Early Gastric Cancer Presenting Pyloric or Prepyloric Stenosis


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ABSTRACT: Out of 390 patients with early gastric cancer (EGC) who underwent gastric resection between Jan. 1968 and Jul. 1987, four patients developed pyloric stenosis and one patient developed prepyloric stenosis. Macroscopic types of EGC were IIc in three cases and IIc + III in two cases. Histologic types were tubular adenocarcinoma in four patients, and poorly differentiated adenocarcinoma in one patient. Cancer existed just right on or immediately adjacent to the pyloric ring in all patients; and extended transversely to the gastric axis in four patients, and longitudinally in one patient. An associated open ulcer and/or ulcer scar in the cancer lesion was seen in four patients, and submucosal fibrosis in three patients to a variety of degree, both of which were thought to be greatly attributed to pyloric or prepyloric stenosis. A duodenal ulcer was not present in any patients.

INTRODUCTION

Pyloric stenosis can be caused by numerous benign and malignant conditions, including gastric and duodenal ulcers, gastric cancer, submucosal lesions and extragastric diseases. EGC, however, rarely develops pyloric stenosis.1)2)3)4)5) We describe five patients with EGC who developed this comparatively rare complication. Since little information is available about pyloric stenosis caused by EGC, this study was undertaken to clarify the mechanism of constituents of pyloric or prepyloric stenosis in EGC on the basis of histologic examination.

PATIENTS AND METHODS

Out of 390 patients who underwent gastric resection for EGC between Jan. 1968 and Jul. 1986 at the First Department of Surgery, Nagasaki University Hospital, only five patients had developed pyloric or prepyloric stenosis before operation. The diagnosis of pyloric and prepyloric stenosis was established, based on clinical symptoms, and radiographic and endoscopic findings. There were three men and two women; and the age was 51.2 years on average, ranging from 33 to 74. Four patients underwent distal gastric resection and one patient had total gastrectomy. All surgical specimens were examined macroscopically and microscopically, making the sequential step-sections approximately 5mm in width.
RESULTS

Tables 1 and 2 list patients' characteristics. Symptoms included nausea, vomiting, abdominal fullness in all patients; and weight loss in four patients, which ranged from 2 to 10 kg. Radiologically, the diagnosis of pyloric stenosis was made in four patients and that of prepyloric stenosis in another patient. Iic type EGC was preoperatively diagnosed in three patients, Borrmann 3 in one patient, and benign gastric ulcer in one patient. On the basis of endoscopic findings, the lesion was diagnosed as gastric ulcer in one patient, advanced cancer simulating EGC of type Iic in two patients, and Borrmann 3 in one patient. Only one patient was diagnosed correctly as having EGC of Iic type. Endoscopic biopsy was done in three cases and adenocarcinoma was proved. On the basis of microscopic findings, the lesions of the five patients were diagnosed as Iic in three and Iic + III in two patients.

To clarify the mechanism of formation of pyloric or prepyloric stenosis due to EGC, several measurements of the resected specimen were applied as shown in Fig. 1 and Table 3. The maximal transverse length of cancer to the gastric axis was from 35 to 60 mm. An ulcer in EGC was seen in four patients and maximal transverse length ranged from 0 to 45 mm. Out of these, an open ulcer was seen in four patients, measuring from 5 to 20 mm. Submucosal fibrosis was observed in all patients and transverse length varied from 10 to 65 mm. The extent of cancer spread was mainly of transverse direction to the gastric axis in four patients, and the ratio of the transverse length of gastric cancer to the transverse length of the gastric wall at the site of the maximal transverse

Table 1. Patients' characteristics, clinical manifestations and macroscopic types of the lesions

<table>
<thead>
<tr>
<th>Case No.</th>
<th>Sex</th>
<th>Age</th>
<th>Symptoms</th>
<th>Preoperative Diagnosis</th>
<th>Macroscopic Types</th>
</tr>
</thead>
<tbody>
<tr>
<td>1 730375</td>
<td>M</td>
<td>50</td>
<td>N, V, AF, WL (5 kg)</td>
<td>GU, PS</td>
<td>Iic + III</td>
</tr>
<tr>
<td>2 800260</td>
<td>F</td>
<td>47</td>
<td>N, V, AF, WL (2 kg)</td>
<td>Borrmann 3, PS</td>
<td>Iic</td>
</tr>
<tr>
<td>3 820208</td>
<td>F</td>
<td>33</td>
<td>N, V, AF</td>
<td>Iic, PS</td>
<td>Iic</td>
</tr>
<tr>
<td>4 830527</td>
<td>M</td>
<td>74</td>
<td>N, V, AF, WL (10 kg)</td>
<td>Iic adv., PS</td>
<td>Iic</td>
</tr>
<tr>
<td>5 840208</td>
<td>M</td>
<td>52</td>
<td>N, V, AF, WL (2 kg)</td>
<td>Iic adv., PPS</td>
<td>Iic + III</td>
</tr>
</tbody>
</table>


