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DOUBLE PYLORUS ACCOMPANIED BY GASTRIC ULCER RESISTANT TO H2-RECEPTOR ANTAGONIST -A CASE REPORT AND REVIEW OF THE LITERATURE-

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INDEXING WORDS

double pylorus: gastric ulcer; H2-receptor antagonist

SYNOPSIS

A 48-year-old female was admitted to our hospital with epigastralgia. Endoscopy revealed an ulcer on the posterior wall of the greater curvature near the pyloric ring and a fistula, distinct from the pyloric ring and adjacent to it. We showed the fistula to connect the antrum and the duodenal bulb by running a cannula through it. We therefore diagnosed this as a double pylorus accompanied by gastric ulcer. We treated the ulcer with an H2 receptor antagonist, but effected no improvement. We therefore administered some prostagladin E1 derivatives as well, and progress is now under observation. Both congenital and acquired origins have been proposed for the double pylorus; we believe this case to have been acquired. We also review 65 cases of double pylorus from the Japanese literature.

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INTRODUCTION

Double pylorus is a relatively rare disease state in which the patient has a passage parallel to and distinct from the true pylorus. Reports of this disease have increased with the frequency of use of gastroduodenal X-rays and endoscopy. We here report a case of double pylorus next to a gastric ulcer which resisted treatment; we also review the Japanese literature.

CASE REPORT

A 48-year-old female was admitted to the Hyogo Medical Center for Adults with epigastralgia. Since about 1985, the patient had sometimes suffered from hunger pains, which were alleviated by eating, and she had not done anything except for taking over-the-counter medication. Around August 1987, the hunger pain in her epigastrium recurred, but this time it was not alleviated by over-the-counter medications. The patient began to be awakened by the pain at night. Her appetite was good. She had a stool once a day, and had not been drinking alcohol or smoking tobacco.

She was 151 cm tall and weighed 41.5 kg. Her blood pressure was 150/84 mmHg and her pulse 72/min; there was no anaemia or jaundice in the eye conjunctiva; no abnormalities were found in the heart, lungs, but oppressive pain was noticed in the epigastrium, and no swelling of the liver or spleen was palpable.

No abnormalities were found in the peripheral blood examinations, and the gastrin value was within the normal range (Table 1).

In the upper gastrointestinal series; barium study (fluoroscopic radiograph) shows an indentation in the greater curvature of the prepyloric area and a somewhat rigidity of the wall. In the compression picture of the same region, a shallow, irregularly-shaped, excavative lesion was found in the antropyloric area, and the mucosal folds were found to converge in the lesser curvature (Fig 1).

At the time of the first observation on September 8, 1987, gastroscopy revealed a shallow, irregularly-shaped ulcer on the posterior wall of the greater curvature near the pyloric ring. Its circumferential mucous membrane was surrounded by a reddish ring

Table 1 laboratory findings

• Urinalysis			• Blood chemistry		
protein	−		TP	8.4	g/dl
sugar	−		Alb	4.5	g/dl
uroblinogen	normal		A/G	1.22	
sediment	normal		T Bil	0.8	
			TTT	1.9	
• Stool			GOT	23	IU/l
Occult blood	(−)		GPT	25	IU/l
			CHE	152	IU/l
• Peripheral blood			ALP	122	IU/l
RBC	438	$\times 10^4$	LAP	14	IU/l
WBC	5000		γ −GTP	34	IU/l
Hb	11.4	g/dl	LDH	346	IU/l
Ht	35.8	%	T. chol	265	IU/l
Plt.	32.6	$\times 10^4$	Amylase	101	IU/l
			BUN	21.5	mg/dl
			Creat.	1.12	mg/dl
			Gastrin	100	PG/ml
			• Tumor marker		
			AFP	10	ng/ml
			CEA	3.2	ng/ml

that seemed to be composed of regenerating epithelium. The marginal elevation of the ulcer remained a litlle close to the greater curvature, and in contact with it was a small ulcer. A fistula was distinct from and adjacent to the pyloric ring. It was anal to and separated a little from the ulcer. Passage of a cannula through the fistula resulted in its appearance in the bulbus of the duodenum. We began treatment for the gastric ulcer with on H2 receptor antagonist (Famotidine, 40 mg/day). The second look gastroscopy on October 20, 1987, revealed worsening of the ulcer and its expansion into the pyloric ring, in spite of the

