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— Case Report —

Gastric acid secretion in a child with postbulbar duodenal ulcer

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Summary: A 14-year-old case was reported with a primary postbulbar duodenal ulcer, which was confirmed by barium meal study and duodenoscopy. In the preoperative study, the patient showed marked gastric hyperacidity: maximal and peak acid output were 0.980 and 1.434 mEq/kg/hr, respectively. As previously described, hyperacidity appears to be a main factor in the pathogenesis of postbulbar duodenal ulcer. Fasting and postprandial serum gastrin secretion was not thought to be responsible for gastric hyperacidity in the present case. Upon histological investigation, the operatively resected stomach did not suggest a possible relationship between hyperacidity and an enlarged parietal cell mass. *Gastroenterol Jpn 1989;24:707–710*

Key words: *child; gastric acid; gastrins; parietal cell; postbulbar duodenal ulcer*

Introduction

Primary postbulbar duodenal ulcer is relatively rare in adults and its clinical features are known to be unique compared with bulbar ulcers of the duodenum. In the pediatric field, there have been only a few reports of postbulbar duodenal ulcers[1-3], and to our knowledge, no detailed studies on the gastric acid secretion of the patients. We report one pediatric case of primary postbulbar duodenal ulcer with concomitant multiple bulbar ulcers. Furthermore, the preoperative and postoperative gastric acid secretion, and serum gastrin response stimulated by test meal are discussed.

Case Report

A 14-year-old boy was admitted to the hospital in May 1988 with a 2-month history of intermittent epigastric pain. The pain occurred especially at night, resulting in frequent sleep disturbance, yet his symptoms seemed not to be aggravated by fasting. Three years ago, he had epigastric discomfort and was diagnosed as anemia. In April 1986, he also complained of severe epigastric pain and hematemesis, and was diagnosed as having a duodenal ulcer.

On admission, barium meal study demonstrated the dilatation of the duodenal bulb, its narrowing and an ulcer crater in the postbulbar area (**Fig. 1**). Duodenoscopy using on Olympus GIF XP10 revealed multiple ulcerations in the bulb and a larger ulceration with a white patch distal to the superior duodenal angle, where it was stenotic (**Fig. 2**).

Sampling of gastric secretion was performed using a no. 16 Salem Sump Tube (Argyle) after overnight fasting, pre- and post-operatively. Gastric contents for basal acid secretion were collected for 45 min and then those for gastrin-stimulated maximal acid secretion (Gastopsin 4 μg/kg, intramuscularly) for 60 min at 15-min intervals. Gastric acid concentration (mEq/l) of aliquots of the 15-min collections was measured by titration to pH 7 using 0.1N NaOH. Basal

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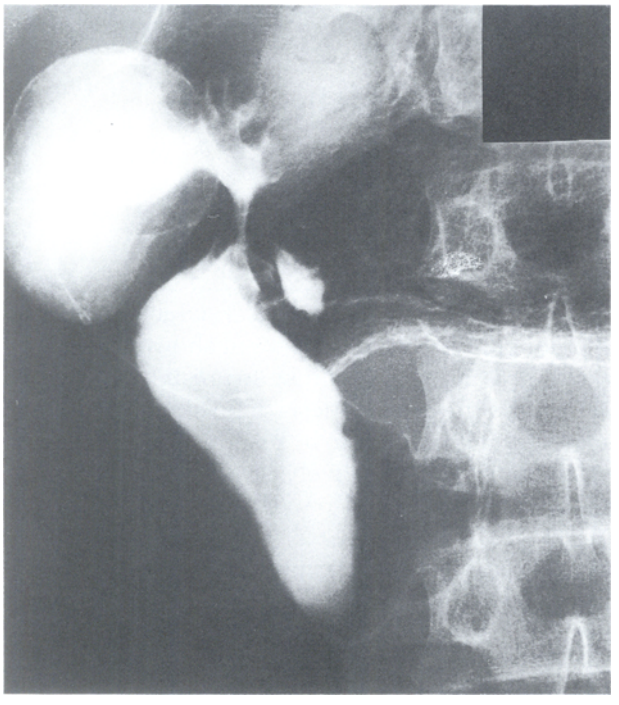

Fig. 1 Preperative barium-contrast roentgenogram. An ulcer crater and narrowing of the lumen are shown in the second portion of the duodenum.

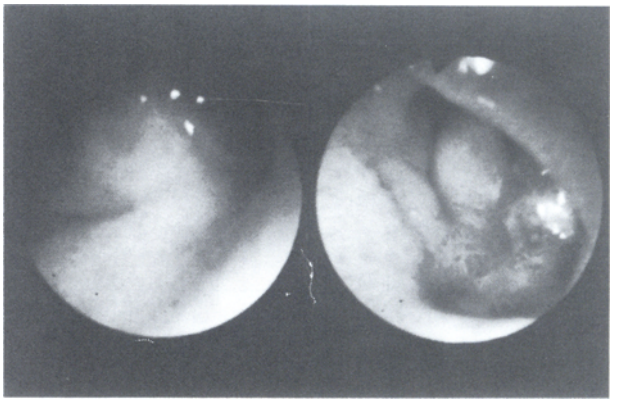

Fig. 2 Duodenoscopy demonstrates the stenosis distal to the bulb of the duodenum (left) and the ulceration in the postbulbar area (right).

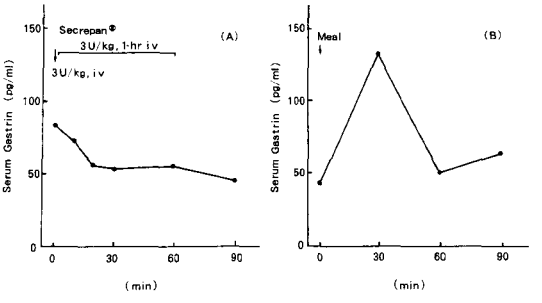

Fig. 3 Preoperative study of serum gastrin responses. Gastrin secretion was stimulated by injection of secretin (A) and test meal (B).