Table 2. Histological findings of the lesions causing pyloric or prepyloric stenosis

<table>
<thead>
<tr>
<th>Case No.</th>
<th>Location of tumor</th>
<th>Size of tumor (mm)</th>
<th>Depth of tumor</th>
<th>Histology</th>
<th>Distance (mm)**</th>
<th>Ulcer</th>
<th>SMF</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>AD, Circ</td>
<td>50×34</td>
<td>m</td>
<td>tub₁</td>
<td>0</td>
<td>U1–III</td>
<td>++ +</td>
</tr>
<tr>
<td>2</td>
<td>AD, Circ</td>
<td>80×60</td>
<td>sm</td>
<td>tub₂</td>
<td>0</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>3</td>
<td>AD, Circ</td>
<td>42×32</td>
<td>sm</td>
<td>por</td>
<td>0</td>
<td>U1–III</td>
<td>++ +</td>
</tr>
<tr>
<td>4</td>
<td>MAC, Min</td>
<td>32×107</td>
<td>m</td>
<td>tub₁</td>
<td>7</td>
<td>U1–I, II</td>
<td>+</td>
</tr>
<tr>
<td>5</td>
<td>A, Circ</td>
<td>75×20</td>
<td>m</td>
<td>tub₂</td>
<td>5</td>
<td>U1–II, III</td>
<td>++ +</td>
</tr>
</tbody>
</table>

A: maximal transverse length of early gastric cancer
B: transverse length of the pyloric canal at the site of maximal length of cancer along the transverse gastric axis
C: transverse length of ulcer in cancer
D: transverse length of open ulcer in cancer
E: transverse length of submucosal fibrosis
F: distance between pyloric ring and distal edge of cancer
G: average thickness of each section specimen at the pyloric ring

Fig. 1. Measurements of several factors that may attribute to the constituents of pyloric or prepyloric stenosis (dimensions of numbers are shown as millimeter, numbers in parentheses show percentages).

Table 3. Results of measuring of the length of several factors and thickness of the pyloric ring (mm)

<table>
<thead>
<tr>
<th>Case No.</th>
<th>A</th>
<th>B</th>
<th>C</th>
<th>D</th>
<th>E</th>
<th>F</th>
<th>G</th>
<th>B/A</th>
<th>C/A</th>
<th>E/A</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>50</td>
<td>50</td>
<td>10</td>
<td>5</td>
<td>50</td>
<td>0</td>
<td>10.4</td>
<td>(100)</td>
<td>(20)</td>
<td>(100)</td>
</tr>
<tr>
<td>2</td>
<td>60</td>
<td>60</td>
<td>0</td>
<td>15</td>
<td>0</td>
<td>10.0</td>
<td>(100)</td>
<td>(0)</td>
<td>(25)</td>
<td></td>
</tr>
<tr>
<td>3</td>
<td>60</td>
<td>55</td>
<td>15</td>
<td>10</td>
<td>55</td>
<td>0</td>
<td>8.5</td>
<td>(92)</td>
<td>(25)</td>
<td>(92)</td>
</tr>
<tr>
<td>4</td>
<td>45</td>
<td>35</td>
<td>10</td>
<td>10</td>
<td>10</td>
<td>7</td>
<td>8.6</td>
<td>(78)</td>
<td>(22)</td>
<td>(22)</td>
</tr>
<tr>
<td>5</td>
<td>65</td>
<td>55</td>
<td>45</td>
<td>20</td>
<td>65</td>
<td>5</td>
<td>7.7</td>
<td>(85)</td>
<td>(70)</td>
<td>(100)</td>
</tr>
</tbody>
</table>

Numbers in parentheses indicate percentages

length of the cancer ranged from 78% to 100%. The distal edge of the lesion was located at the pyloric ring area per se in three out of five patients, and being 7 mm or less from the pyloric ring at maximum in all patients. The average length between the distal edge of the lesion and the pyloric ring was 2.4 mm. The thickness of the wall at the pyloric ring in five patients was 9.0 mm in average, ranging from 7.7 to 10.4 cm.