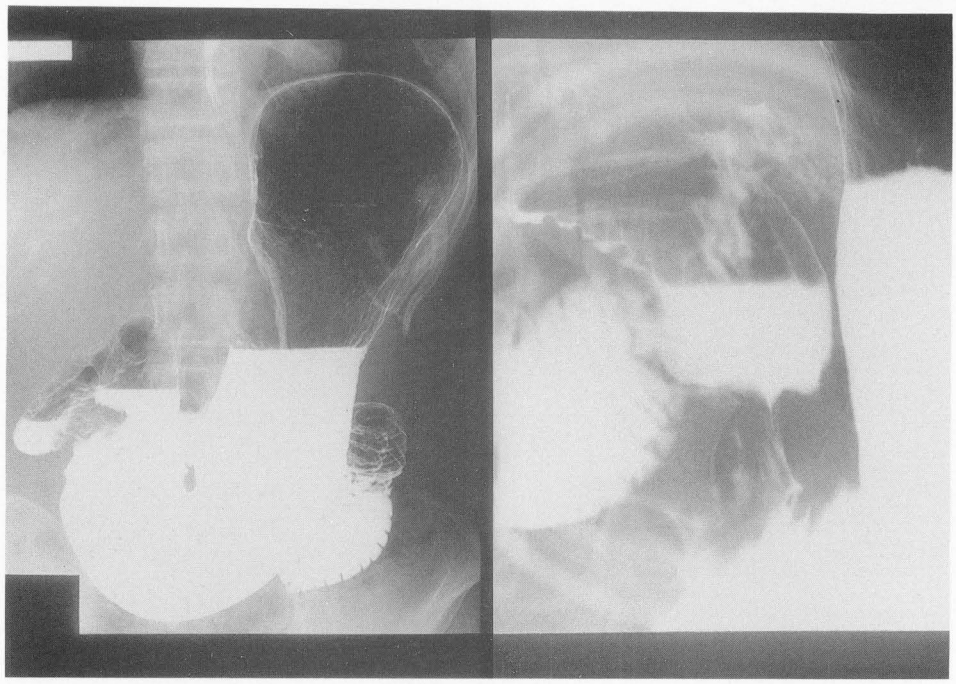

Fig. 1 Upper gastrointestinal series
Left : barium-filled X-ray picture
Right : compression X-ray picture

Famotidine. We began to administer Ranitidine, 300 mg/day, as well. About two months later, on January 14, 1988, the third look gastroscopy was carried out. However, no improvement of the ulcer was found (Fig. 2). Thus, we added a prostaglandin E_1 derivative to the treatment regimen and the progress is now under observation.

DISCUSSION

Double pylorus is a structural abnormality of the gastric system in which a passage other than the pylorus connects the antropyloric area and the duodenal bulb. This was reported by Mohr et al.[9] in 1942 as a complication of ulcers in the antropyloric area. Recently, reports of double pylori have indicated increasingly frequent detecion as gastroduodenal X-rays and endoscopic

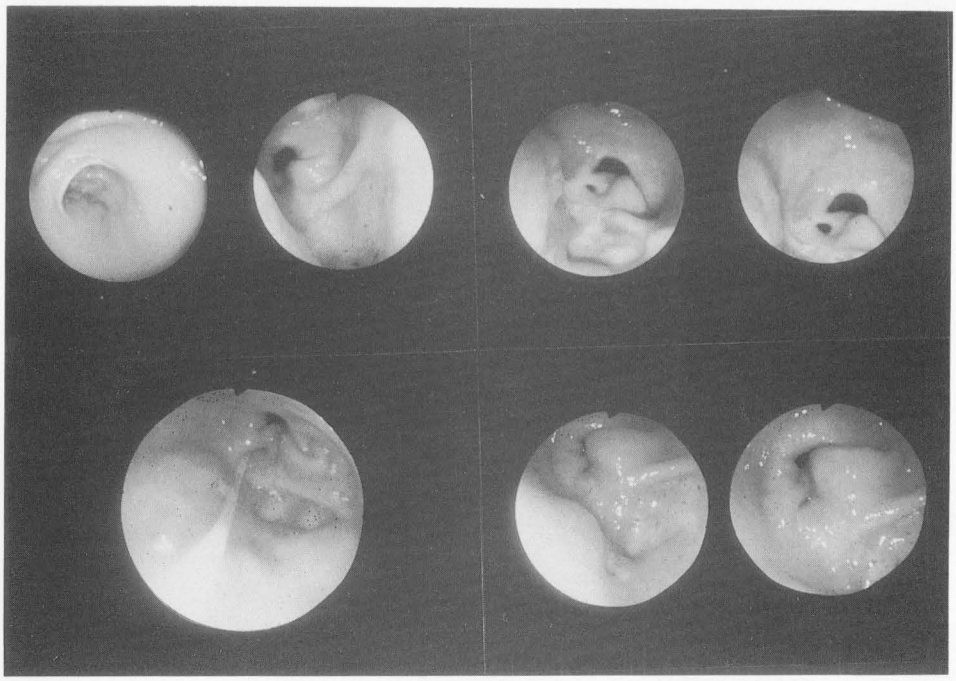

Fig. 2 a: The first endoscopic examination on September 8, 1987
b: The picture shows the endoscopic cannula which was inserted from the second channel and was observed through the true pyloric channel.
c: The second endoscopic examination October 20, 1987
d: The third endoscopic examination on January 14, 1988

