Gastric remnant necrosis following splenic infarction after distal gastrectomy in a gastric cancer patient

Imamura Hajime*, Enjoji Akihito, Nakashima Hiroharu, Hirabaru Masataka, Moruchi Hiroki, Yamaguchi Junzo

Department of Surgery, National Hospital Organization Saga National Hospital, Saga, Japan

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A B S T R A C T

INTRODUCTION: Distal gastrectomy with lymph node dissection is the standard treatment for gastric cancer. Remnant gastric necrosis after distal gastrectomy is very rare and fatal complication.

PRESENTATION OF CASE: A 78-year-old male diagnosed with advanced gastric cancer underwent distal gastrectomy with lymph node dissection. Postoperative gastric remnant necrosis occurred following splenic infarction. There was thought to be an insufficient blood supply to the gastric remnant due to the lymph node dissection along the proximal splenic artery during the initial surgery. Non-contrast abdominal computed tomography did not reveal any necrosis in the remnant stomach. An endoscopic examination confirmed this diagnosis. Total remnant gastrectomy was performed, and the patient thereafter successfully recovered.

DISCUSSION: Careful management of blood vessels and lymph node dissection above the pancreas should be performed to avoid restricting the blood flow and also to prevent gastric remnant necrosis.

CONCLUSION: The knowledge of this fatal complication is crucial for management of postoperative complication. For early and accurate diagnosis, upper gastrointestinal endoscopy is necessary in case of remnant gastric necrosis.

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1. Introduction

Distal gastrectomy with lymph node dissection is the standard treatment for gastric cancer. The incidence of mortality is quite low in Japan. The stomach can resist ischemic necrosis, and therefore gastrectomy can be safely performed, and gastric remnant necrosis is a rare postoperative complication. This report presents a case of gastric remnant necrosis followed by spleen infarction after distal gastrectomy with lymph node dissection.

2. Case report

A 78-year-old male with hypertension, hyperlipidemia and chronic kidney disease presented with anorexia and dysphagia. He had been treated stenting of the left common iliac artery for atherosclerosis obliterans 2 years earlier. He had no history of smoking or diabetes. Endoscopic examination of the stomach revealed a type 3 tumor circumferentially located in the antrum of the stomach. A histological examination of biopsied specimens showed poorly differentiated adenocarcinoma. The results of blood tests, including the tumor marker levels (CEA and CA19-9) were within the normal limits except for creatinine, which was 1.36 mg/dl. Computed tomography (CT) of the abdomen revealed wall thickening of the antrum and calcification of the splenic artery due to arteriosclerosis (Fig. 1). There was no evidence of metastasis in the liver, other organs and lymph nodes. Distal gastrectomy and lymph node dissection (D2) and resection of the posterior layer of the transverse mesocolon was performed due to invasion through the posterior layer of the transverse mesocolon. A Billroth I reconstruction was performed and a J-VAC drain® was placed under the anastomosis. A histological examination revealed poorly differentiated adenocarcinoma that had invaded the serosa of the gastric wall with lymph node metastasis. The TNM stage of the tumor was T4aN2M0.

High fever and left upper quadrant pain occurred on postoperative day 3. Laboratory data demonstrated leukocytosis (WBC count: 27,900/μl) and a high C-reactive protein level (26.76 mg/dl). Computed tomography of the abdomen revealed fluid collection anterior to the spleen, around the tail of pancreas. In addition, Doppler ultrasound could not demonstrate the presence of a blood flow to the spleen. He was diagnosed to have spleen infarction. The fluid collection was found to be reactive abdominal fluid. We therefore continued to observe the patient under total parenteral nutrition. The high fever and inflammation (WBC count: 18,700/μl and high C-reactive protein level: 16.39 mg/dl) continued, but the left upper quadrant pain gradually subsided. An upper gastrointestinal series revealed good passage through the anastomosis and no leakage on postoperative day 8 (Fig. 2). Oral intake was permitted.

* Corresponding author at: Department of Surgery, Nagasaki University, Graduate School of Biomedical Sciences, 1-7-1, Sakamoto, Nagasaki, 852-8501, Japan.
Tel.: +81 95 819 7316; fax: +81 95 819 7319.
E-mail address: hajimeimamura0630@gmail.com (I. Hajime).