Case report (Case 5: prepyloric stenosis due to EGC of type IIc + III): A 52-year-old man developed abdominal fullness, nausea and vomiting. He had lost 2 kg during the last month. On the basis of UGIS (Fig. 2, 3) and endoscopic findings (Fig. 4), prepyloric stenosis due to advanced gastric cancer simulating EGC of type IIc was diagnosed. The distal four-fifths of the stomach was resected with lymph node dissection. The resected specimen showed a linear ulcer along the transverse axis of the stomach (Fig. 5). Fig. 6 shows microscopic findings of the pyloric area. An open ulcer, Ul-III, and submucosal fibrosis are seen at the prepyloric area.

Fig. 2. Barium swallow study in the upright position shows a markedly dilated stomach with prepyloric stenosis and a delayed passage of barium meal. The shortening of the lesser curvature of the stomach is also noted.
Fig. 3. Double contrast study in the supine position. Prepyloric stenosis is apparently seen. Pliability of the stomach proximal to the stricture is well preserved, unlike in advanced gastric cancer.

Fig. 4. Endoscopic picture. Prepyloric stenosis forms pseudopyloric ring. Deformed and narrowed prepyloric area accompanied by irregular mucosal surface with erosions and bleeding is noted. It is difficult to determine the depth of the cancer invasion from these findings.

**COMMENTS**

Pyloric or prepyloric stenosis is mostly seen in benign ulcers and advanced gastric cancer. EGC rarely causes this complication. The incidence of pyloric stenosis in benign ulcer ranges from 1.5 to 8.6%.\(^2\)\(^6\) From the radiologic study, Takada\(^3\) reported that the incidence of pyloric stenosis in EGC of depressed type to be 2.9%. In our present series, pyloric or prepyloric stenosis accounted for 1.3% of the patients with EGC.

Mechanism of constituents of prepyloric or pyloric stenosis due to EGC is complicated, and appears to be attributed to several determinants including the length between the pyloric ring and the distal edge of the cancer lesion, the extent of cancer, especially its spread along the transverse axis of the stomach, submucosal edema and fibrosis, inflammatory cell infiltration, and coexistent ulcer in carcinoma. Among these factors, the short length between the pyloric ring and the distal edge of cancer, and the wide transverse spread of cancer to the longitudinal gastric axis appeared to be attributable to pyloric stenosis to a great extent, because these findings were of consistence in all cases. The former factor ranged from 0 to 7 mm in the present study, and the latter factor (B/A) was 78% or over. Murakami\(^7\) showed that all patients developed pyloric stenosis when the central portion of the lesion existed within 2 cm from the pyloric ring, even if it was a small lesion; but did not when the lesion was located farther than 3 cm from the pyloric ring. Therefore, the short length between the lesion and the pyloric ring is a major factor in developing a pyloric stenosis.

Although it seems that coexistence of ulcer or ulcer scar in cancer and subsequent constriction play an important role in causing the pyloric stenosis, the present study showed a variety of the lengths of ulcer: from 0 to 45 mm, and
Fig. 5. Gross appearance of the antrum of the resected specimen opened along the greater curvature with schematic illustration. The linear ulceration and widespread submucosal fibrosis are noted along the transverse gastric axis at the prepylorus.

Fig. 6. Microscopic findings. Ul-III and submucosal fibrosis are noted at the prepyloric area.

a variety of the lengths of open ulcer: from 0 to 20 mm. ITAND, et al. described six cases of EGC with pyloric stenosis, and concluded that the pyloric stenosis might occur when an ulceration in a depressed type cancer existed, the length between the pyloric ring and the anal edge was less than 1.5 cm or there was duodenal invasion, the ratio of the length of the lesion to the pyloric canal is over 65% and the ratio of the diameter of the ulceration to the lesion was over 50%. In the present study, however, ulceration in cancer lesion was not necessarily present, and it was seen in one of five patients.

TAKAHARA emphasized the importance of submucosal fibrosis which was seen in all patients.
in the present study. But, since its severity varied, it may be difficult to determine how much submucosal fibrosis attributes to a cause of pyloric or pyloric stenosis.

Presumably a certain functional change of the antrum may also contribute to development of pyloric stenosis. 8)

In summary, we described five rare cases of EGC causing pyloric or prepyloric stenosis. The mechanism of development of pyloric stenosis is complicated. Although this rare complication may be attributed to many factors, we conclude that EGC developing pyloric or prepyloric stenosis is mostly of depressed types (IIc or IIc+III) which spread transversely to the gastric axis, and often is associated with an open or healed ulcer and submucosal fibrosis in the lesion.

REFERENCES

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