(BAO), maximal (MAO) and peak acid output (PAO) were expressed as mEq/hr and mEq/kg/hr, respectively. Responses of serum gastrin were investigated by injection of secretin (Secrepan, intravenous injection of 3U/kg followed by a 1-hr infusion of 3U/kg) and test meal (200g of rice and two eggs) during the fasting period. Serum gastrin level (pg/ml) was evaluated using radioimmunoassay (Gastrin RIAKIT II, Dinabot).

Laboratory data revealed the following: hemoglobin 12.5 g/dl, white blood cells 6.9 × $10^3/mm^3$, liver and renal functions were normal. The CT scan of the abdomen revealed no abnormal findings. The secretin test showed no paradoxical response of serum gastrin (**Fig. 3A**), which ruled out Zollinger-Ellison syndrome. Serum gastrin stimulated by test meal slightly increased up to 134pg/ml (**Fig. 3B**). When compared with the results of the age-matched control study by Christie et al. (mean values of BAO and MAO, 0.035 and 0.248 mEq/kg/hr)[4], gastric acid secretion test in the preoperative state revealed hyperacidity in the patient (**Table 1**). In particular, gastrin-stimulated MAO was of a markedly high value 0.980 mEq/kg/hr and the MAO/BAO ratio was 10.5.

A combination of cimetidine 400 mg twice a day and sucralfate was administered, as the lesion was generally thought to be intractable to medical treatment. Repeat endoscopy performed one month following admission indicated the bulbar scars and almost healed postbulbar ulceration of the duodenum. However, the postbulbar lumen was more stenotic, although it was possible to insert the endoscope to the distal side. Afterward, because of the duodenal stenosis and the marked gastric hyperacidity, he underwent distal gastrectomy with gastrojejunostomy. Unfortunately, panperito-

Table 1 Preoperative and postoperative gastric acid secretion

	mEq/hr			mEq/kg/hr		
	BAO	MAO	PAO	BAO	MAO	PAO
Preoperative	3.4	35.8	52.3	0.093	0.980	1.434
Postoperative	1.4	2.6	3.0	0.031	0.057	0.065
% Decrease of acid output				66.7%	94.2%	95.5%

nitis due to leakage from the duodenal stump occurred which resulted in another operation. Since then his condition has been stable.

Gastric acid secretion was also postoperatively studied. The result was summarized in **Table 1**. The BAO and MAO decreased to 33.3 and 5.8% of the preoperative values, respectively. Histological investigation of the specimen of the resected stomach revealed that the distance between the pyloric ring and the proximal border of the pyloric gland was 5.5 cm on the lesser curvature.

Discussion

Postbulbar ulceration of the duodenum, especially multiple, may be associated with Zollinger-Ellison syndrome[5]. However, chronic postbulbar duodenal ulcers, when not due to the underlying disease, are very rare in children[1-3] and there are few detailed reports. In adult studies, the incidence of postbulbar duodenal ulcers range from 3.8 to 17%[6-9]. The diagnosis of postbulbar ulcers is difficult and frequently requires more than two barium meal studies[6]. Endoscopic examination cannot be performed in some cases with postbulbar ulcers because of the stenosis of the duodenal lesions. Consequently, this clinical entity can be misdiagnosed, hence, the accurate incidence in children remains to be establised. In the past seven years (1981-1988), we found only one postbulbar ulcer patient among a total of endoscopically-confirmed 31 patients with duodenal ulcers (3%).

In general, patients with postbulbar ulcers show gastric hyperacidity, which appears to be a main factor in the pathogenesis of ulcer formation. In gastric acid studies of pediatric duodenal ulcers, the ulcer group had significantly higher values of MAO and PAO than the non-ulcer controls[4,10]. However, it is disputable whether postbulbar ulcers are associated with higher gastric acidity as opposed to bulbar ulcers of the duodenum[11,12]. The present case showed marked gastric hyperacidity: particularly, tetragastrin-stimulated acid output was high. As previously reported, duodenal ulcer patients revealed excessive gastric acidity during fasting or meal-stimulated conditions[13]. The serum gastrin response by test meal in this patient was similar to the data of control study by Yamashiro et al[14]. Therefore, serum gastrin appears not to be responsible for hyperacidity in this case. MAO is generally thought to have a positive correlation to the patietal cell mass and it is possible that high MAO represents an enhanced parietal cell function. With regard to the parietal cell mass, the histological distance between the proximal border of the pyloric gland and the pyloric ring on the lesser curvature was investigated in bulbar and postbulbar ulcer patients[12]. The results showed there was no significant difference between both groups, although the distance in 4 of 6 postbulbar ulcer patients was within 4 cm. An antrum with the distance within 4 cm on the lesser curvature was reported to be rare[15,16]. This fact may lead to the possible relationship between gastric hyperacidity and the enlarged parietal cell mass in postbulbar ulcers. Although our patient was not age-matched with adult studies, he did not necessarily have a larger parietal cell mass than other duodenal ulcer patients.

It remains controversial whether this type of ulcer should be initially treated by a surgical approach[6,17-19]. Unless associated with complications such as massive bleeding, stenosis or

perforation, the ulceration may need medical means for the time being. However, we should recognize that postbulbar ulcer is intractable to medical treatment and frequently results in duodenal hemorrhage[17,18]. Surgical procedures in adults include vagotomy with pyloroplasty[20] or anterectomy[12], or partial gastrectomy[6,18]. It will require further investigation to estimate which of them is preferable in children.

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