**: 121-124 [PMID: 26440958 DOI: 10.1016/j.ajg.2015.07.004]

65 **Kochhar R**, Sriram PV, Ray JD, Kumar S, Nagi B, Singh K. Intralesional steroid injections for corrosive induced pyloric stenosis. *Endoscopy* 1998; **30**: 734-736 [PMID: 9865568 DOI: 10.1055/s-2007-1001400]

66 **Ashcraft KW**, Holder TM. The expeimental treatment of esophageal strictures by intralesional steroid injections. *J Thorac Cardiovasc Surg* 1969; **58**: 685-691 passim [PMID: 5348158]

67 **Ketchum LD**, Smith J, Robinson DW, Masters FW. The treatment of hypertrophic scar, keloid and scar contracture by triamcinolone acetonide. *Plast Reconstr Surg* 1966; **38**: 209-218 [PMID: 5919604 DOI: 10.1097/00006534-196609000-00005]

68 **Gandhi RP,** Cooper A, Barlow BA. Successful management of esophageal strictures without resection or replacement. *J Pediatr Surg* 1989; **24**: 745-749; discussion 749-750 [DOI: 10.1016/S0022-3468(89)80529-9]

69 **Kochhar R**, Ray JD, Sriram PV, Kumar S, Singh K. Intralesional steroids augment the effects of endoscopic dilation in corrosive esophageal strictures. *Gastrointest Endosc* 1999; **49**: 509-513 [PMID: 10202068 DOI: 10.1016/S0016-5107(99)70052-0]

70 **Lee M**, Kubik CM, Polhamus CD, Brady CE, Kadakia SC. Preliminary experience with endoscopic intralesional steroid injection therapy for refractory upper gastrointestinal strictures. *Gastrointest Endosc* 1995; **41**: 598-601 [PMID: 7672557 DOI: 10.1016/S0016-5107(95)70199-0]

71 **Boron B**, Gross KR. Successful dilatation of pyloric stricture resistant to balloon dilatation with electrocautery using a sphinctertome. *J Clin Gastroenterol* 1996; **23**: 239-241 [PMID: 8899513 DOI: 10.1097/00004836-199610000-00020]

72 **Hagiwara A**, Sonoyama Y, Togawa T, Yamasaki J, Sakakura C, Yamagishi H. Combined use of electrosurgical incisions and balloon dilatation for the treatment of refractory postoperative pyloric stenosis. *Gastrointest Endosc* 2001; **53**: 504-508 [PMID: 11275897 DOI: 10.1067/mge.2001.113281]

73 **Chao HC**, Luo CC, Wang CJ. Elimination of postoperative pyloric stricture by endoscopic electrocauterization and balloon dilatation in an infant with congenital antral web. *Pediatr Neonatol* 2011; **52**: 106-109 [PMID: 21524632 DOI: 10.1016/j.pedneo.2011.02.005]

74 **Ibarguen-Secchia E**. Endoscopic pyloromyotomy for congenital pyloric stenosis. *Gastrointest Endosc* 2005; **61**: 598-600 [PMID: 15812419 DOI: 10.1016/S0016-5107(05)00075-1]

75 **Khashab MA**, Stein E, Clarke JO, Saxena P, Kumbhari V, Chander Roland B, Kalloo AN, Stavropoulos S, Pasricha P, Inoue H. Gastric peroral endoscopic myotomy for refractory gastroparesis: first human endoscopic pyloromyotomy (with video). *Gastrointest Endosc* 2013; **78**: 764-768 [PMID: 24120337 DOI: 10.1016/j.gie.2013.07.019]

76 **Mekaroonkamol P,** Li LY, Dacha S, Xu Y, Keilin SD, Willingham FF, Cai Q. Gastric peroral endoscopic pyloromyotomy (G-POEM) as a salvage therapy for refractory gastroparesis: a case series of different subtypes. *Neurogastroenterol Motil* 2016; Epub ahead of print [PMID: 27197717 DOI: 10.1111/nmo.12854]

77 **van Hooft J**, Mutignani M, Repici A, Messmann H, Neuhaus H, Fockens P. First data on the palliative treatment of patients with malignant gastric outlet obstruction using the WallFlex enteral stent: a retrospective multicenter study. *Endoscopy* 2007; **39**: 434-439 [PMID: 17516350 DOI: 10.1055/s-2007-966338]

78 **Topazian M**, Ring E, Grendell J. Palliation of obstructing gastric cancer with steel mesh, self-expanding endoprostheses. *Gastrointest Endosc* 1992; **38**: 58-60 [PMID: 1377147 DOI: 10.1016/S0016-5107(92)70334-4]

79 **Adler DG**. Enteral stents for malignant gastric outlet obstruction: testing our mettle. *Gastrointest Endosc* 2007; **66**: 361-363 [PMID: 17643713 DOI: 10.1016/j.gie.2006.12.053]

80 **Tringali A**, Didden P, Repici A, Spaander M, Bourke MJ, Williams SJ, Spicak J, Drastich P, Mutignani M, Perri V, Roy A, Johnston K, Costamagna G. Endoscopic treatment of malignant gastric and duodenal strictures: a prospective, multicenter study. *Gastrointest Endosc* 2014; **79**: 66-75 [PMID: 23932009 DOI: 10.1016/j.gie.2013.06.032]

81 **Pontone S**, Pironi D, Eberspacher C, Pontone P, Filippini A. Endoscopic management of multiple large antral hyperplastic polyps causing gastric outlet obstruction. *Ann Ital Chir* 2011; **82**: 297-300 [PMID: 21834480]

82 **Søreide K**, Sarr MG, Søreide JA. Pyloroplasty for benign gastric outlet obstruction--indications and techniques. *Scand J Surg* 2006; **95**: 11-16 [PMID: 16579249]

83 **Khullar SK**, DiSario JA. Gastric outlet obstruction. *Gastrointest Endosc Clin N Am* 1996; **6**: 585-603 [PMID: 8803569]

84 **Al-Rashedy M**, Dadibhai M, Shareif A, Khandelwal MI, Ballester P, Abid G, McCloy RF, Ammori BJ. Laparoscopic gastric bypass for gastric outlet obstruction is associated with smoother, faster recovery and shorter hospital stay compared with open surgery. *J Hepatobiliary Pancreat Surg* 2005; **12**: 474-478 [PMID: 16365822 DOI: 10.1007/s00534-005-1013-0]

**P-Reviewer:** Rustagi T, Tomizawa M, Tanimoto MA **S-Editor:** Qi Y **L-Editor: E-Editor:**

 **Table 1 Etiology of gastric outlet obstruction in children**

|  |
| --- |
| Idiopathic hypertrophic pyloric stenosisPeptic ulcer diseaseCaustic injuryCongenital causesGastric autral webDuplication cystEctopic pancreasAunular panaeasGastric volvulus |
| Inflammatory causesCholecystitisPancreatitisEosinophilic gastritis Crohn’s diseaseTuberculosis |
| NSAID induced stricture |
| Iatrogenic (secondary to surgery)Post-anastomosis stricturePost-pylorotomyPost-esophagectomyPost-vagotomy |
| Polyps/tumorsHyperplastic polypInflammatory polypAdenomyomaInflammatory myofibroblastomaLymphoma |
| Other causesBezoars (lactobezoar, trichobezoar)Cytomegalovirus infectionLate onset primary gastric outlet obstructionIdiopathic gastric outlet obstructionIdiopathic or acquired gastric volvulusFoveolar cell hyperplasia  |