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The high fever and inflammation gradually decreased and his appetite recovered to a normal level (Fig. 3). However, high fever occurred again on postoperative day 22 (C-reactive protein level: 12.03 mg/dl). The structure of the gastric remnant wall was confirmed on non-contrast abdominal CT (Fig. 4). Furthermore, the upper gastrointestinal series showed the leakage of contrast medium from the greater curvature to the abdominal cavity. Gastrointestinal endoscopy was performed to observe the mucosa of gastric remnant and confirm whether the cause of the perforation was ischemia. Endoscopy revealed shedding and necrotic change of the gastric remnant mucosa, with circumferential involvement from the oral side to the site of anastomosis (Fig. 5).

Emergency laparotomy was performed on the same day under a diagnosis of necrosis of the remnant stomach. The laparotomy revealed necrosis of the entire gastric remnant from the esophageal-gastric junction to the anastomosis. The remnant stomach adhered to the inferior side of the lateral liver. A total gastrectomy with Roux-en-Y reconstruction and abscess drainage was performed (Fig. 6). The histopathology revealed total mucosal necrosis in the gastric remnant. He recovered and was discharged with no problems 46 days after the reoperation.

3. Discussion

The stomach can resist ischemic necrosis, because of its rich vascular supply and extensive submucosal plexus. There are a few reports of gastric necrosis from various etiologies in the literature. These include bulimia, abdominal trauma, and infectious diseases. Vascular causes include therapy transcatheter embolization, infusion of vasopressin, splenectomy, and splenic vein thrombosis accompanying splenic infarction. Remnant gastric necrosis after distal gastrectomy is very rare. There are few case reports of remnant gastric necrosis after gastrectomy.

An interruption of the blood flow to the gastric remnant may have caused necrosis of the gastric remnant. The etiology of such an interruption of the blood flow can be caused by arteriosclerosis, atrial fibrillation, sepsis, or venous thrombosis. Smoking,
diabetes, hypertension, and hyperlipidemia are also risk factors of these conditions. In one case, the authors mentioned that the anatomical variation of the gastric arterial supply was a risk factor. The deficient common hepatic artery (Adachi type VI\textsuperscript{15}) and the short gastric artery noted by abdominal enhanced CT, may be related to an imbalance of the blood supply after gastrectomy.\textsuperscript{12}

In this regard, it is important to confirm the presence of any anatomical variation before the operation in order to prevent the development of necrosis. The patient had a history of arteriosclerosis obliterans, hyperlipidemia, and calcification of the splenic artery due to arteriosclerosis was recognized on preoperative abdominal CT (Fig. 1). In this case, calcification of the artery may have been risk factors for the observed reduction of the blood flow. In addition, lymph node dissection above the pancreas may have induced the reduction of the blood flow. Lymph node dissection above the pancreas was performed around the splenic artery, so the possibility of stenosis, overextension or spasm of the artery was also considered during the surgical management.

Table 1 shows the clinical findings of gastric remnant necrosis reported in Japan. In many cases, the necrosis occurred about 2–3 weeks after surgery. Only four of 10 previous cases were diagnosed before surgery. The possibility of gradually developed necrosis was suggested, however, there was no direct evidence. The current patient developed necrosis 22 days following surgery.

The infarction is often diagnosed with contrast-enhanced CT.\textsuperscript{13} However, the diagnosis of necrosis of the gastric remnant is difficult if the blood flow is maintained partially. The blood flow to the gastric remnant is partially maintained in some cases.\textsuperscript{14} Early diagnosis requires observation of mucosa of the gastric remnant by gastrointestinal endoscopy,\textsuperscript{11,12} which reveals necrotic change of the gastric remnant mucosa.

In general, anastomotic leakage would be considered in the differential diagnosis of cases like ours, so we should be examined at first by the upper gastrointestinal series. However, early diagnosis the gastric remnant necrosis could not be confirmed in the current case, because patient did not show peritonitis and splenic infarction. The mortality rate of remnant gastric necrosis is 40–70%\textsuperscript{12,14} so it is important to carefully observe the postoperative progress for prevention and early diagnosis of this complication. In this case, the leakage of contrast medium was confirmed from the greater curvature, so we suspected unusual types of leakage. This is the reason why we cannot avoid endoscopic examination. Endoscopic examinations should therefore be performed to make an early diagnosis of gastric remnant necrosis. As to gastric remnant necrosis, total gastrectomy and drainage was required treatment.

