

Gastric Outlet Obstruction in Pediatric Patients

Ju-Bei Yen, MD; Man-Shan Kong¹, MD

Background: This study reports the etiologies, management and outcome of children with gastric outlet obstruction (GOO) in a children's hospital.

Methods: The medical records of 11 children with GOO not associated with idiopathic hypertrophic pyloric stenosis (IHPS) were reviewed. They were categorized into one group of anatomic abnormality (AA group) and one group of peptic ulcer disease (PD group). One case underwent episodes of GOO caused by anatomic abnormality and peptic ulcer disease, respectively.

Results: Six cases belonged to the AA group. Mean age was 58 months with a male to female ratio of two to four. Underlying etiologies were prepyloric mass (2), web (2) and gastric volvulus (2). Four patients underwent surgery. One patient was lost to follow-up. GOO did not recur in the follow-up period (mean duration 24 months) in the remaining cases. One case in the AA group and the remaining five patients composed the PD Group. Mean age was 49 months and all were male. Underlying causes were gastric ulcers (4) and chronic duodenal ulcers (2). Two of the five patients had *Helicobacter pylori* infection found by rapid urease test. Four patients recovered after medical management and another two, with normal serum gastrin levels, underwent surgery because of poor response to medical treatment. One case was lost to follow-up. No recurrence of GOO was noted in the follow-up period (mean duration 27 months) in the remaining cases.

Conclusions: In our study, peptic ulcer disease was as important as anatomic abnormalities as the etiology for GOO not associated with IHPS, and medical management could release GOO caused by it. Compared to adult patients, *H. pylori* infection played a less important etiologic role in pediatric patients with GOO. (*Chang Gung Med J* 2006;29:401-5)

Key words: gastric outlet obstruction, anatomic abnormality, peptic ulcer disease.

Gastric outlet obstruction (GOO) is a classic indication for surgery in complicated peptic ulcer diseases.⁽¹⁾ In children, GOO is a rare condition and the most common cause is idiopathic hypertrophic pyloric stenosis (IHPS).⁽²⁾ Other causes include peptic ulcer diseases, motility disorders and anatomic lesions, such as atresia, stenosis, web and diaphragm. We retrospectively reviewed the medical records of

patients with GOO without IHPS treated at our hospital over a period of eight years. The clinical and laboratory characteristics, treatment and outcome of these patients were analyzed.

METHODS

Between July 1992 and June 2000, 142 patients

From the Department of Pediatrics, Chang Gung Memorial Hospital, Chiayi; ¹Department of Pediatrics, Chang Gung Children's Hospital, Taipei.

Received: Sep. 21, 2005; Accepted: Mar. 7, 2006

Correspondence to: Dr. Ju-Bei Yen, Department of Pediatrics, Chang Gung Memorial Hospital, 5 West, Chia-Pu Road, Putz City, Chiayi, Taiwan 613, R.O.C. Tel.: 886-5-362100 ext. 2536; Fax: 886-5-3623002; E-mail: irishcorners@yahoo.com.tw

with GOO were admitted to our hospital. Eleven cases (7.75%) were diagnosed with GOO not caused by IHPS by ultrasound (US), upper gastrointestinal (UGI) endoscopy or UGI series, and one case underwent two episodes of GOO. They were categorized into one group with anatomic abnormalities (AA group) and the other with peptic ulcer diseases (PD group). One case underwent episodes of GOO caused by anatomic abnormality and peptic ulcer disease, respectively. In the PD group, five patients were tested for *Helicobacter pylori* infection using a rapid urease test (CLO test, Delta West Pty Ltd, Bentley, Western Australia), and two patients had their serum gastrin levels measured.

RESULTS

Six cases belonged to the AA group (Table 1). Mean age was 58 months and the male to female

ratio was two to four. Their major presentations were nonbilious vomiting and abdominal distension, and the time interval between symptom onset and diagnosis was usually less than one week. The underlying causes were: prepyloric mass (one ectopic pancreas, one hyperplastic polyp), antral or prepyloric web (two) and gastric volvulus (one recurrent gastric volvulus, one with diaphragmatic hernia). Three patients underwent surgery and one underwent endoscopic polypectomy. No further interventions were performed for the case with an ectopic pancreas or the case with simple gastric volvulus. The mean follow-up period was 24 months.

One case in the AA group and the remaining five patients, total six cases, made up the PD group (Table 2). Their mean age was 54 months and all were male. The time interval between symptom onset and diagnosis was often more than two weeks. The major presentation was nonbilious vomiting.