examination have been prevelant. Since the report by Matuki et al.[8] in 1977, we have done a careful investigation of reports published in Japan and found 65 cases. Adding our own single case brings the total to 66. Table 2 shows their characteristics: their average age was 61.4 (range 38 to 82); there were males and females, and the aged males account for far the largest number; the location of the fistula varied; 49 cases in the lesser curvature, 12 in the greater curvature, 1 on the anterior wall, and 4 without any region stated. Most were thus in the lesser curvature. They occurred as a complication of ulcers; 34 cases with a gastric ulcer, 10 with a duodenal ulcer, and 16 complicated both by a gastric and a duodenal ulcer. Thus, 90.9% occurred with

Table 2 The cases of double pylorus reported in Japan

No.	Auther		Age	Sex	Site of fistula	Accompanying ulcer	Underlying disease	Ulcergenic drug	Etiology
1	Matsuki	1977	43	M	L.C.	GU			
2	Tashiro	1977	49	M	L.C.	GU			
3	Fukuhara	1978	57	M	not described	GU	gastric cancer		A
4	Kohno	1979	67	M	G.C.	GU			A
5	Ohkubo	1980	73	F	L.C.	DU	RA	steroid	
6	Asaki	1980	59	M	L.C.	GU	hypertension		
7	Shebena	1980	71	M	L.C.	GU			Ⓐ
8	Tamura	1980	53	M	L.C.	GU	Zollinger-Ellison Synd.		C
9	Kubo	1980	68	M	G.C.	(-)			A
10	Saitoh	1980	69	M	L.C.	GU+DU	RA		A
11	Tamura	1980	56	M	L.C.	GU+DU			A
12	Ohmori	1980	60	M	L.C.	GU		analgesic	A
13	Kitada	1981	63	M	L.C.	GU+DU			A
14	Kitada	1981	53	M	L.C.	DU			A
15	Ohdate	1981	76	M	L.C.	GU		analgesic	A
16	Katsumine	1981	62	F	L.C.	DU			A
17	Niwa	1982	41	M	L.C.	GU	DM, hyperlipidemia		
18	Hashimoto	1982	68	M	L.C.	(-)			
19	Satoh	1982	38	F	G.C.	(-)			A
20	Yamazaki	1982	71	M	G.C.	DU			C
21	Nakano	1982	68	M	L.C.	GU+DU			Ⓐ
22	Munehisa	1982	50	F	L.C.	GU	SLE	steroid	Ⓐ
23	Suto	1982	40	F	L.C.	not described			A
24	Matuo	1983	72	M	L.C.	GU+DU	DM		Ⓐ
25	Horiuchi	1983	53	F	L.C.	GU	SLE	steroid	Ⓐ
26	Katayama	1983	51	M	L.C.	GU	DM		Ⓐ
27	Satoh	1983	59	M	L.C.	DU			A
28	Shaura	1983	56	M	L.C.	DU	RA, hypertension	steroid	Ⓐ
29	Mibayashi	1983	77	M	L.C.	GU+DU	pyothorax		Ⓐ
30	Shinohara	1983	68	M	A.W.	GU			Ⓐ
31	Takahashi	1983	71	M	L.C.	GU+DU			
32	Izumi	1984	40	F	G.C.	GU			A
33	Aseda	1984	78	M	G.C.	GU		analgesic	
34	Kimura	1984	56	M	L.C.	GU+DU			A
35	Watanabe	1984	71	F	not described	GU	RA	steroid	A
36	Shinozaki	1984	67	M	L.C.	GU	lung cancer		Ⓐ
37	Nagayoshi	1984	77	F	not described	GU			A
38	Kida	1984	60	F	L.C.	GU	RA	steroid	Ⓐ
39	Ishii	1984	53	M	L.C.	GU+DU			Ⓐ
40	Ishii	1984	62	F	L.C.	DU	RA		
41	Yokota	1984	68	F	L.C.	GU			
42	Nakazima	1984	51	M	L.C.	DU			
43	Ichikura	1984	41	M	L.C.	GU	splenic vein thrombosis		
44	Ishine	1984	71	F	L.C.	GU	RA	steroid	Ⓐ
45	Sakurada	1984	59	M	L.C.	GU			A
46	Tamura	1984	58	M	G.C.	GU			A
47	Ohishi	1985	72	M	L.C.	GU+DU	DM		Ⓐ
48	Jou	1985	47	M	L.C.	GU+DU			A
49	Kinoshita	1985	81	F	G.C.	GU			A
50	Sugiura	1985	69	M	L.C.	GU			Ⓐ
51	Tei	1985	47	M	L.C.	GU+DU			A
52	Terada	1985	73	M	L.C.	(-)	choledocholithiasis		
53	Hirose	1985	50	M	L.C.	GU			Ⓐ
54	Kumada	1985	71	M	L.C.	GU	choroiditis	steroid	A
55	Matsumoto	1986	41	M	L.C.	GU+DU			A
56	Matsumoto	1986	73	M	L.C.	GU			A
57	Hoshino	1986	74	M	L.C.	GU			A
58	Murata	1986	82	M	L.C.	DU			A
59	Nakaseto	1986	45	M	G.C.	GU			
60	Torii	1986	77	F	G.C.	GU+DU	RA DM	analgesic	A
61	Shiraki	1986	79	M	G.C.	(-)	RA		C
62	Ishii	1986	62	F	L.C.	GU			A
63	Kin	1986	41	M	not described	GU+DU			Ⓐ
64	Nagai	1986	67	M	L.C.	GU+DU		analgesic	Ⓐ
65	Satoh	1987	62	M	L.C.	DU			Ⓐ
66	Yoshimura	1988	48	F	G.C.	GU			A

