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Early Gastric Cancer Accompanied by Perforated Peritonitis

Tatsuo HIRANO, Toshio MIURA, Hiroyuki KUSANO, Yukio ICHIKAWA, Masayuki OHBATAKE, Masaaki JIBIKI, Masafumi MORINAGA, Terumitsu SAWAI, Yutaka FUKUDA, Teruhisa SHIMIZU, Tohru NAKAGOE, Takatoshi SHIMOYAMA, Masao TOMITA

The First Department of Surgery, Nagasaki University School of Medicine

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ABSTRACT : An exceedingly rare case of perforated early gastric cancer is reported. A 47-year-old man developed a peritonitis due to perforation of early gastric cancer. Emergency operation was performed and followed by uneventful recovery. The histologic examination of the surgical specimen showed early gastric cancer (EGC) of type III, composed of signet ring cell carcinoma. Thirteen years after surgery, the patient died of a cerebrovascular accident with no evidence of tumor recurrence. The rarity of this complication in EGC is discussed and the Japanese literature reviewed.

INTRODUCTION

Early gastric cancer (EGC) occasionally present symptoms mimicking peptic ulcer such as bleeding, and rarely pyloric stenosis. Although a large number of patients with EGC have been operated on nationwide in Japan, there have been only 28 case reports of EGC deveoloping perforated peritonitis in the Japanese literature.

No cases have been described outside Japan. Here we report an additional case of EGC associated with such a rare complication.

CASE REPORT

A 47-year-old man (patient number : 9493) was admitted to the First Department of Surgery, Nagasaki University Hospital on May 11, 1964 because of severe epigastric pain of a sudden onset with 14 hours' duration. He had had a dyspepsia and melena 6 years before, and since then suffered from dyspepsia. His family history was not contributory. Physical examination on admission revealed that the patient was alert but obviously acutely ill. His blood pressure was 160/110 mmHg, pulse 76/min, regular. His abdomen was slightly prominent, rigid and tender diffusely. Rebound tenderness was noted especially in the epigastrium. The hepatic dullness was present. The white blood cell count was 14,000. The chest film taken in the upright position revealed no free air under the diaphragm. The patient was transferred to the operating room with the diagnosis of acute peritonitis probably due to a perforated peptic ulcer. At laparotomy, there was a perforation, measuring 5×5 mm at the gastric antrum and remarkable inflammatory changes including purulent peels were noted around the stomach. The operative diagnosis was a perforated gastriculcer, and Billroth II gastrectomy was carried out with no lymph node dissection. The postoperative convalescence was uneventful. Thirteen years later, he died of cerebral hemorrhage with no cliniclal evidence of cancer recurrence.

On macroscopic findings, as shown in Fig. 1, the resected stomach showed an apparently

benign ulcer with a 5×5 mm perforation. There was a remarkable shortening of the stomach along the lesser curvature with a thick and hard induration. The macroscopic diagnosis was peritonitis due to perforation of the benign gastric ulcer. There was no evidence showing gastric cancer on gross findings. Microscopic examination of the lesion, however, revealed signet ring cell carcinoma which was located in the margin adjacent to the ulcerated edge, being confined to the mucosa (Fig.2). The ulcer was of callosum type associated with a remarkable fibrosis through the stomach wall. The gastric lesion was finally diagnosed as a perforated EGC of typical Type III.[3)]

DISCUSSION

EGC simulating peptic ulcer without macroscopically definitely recognized cancer area, as seen in our case, is classified as EGC of type III. Since EGC is defined as gastric cancer confined to the mucosa or mucosa and submucosa [3)] (regardless of lymph node metastasis,) the portion of EGC *per se* can not be perforated. In other words, it is the excavated ulcerated portion that develops perforation, and at the ulcerated portion no cancer exists. Therefore, this complication of EGC, virtually identical with that of peptic ulcer, occurs in EGC which is always associated at least with a component of type III −deep peptic ulcer area, including type III *per se*, type IIc +III, type III +IIc, or other combined types of EGC accompanied by type III. NISHI, *et al.*[4)] described two histological different patterns of perforated gastric cancer : one is perforation caused by penetration of cancer lesion itself, and the other is per foration of thin ulcer floor consisting of connective tissues that are free from cancers. Perforation of EGC always belongs to the latter pattern and occurs at the nadir of ulcerated portion. They also reported that, in either case, unlike in the present case, no scarification took place in the surrounding areas, and the whole picture showed findings of acute ulcer.