Table 1. Etiologies and Management of Pediatric Patients with Gastric Outlet Obstruction due to Anatomic Abnormalities

Patient	Age	Gender	Diagnosis	Surgical intervention	Duration of symptoms before diagnosis	Follow-up
1	5 mo	M	Gastric polyp	Endoscopic polypectomy	1 wk	4 yr 5 mo
2	14 yr 8 mo	F	Ectopic pancreas	ND	6~7 yr	10 mo
3	4 d	M	Prepyloric web	Web excision with pyloroplasty	3 d	3 yr 2 mo
4	8 yr 5 mo	F	Pyloric web	Web excision with pyloroplasty	6 mo	1 yr 2 mo
5	1 yr 7 mo	F	Diaphragmatic hernia with gastric volvulus	Reduction of gastric volvulus and repair of diaphragmatic hernia	1 d	1 mo
6	4 yr	F	Gastric volvulus	ND	1 d	2 yr 3 mo

Abbreviations: yr: year; mo: month; wk: week; d: day; M: male; F: female; ND: not done.

Table 2. Clinical Findings and Management of Cases with Gastric Outlet Obstruction due to Peptic Ulcer Diseases

Patient No	Age	Gender	Associated disease	Rapid urease test	Serum gastrin pg/mL	Surgical intervention	Medical therapy	Symptoms	Follow up
1	5 mo,	M	Gastric ulcer	(-)	ND	ND	Cimetidine, 7 d coffee ground vomitus, 4 d	Vomiting with	4 yr 5 mo
7	1 yr 3 mo,	M	Gastric ulcer	(-)	27.9	∨	Antacids & Cimetidine, 28 d	Vomiting, 10 d	10 mo
8	1 yr 9 mo,	M	Gastric ulcer	(+)	48.3	∨	Cimetidine, 10 d	Vomiting, 1 mo	2 mo
9	3 yr,	M	Gastric ulcer	(+)	ND	ND	Cimetidine, 24 d; then Omeprazole, 7 d	Abdominal pain and vomiting, 2 wk	lost
10	9 yr 4 mo,	M	Chronic duodenal ulcer	ND	ND	ND	Antacids, 3 d	Vomiting, 2 wk	7 yr 5 mo
11	11 yr 4 mo,	M	Chronic duodenal ulcer	(-)	ND	ND	Omeprazole, 14 d	Vomiting, 2 wk	3 yr

Abbreviations: yr: year; mo: month; wk: week; d: day; M: male; F: female; (+): positive; (-): negative; ND: not done; ∨: bilateral truncal vagotomy and pyloroplasty.

GOO with gastric ulcers (four cases) and chronic duodenal ulcers (two cases) were diagnosed by endoscopy during or after the episodes of GOO. After medical treatment with antacids, cimetidine or omeprazole, four patients showed clinical improvement; one was found to have *H. pylori* infection after performing a CLO test. The other two cases underwent bilateral truncal vagotomy and pyloroplasty due to their poor response to medical treatment. Their serum gastrin levels were 48.3 and 27.9 pg/mL, respectively (normal range: 25-115 pg/mL). *H. pylori* infection was reconfirmed in one patient by a histological study after surgery and no antibiotic therapy was given afterwards. Apart from one patient who was lost to follow-up, the mean follow-up duration of the remaining patients was 38 months and no recurrence of GOO was noted. It appeared that younger male children with peptic ulcer diseases had more GOO than other patients.

DISCUSSION

Anatomic lesions in the prepyloric or antral area of the stomach could cause GOO. Symptoms of gastric atresia, stenosis or web often develop in infancy. However, in this series, one case with web was not diagnosed until the patient was school age, and we assumed her condition was not congenital in nature. In fact, some studies report that gastric webs can be acquired.⁽³⁾ In addition, epidermolysis bullosa or esophageal atresia may be accompanied by gastric atresia or webs⁽⁴⁾ but such a condition was not noted in our two patients. Prepyloric tumors were found in two cases (hyperplastic polyps and ectopic pancreas). Gastric tumors are uncommon in children⁽⁵⁾ and hyperplastic polyps are reported to be the most commonly identified gastric polyps in children.⁽⁶⁾ Another study showed that an ectopic pancreas, not common in children, may occur in 1~2% of autopsies⁽⁷⁾ and the most likely symptom is epigastric pain.⁽⁸⁾ Furthermore, gastrointestinal bleeding, obstruction or malignant transformation could develop in long-term follow-up, and surgical removal was suggested under such circumstances.⁽⁹⁾ Gastric volvulus is abnormal rotation of one part of the stomach around another.⁽¹⁰⁾ Some congenital defects, such as diaphragmatic defects,⁽¹¹⁾ asplenic syndrome⁽¹²⁾ or wandering spleen⁽¹³⁾ may be noted with gastric volvulus. In our two cases with gastric volvulus, one had diaphrag-

matic hernia and the other suffered from two episodes of acute gastric volvulus with spontaneous resolution.