Careful management of blood vessels and lymph node dissection above the pancreas should be performed to avoid restricting the blood flow and also to prevent gastric remnant necrosis.

**Table 1**

The clinical findings of gastric remnant necrosis in Japan.

<table>
<thead>
<tr>
<th>Case no.</th>
<th>Year</th>
<th>Age</th>
<th>Sex</th>
<th>Reconstruction</th>
<th>Post-op (days)</th>
<th>Pre-operative diagnosis</th>
<th>Treatment</th>
<th>Results</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>1991</td>
<td>n.d.</td>
<td>n.d.</td>
<td>n.d.</td>
<td>26</td>
<td>Necrosis</td>
<td>Conservative</td>
<td>Died</td>
</tr>
<tr>
<td>2</td>
<td>1995</td>
<td>61</td>
<td>F</td>
<td>B-I</td>
<td>7</td>
<td>Peritonitis</td>
<td>Total gastrectomy</td>
<td>Survived</td>
</tr>
<tr>
<td>3</td>
<td>1996</td>
<td>49</td>
<td>M</td>
<td>B-I</td>
<td>14</td>
<td>Peritonitis necrosis</td>
<td>Total gastrectomy</td>
<td>Survived</td>
</tr>
<tr>
<td>4</td>
<td>1996</td>
<td>60</td>
<td>M</td>
<td>B-I</td>
<td>8</td>
<td>Peritonitis</td>
<td>Total gastrectomy</td>
<td>Survived</td>
</tr>
<tr>
<td>6</td>
<td>1999</td>
<td>53</td>
<td>M</td>
<td>B-I</td>
<td>20</td>
<td>Peritonitis</td>
<td>Total gastrectomy</td>
<td>Died</td>
</tr>
<tr>
<td>7</td>
<td>2004</td>
<td>72</td>
<td>M</td>
<td>R-Y</td>
<td>15</td>
<td>Peritonitis</td>
<td>Total gastrectomy</td>
<td>Survived</td>
</tr>
<tr>
<td>8</td>
<td>2006</td>
<td>68</td>
<td>M</td>
<td>B-I</td>
<td>7</td>
<td>Peritonitis</td>
<td>Total gastrectomy</td>
<td>Died</td>
</tr>
<tr>
<td>9</td>
<td>2008</td>
<td>72</td>
<td>M</td>
<td>B-I</td>
<td>14</td>
<td>Peritonitis</td>
<td>Total gastrectomy</td>
<td>Died</td>
</tr>
<tr>
<td>10</td>
<td>2009</td>
<td>50</td>
<td>M</td>
<td>B-I</td>
<td>26</td>
<td>Necrosis</td>
<td>Total gastrectomy</td>
<td>Survived</td>
</tr>
<tr>
<td>11</td>
<td>2011</td>
<td>64</td>
<td>M</td>
<td>R-Y</td>
<td>21</td>
<td>Necrosis</td>
<td>Total gastrectomy</td>
<td>Survived</td>
</tr>
<tr>
<td>Our case</td>
<td>2011</td>
<td>78</td>
<td>M</td>
<td>B-I</td>
<td>22</td>
<td>Necrosis</td>
<td>Total gastrectomy</td>
<td>Survived</td>
</tr>
</tbody>
</table>

n.d.: not described; B-I: Billroth I; R-Y: Roux-en Y; and post-op: post-operative days.
4. Conclusion

Upper gastrointestinal endoscopy is necessary to make an accurate diagnosis of remnant gastric necrosis. It is important to observe the blood flow in the remnant gastric mucosa in patients that develop splenic infarction and this should be confirmed by upper gastrointestinal endoscopy.

Conflict of interest statement

None of the authors have any conflict of interest to declare.

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Ethical approval statement

Written informed consent was obtained from the patient for publication of this case report and accompanying images.

Author contributions

All authors contributed to this study.

References