In Japan, only 28 cases of EGC associated with perforated peritonitis have been described. To our knowledge, there have been no such reports from outside Japan. The first reason for this is raity of this complication in EGC. In our surgical department, over 400 cases of EG C were operated on since 1964, and this is the only case of EGC where perforation developed, comprising less than 0.25% of all EGC cases. Secondly,it is possible that perforation of EGC, even if any, could have been estimated as that of benign peptic ulcer because of their morphological similarity on gross findings. If the microscopic study of the resected specmen was not performed, the final diagnosis of EGC could not be obtained. Thirdly, since the nadir of ulcerated area or the perforated portion is totally free from cancer in EGC and is not composed of cancer cells, it is also possible that the perforation of this type of cancer is not easily recognized as a perforated EGC but as a perforated benign ulcer.

Although a large number of patients with E GC have been operated on nationwide in Japan, there have been only 28 case reports of EGC developing perforated peritonitis in the Japanese literature, as mentioned above. We have reviewed 29 cases including our additional case. The summarized data are listed in Table 1. There were 12 men and three women. Perforation of EGC was far more frequently seen in the male. Sex was not described (ND) in 14 patients. The mean age was 47.7 years, ranging from 25 to 63. The preoperative diagnosis was perforated gastric or duodenal ulcer in 12 patients, perforated EGC IIc +III in one patient, and ND in 15 patients. In only one case the preoperative diagnosis of IIc +III was established prior to the second operation ; the diagnosis of EGC had not been made prior to the first operation (simple suture closure) for perforated stomach disease. The intraoperative di agnosis of the lesion was perforated gastric ulcer in seven patients, perforated EGC type IIc+III in three patients and ND in 15 patients. The lesion was located at the lower third of the stomach in seven patients, at the middle third in nine patients and ND in 11 patients. With regard to macroscopic types of EGC, the most common type was IIc+III, seen in 16 patients, and followed by type III in six, III+IIc in three, III +IIc +IIb in one, and ND in three patients. In terms of the depth of cancer, 12 patients had mucosal carcinoma, whereas eight patients

submucosal, and ND in nine patients. Histologic types of EGC showed tubular adenocarcinoma in 10 cases, signet ring cell carcinoma in 10, poorly differentiated adenocarcinoma in one, and not stated in eight. Fouteen patients underwent distal gastrectomy, of which two patients had had two operations : simple closure at the first operation and gastrectomy at the second operation. No description about operative procedures was given in 15 cases. R_1 lymph node dissection was performed in one patient, R_2 in five, R_0 in one, and not stated in 22 patients. Lymph node metastasis was microscopically noted in one patient out of only four documented cases.

Some diagnostic and surgical problems exist regarding EGC accompanied by perforation. The similarity of type III, type III +IIc and sometimes type IIc +III to a benign peptic ulcer makes establishing the correct diagnosis difficult, although it is of fundamental importance to observe the fresh resected specimen carefully during the operation. The similarity of these excavated types of EGC and a benign gastric ulcer is not only morphological but also biological ; for example, in terms of patient age (predominant in the 40's to 50's), gastric acidity (normal to high),common location of the lesion, and good response to medication. [5] Therefore, it is reasonable to consider that the mechanism of ulceration in EGC is the same as that in a benign peptic ulcer disease.[4] Because of these morphologic similarities, the correct diagnosis of EGC on macroscopic findings is frequently difficult to establish.

The second problem is that of surgery. Both proximal and distal edge of the surgical specimen must be negative for cancer. It seems to be the most important that one should take the possible association of EGC into consideration no matter when perforation of gastric ulcer may take place. It is desirable to perform a microscopic examination of both edges with frozen sections in all cases, but this can not be of clinical practice at every hospital and at every time. Another concern is how extensive lymph node dissection should be performed, if EGC is identified during the emergency operation. Many authors have recommended radical extended operation. [1][2] When the patient's condition is acceptable, should more radical operation for gastric cancer be recommended ? When the first operation had been simple gastrectomy without lymph node dissection and the diagnosis of EGC was established by a microscopic examination after surgery, should reoperation be performed for adequate lymph nodal clearing ? SHIBATA, *et al.* mentioned there was no need of additional dissection and chemotherapy, when the cancer was limited to the mucosa with no microscopic lymph node metastasis. They recommended additional clearance of lymph node only when perigastric nodal involvement was noted. The intraoperative determination on what kind of operation should be done, and how extensive lymph node dissection should be performed still remains controversial and may depend on the patient's systemic and local condition and extent of gastric cancer spread at the first operation. When simple closure is carried out for a perforated gastric lesion, endoscopic follow-up study may be required in consideration of the possibility of the associated gastric cancer.