GU : gastric ulcer DU : duodenal ulcer LC : lesser curvature GC : greater curvature
AW : anterior wall Ⓐ : acquired (observed either befor and after fistula formation)
A : acquired C : congenital

ulcers and in 5 cases no ulcer was reported (a proportion of 7.6%), and one case was not described.

Both congenital and acquired origins have been suggested. The congenital hypothesis calls for canal formation[4,5] due to gastric duplication and an aberrant pancreas[3]. Clinical support for this opinion includes the following 1,5,7,10): ulcers and ulcerous scars are unnoticeable; the septum between the 2 passages is bandlike; the bypass is long and situated in the greater curvature; both outlets have about the same diameter and perform the normal shrinking mortion, etc. In addition, histologically, the normal mucosa of the pyloric band, gastric glands, and

muscularis mucosa are observable, and the absence of the muscle layer is characteristic[7,12]. Conversely, acquired cases would most likely arise from the perforation of a gastric or duodenal ulcer. Rokitansky[11] advocates the hypothesis that if an ulcer exists in the neighborhood of the pylorus and is in contact with the duodenal bulb both regions would conglutinate, and, furthermore, that if the ulcerous bottoms of both were perforated a gastroduodenal fistula would be formed. If a double pylorus of acquired origin is due to perforation, the presence of a fistula before confirmation of the double pylorus must be first disproved. And the existence of ulcers and scars in the environs of the double pylorus do suggest acquired origins. However, even through congenital double pylori occur, they may represent only a state of reduced resistance 2) that easily leads to ulceration and may not necessarily confirm acquired origins. Furthermore, one report[6] of a pathological presentation considered to be an acquired fistula based on the observation of pre-post fistula formation indicated the same as that of the congenital double pylorus. Hence, differentiating congenital and acquired fistulas is not easy. Among the reports published in Japan, there were 3 diagnoses of congenital fistulas out of 65 cases and 48 diagnoses of acquired fistulas including 19 cases in which pre-post fistula formation was observed. In addition, there were 14 cases of undecided origin. Our present case was diagnosed as acquired double pylorus based on the following observations: the history of ulcerous symptoms and of complications stemming from pyloric ulcer, the narrower bypass compared with the pylorus, and an absence of shrinking motion. Nevertheless, congenital origins cannot be ruled out completely.

The 66 cases of double pylorus included the following basic systemic diseases: 8 cases of chronic articular rheumatism; 4 of diabetes; 2 of systemic lupus erythematosus; and one of both chronic articular rheumatism and diabetes. Of the cases that were subject to chronic articular rheumatism as a basic disease, those which had received steroid and anti-inflammatory analgetica over a long period accounted for the majority. One might duduce an association of these drugs with the formation of double pylorus. In addition, there were found 5 cases of diabetes, one of which was complicated with chronic articular rheumatism, and in diabetes the lack of microcirculation in the mucous membrane may have

caused the ulcer. We conclude that these basal diseases and these drugs have an important relationship with the origin of this symptom. To treat this symptom, surgery was used mostly on the post cases, but recently conservative therapy has been used in practically all cases. Especially after initiation of H2 receptor antagonist therapy, conservative therapy were often carried out. However, in some cases this fails. It may be inferred that since the double pylorus does not perform the same function as does the physiological pylorus, it causes the duodenum to be exposed to gastric juice, and that bilious regurgiation causes a state in which ulcers occur readily. Consequently, a concurrent gastric ulcer may resist treatment, as in the present case. On the other hand, some cases found[13] have shown a favorable clinical course after prolonged administration of anti-ulcer medicines. Therefore, we would advise that surgery should be applied according to the co-existing ulcer's adaptability to surgical repair.

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