GOO is a common complication of peptic ulcer diseases in adults⁽¹⁾ but it rarely occurs in childhood. In peptic ulcer diseases, GOO is usually caused by a combination of edema, spasm, fibrotic stenosis and gastric atony.⁽¹⁴⁾ Chan et al. reported their experience with 32 children with duodenal ulcers.⁽¹⁵⁾ Only one had GOO and no special risk factor was identified.⁽¹⁵⁾ Huang et al. reported on their seven child patients with ulcer-induced GOO in a medical center in Taiwan. The age range of those patients, who underwent surgical treatment, was from five to 43 months.⁽¹⁴⁾ In our six patients with GOO induced by peptic ulcer diseases, four were less than three years old and all were male. It appears that younger male children with peptic ulcer diseases have more GOO than others. GOO caused by peptic ulcer diseases can be resolved by medical treatment,^(14,16) vagotomy, pyloroplasty⁽¹⁶⁾ or endoscopic balloon catheter dilatation.^(17,18) However, the duration of medical treatment before surgical intervention was not determined. Before using omeprazole with/without antibiotics, five out of seven patients reported by Huang et al. underwent surgery after treatment with cimetidine lasting 12 to 46 days.⁽¹⁴⁾ Weiland et al. suggested that failure to respond to medical treatment within five days was an indication for surgical treatment.⁽¹⁹⁾ In our patients, four out of six patients gained spontaneous resolution of GOO after medical management (including antacids, cimetidine or omeprazole) lasting three to 31 days. The data suggest that a longer duration of medical management may be needed. Apart from this, reversal of GOO after eradication of *H. pylori* infection has been reported.⁽²⁰⁾ The two cases with *H. pylori* infection that we observed in this series were not treated with antibiotic therapy: one had clinical improvement with medical management and the other underwent surgical intervention eventually. According to our data, we propose that *H. pylori* infection in children with GOO plays a less important role than it does in adults.

According to our study, except for IHPS, peptic ulcer diseases were as important as anatomic abnormalities in the etiologies of GOO in pediatric patients. Female predominance in the AA group and male predominance in the PD group was observed. The time interval between onset of symptoms and

diagnoses was shorter in the AA group. Surgical intervention was often needed for treatment of GOO in the AA group.

REFERENCES

1. Behrman SW. Management of complicated peptic ulcer disease. *Arch Surg* 2005;140:201-8.
2. Sharma KK, Agrawal P, Toshniwal H. Acquired gastric outlet obstruction during infancy and childhood: a report of five unusual cases. *J Pediatr Surg* 1997;32:928-30.
3. van der Reis L, Fisher JH, Baker M. The pyloric septum: acquired or congenital? *Aust N Z J Med* 1977;7:302-3.
4. Shaw DW, Fine JD, Piacquadio DJ, Greenberg MJ, Wang-Rodriguez J, Eichenfield LF. Gastric outlet obstruction and epidermolysis bullosa. *J Am Acta Dermatol* 1997;36:304-10.
5. Dehner LP. *Pediatric Surgical Pathology*. 2nd ed. Baltimore: Lippincott, Williams & Wilkins Co., 1987: 339-46.
6. Attard TM, Yardley JH, Cuffari C. Gastric polyps in pediatrics: an 18-year hospital-based analysis. *Am J Gastroenterol* 2002;97:298-301.
7. Murphy S, Shaw K, Blanchard H. Report of three gastric tumors in children. *J Pediatr Surg* 1994;29:1202-4.
8. Strobel CT, Smith LE, Fonkalsrud EW, Isenberg JN. Ectopic pancreatic tissue in the gastric antrum. *J Pediatr* 1978;92:586-8.
9. Özcan C, Çelik A, Güçlü C, Güçlü C, Balik E. A rare cause of gastric outlet obstruction in the newborn: Pyloric ectopic pancreas. *J Pediatr Surg* 2002;37:119-20.
10. Walker WA, Durie PR, Hamilton JR, Walker-Smith JA, Watkins JB. *Pediatric Gastrointestinal Disease: Pathophysiology, Diagnosis, Management*. 3rd ed. Hamilton: B.C. Decker Inc., 2000:378-82.
11. Elhalaby EA, Mashaly EM. Infants with radiologic diagnosis of gastric volvulus: are they over-treated? *Pediatr Surg Int* 2001;17:596-600.
12. Aoyama K, Tateishi K. Gastric volvulus in three children with asplenic syndrome. *J Pediatr Surg* 1986;21:307-10.
13. Garcia JA, Garcia-Fernandez M, Romance A, Sanchez JC. Wandering spleen and gastric volvulus. *Pediatr Radiol* 1994;24:535-6.
14. Huang FC, Chuang JH, Ko SF. Clinical experience in the treatment of ulcer-induced gastric outlet obstruction in seven children. *Acta Paediatr Taiwan* 2000;41:189-92.
15. Chan KL, Tam PK, Saing H. Long-term follow-up of childhood duodenal ulcers. *J Ped Surg* 1997;32:1609-11.
16. Feng J, Gu W, Li M, Yuan J, Weng Y, Wei M, Zhou X. Rare causes of gastric outlet obstruction in children. *Pediatr Surg Int* 2005;21:635-40.
17. Chan KL, Saing H. Balloon catheter dilatation of peptic pyloric stenosis in children. *J Pediatr Gastroenterol Nutr* 1994;18:465-8.
18. Lam YH, Lau JY, Fung TM, Ng EK, Wong SK, Sung JJ, Chung SS. Endoscopic balloon dilatation for benign gastric outlet obstruction with or without *Helicobacter pylori* infection. *Gastrointest Endosc* 2004;60:229-33.
19. Weiland D, Dunn DH, Humphrey EW, Schwartz ML. Gastric outlet obstruction in peptic ulcer disease: an indication for surgery. *Am J Surg* 1982;143:90-3.
20. de Boer WA, Driessen WM. Resolution of gastric outlet obstruction after eradication of *Helicobacter pylori*. *J Clin Gastroenterol* 1995;21:329-30.