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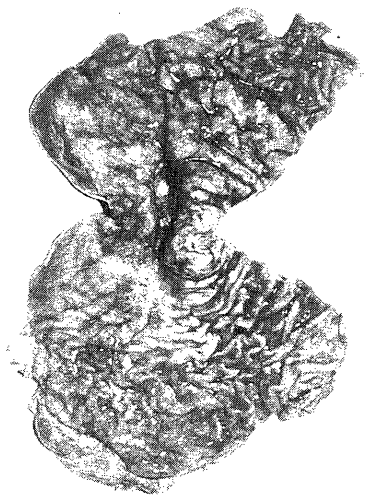

Fig. 1. The resected stomach showing a perforation in the ulcer and marked shortening of the lesser curvature.

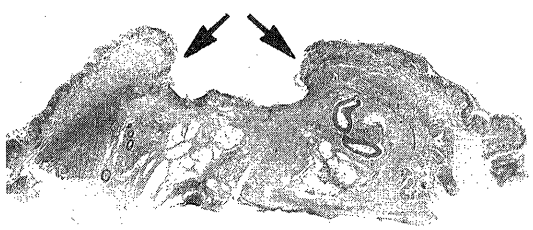

Fig. 2. Cut section of the resected stomach. Arrows indicate carcinoma. (H.E. stain, original magnification ×1)

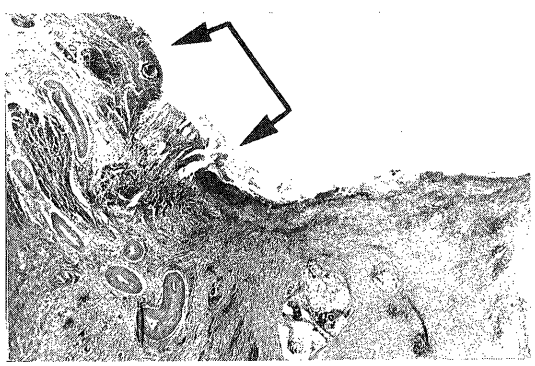

Fig. 3. Magnification of the left arrow in Fig.2, shows signet ring cell carcinoma area confined to the mucosal layer.

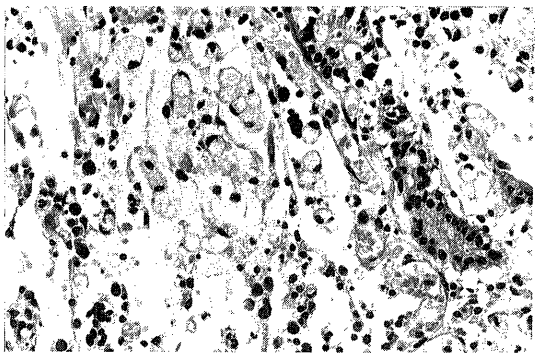

Fig. 4. Signet ring cell carcinoma is revealed.

Table 1. Twenty-nine cases of EGC with perforated peritonitis

		described		not described
Sex	15	male:female=12: 3		14
Age	15	47.7 years (25−63)		14
Preop Dx	13	perforated PU	9	16
		Panperitonitis	4	
Location	16	A ant	3	13
		A post	1	
		A min	3	
		M ant	5	
		M min	4	
Macroscopic types	26	IIc+III	16	3
		III	6	
		III+IIc	3	
		III+IIc+IIb	1	
Depth	20	mucosal	12	9
		submucosal	8	
Histology	21	tub	10	8
		por	1	
		sig	10	
Operative DX	12	gastric ulcer	7	17
		IIc+III	2	
		frozen *	2	
		maligancy	1	
Operation	14	gastrectomy	14	15
		(suture	2)	
Lymph node dissection	7	R0	1	22
		R1	1	
		R2	5	

*:positive peroperative frozen section for malignancy, Abbreviations: Dx: diagnosis, A: lower third of the stomach, M: middle third, ant: anterior wall, post: posterior wall, min: lesser curvature side, PU: peptic ulcer, tub: tubular adenocarcinoma, por: poorly differentiated adenocarcinoma, sig: signet ring cell carcinoma