兒童非自發性胃幽門肌肉肥厚的胃出口阻塞

顏如貝 江文山¹

背景： 本研究報告在一個兒童醫院的病童罹患胃出口阻塞的病因、處置和結果。

方法： 從西元 1992 年 7 月到西元 2000 年 6 月，共有 142 個病童藉由超音波、內視鏡或鋇劑消化道攝影被證實罹患胃出口阻塞，我們分析他們的病歷紀錄做成此研究。排除 130 位罹患自發性胃幽門肌肉肥厚的病人和 1 位罹患胃幽門肌肉痙攣的病人，共有 11 位病人選入本研究中。他們被分成解剖學異常組和潰瘍性疾病組。有 1 位病人各因解剖學異常組和潰瘍性疾病共發生兩次胃出口阻塞。在潰瘍性疾病組中有五位病人接受快速尿素酶試驗來偵測是否有胃幽門螺旋桿菌感染，有兩位病人抽血測試血液中胃泌素濃度。

結果： 解剖學異常組共有 6 個病例，其平均診斷年齡為 58 個月大，男女比例為 2 比 4。病因有胃幽門前腫瘤 (2)，網狀組織 (2) 及胃扭轉 (2)。4 個病人接受手術治療。有 1 個病人未繼續追蹤，其他病人在追蹤過程中 (平均追蹤期間為 24 個月) 沒有復發胃出口阻塞。潰瘍性疾病組共有 6 個病例，其平均診斷年齡為 49 個月大，全都為男性。病因有胃潰瘍 (4) 和慢性十二指腸潰瘍 (2)。5 個病人接受快速尿素酶試驗，其中有 2 個病人證實有胃幽門螺旋桿菌感染。4 個病人藉由藥物治療即得到緩解另外兩位則接受手術才復原他們血液中的胃泌素濃度為正常。1 個病人未繼續追蹤，其他病人在追蹤過程中 (平均追蹤期間為 27 個月) 沒有復發胃出口阻塞。

結論： 排除自發性胃幽門肌肉肥厚，在我們的病人中造成胃出口阻塞的主因為潰瘍性疾病。在我們的研究中，如果是因解剖學異常造成胃出口阻塞，通常需要用手術方法來解決。如果是因潰瘍性疾病造成，通常使用較長時期的藥物可以解決胃出口阻塞。在我們的病人中，胃幽門螺旋桿菌感染不具有重要角色。
(長庚醫誌 2006;29:401-5)

關鍵字： 胃出口阻塞，解剖學異常，潰瘍性疾病。

長庚紀念醫院 嘉義院區 小兒科，¹長庚紀念醫院 林口院區 小兒科

受文日期：民國94年9月21日；接受刊載：民國95年3月7日

通訊作者：顏如貝醫師，長庚紀念醫院 嘉義院區 小兒科。嘉義縣613朴子市嘉朴路西段6號。Tel: (05)3621000 ext. 2536; Fax: (05)3623002; E-mail: corner@adm.cgmh.org.tw