Duodenocolic Fistula Caused by a Peptic Stomal Ulcer Following Distal Gastrectomy

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Abstract

We herein describe the case of a 51-year-old man with a duodenocolic fistula (DCF) caused by a stomal ulcer. The patient complained of watery diarrhea, dysgeusia and malnutrition. His medical history included distal gastrectomy with Billroth I reconstruction for duodenal ulcer perforation. A combination study using endoscopy and contrast imaging confirmed the presence of DCF. Laparotomic fistulectomy was performed, which resulted in the patient’s recovery from diarrhea and malnutrition. The histological findings suggested that the fistula had originated from a stomal ulcer. In patients with chronic watery diarrhea of obscure origin following gastrectomy, DCF is a possible cause of the diarrhea.

Key words: diarrhea, duodenocolic fistula (DCF), peptic stomal ulcer, endoscopy, contrast imaging

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Introduction

Duodenocolic fistula (DCF) is a relatively rare complication characterized by a communication between the duodenum and colon. In the past, the most common cause of DCF was peptic ulcers (1). Currently, most cases of DCF arise from malignant tumors or inflammatory bowel diseases, since peptic ulcers can now be treated with highly effective methods, including H2 receptor antagonists (H2RA), proton pump inhibitors (PPI) and the eradication of underlying Helicobacter pylori (H. pylori) infection (2).

We herein present a case of DCF arising from a peptic stomal ulcer following distal gastrectomy with Billroth I (B-I) reconstruction. Several reports have shown gastrojejuno-colic fistula to be a complication of partial gastrectomy and gastrojejunostomy with Billroth II (B-II) reconstruction (3, 4). To our knowledge, this is the first report of DCF caused by a stomal ulcer developing 30 years after distal gastrectomy with B-I reconstruction. We believe that this case is highly educational due to the recent rarity of this condition in Japan.

Case Report

A 51-year-old Japanese man with a medical history of a pneumothorax at 18 years of age and distal gastrectomy due to duodenal ulcer perforation 30 years earlier visited our hospital in March 2012. He complained of diarrhea, dysgeusia and general fatigue lasting for seven months. H2RA and gastric mucosal protective agents were administered. The patient’s medical compliance was extremely poor, and he had both a smoking (10 cigarettes/day) and drinking (500 mL beer/day) habit. He had no family history of gastrointestinal disease. A physical examination revealed pedal edema. Laboratory tests disclosed hypoproteinemia, hypoalbuminemia, mild anemia and trace element deficiencies (Table 1).

First, endoscopic examinations of the upper and lower gastrointestinal tract were performed. Upper gastrointestinal endoscopy demonstrated poor findings due to the presence...
of significant residual fecal-like matter (Supplementary Fig. 1A); however, two passages were observed over the anastomosis (Supplementary Fig. 1B-D). At that time, no records of the patient’s previous operation were available. We then suspected that distal gastrectomy with B-II reconstruction had been performed. Colonoscopy yielded inconclusive data due to the large amount of residual fecal matter and showed no significant findings (Supplementary Fig. 1E). Next, a barium meal series revealed rapid transit of barium into the colon. We subsequently diagnosed the presence of a fistula (Fig. 1*).

We again attempted to perform upper gastrointestinal endoscopy, this time with gastrografin. The patient was instructed to have nothing per os after dinner on the day prior to the examination. Two liters of polyethylene glycol lavage (Niflec®; Ajinomoto Pharmaceuticals Co., Ltd., Tokyo, Japan) and 10 mL of dimethicone (Gascone drop 5%®; Kissei Pharmaceuticals Co., Ltd., Tokyo, Japan) were administered orally for bowel preparation on the day of the endoscopic study. We confirmed the presence of a fistulous opening just over the stomal ulcer near the anastomosis (Fig. 2A). The series performed with gastrografin demonstrated an abnormal passage from the duodenum to the transverse colon (Fig. 2B). We diagnosed the patient with DCF developing after distal gastrectomy with B-I reconstruction, not B-II reconstruction. Subsequently, we received the previous surgical records, which indicated that distal gastrectomy had been performed with B-I reconstruction. The patient exhibited markedly high levels of anti-*H. pylori* IgG antibodies (38 U/}

Table 1. Laboratory Findings on Visiting Our Hospital

<table>
<thead>
<tr>
<th>Peripheral blood</th>
<th>normal range</th>
<th>Blood chemistry</th>
<th>normal range</th>
</tr>
</thead>
<tbody>
<tr>
<td>WBC 6,300 /µL</td>
<td>(4,000-9,000)</td>
<td>TC 115.9 mg/dL</td>
<td>(128-219)</td>
</tr>
<tr>
<td>RBC 362×10⁶ /µL</td>
<td>(410-550×10⁶)</td>
<td>TG 51 mg/dL</td>
<td>(30-150)</td>
</tr>
<tr>
<td>Hb 11.1 g/dL</td>
<td>(13.6-16.8)</td>
<td>HDL-C 47 mg/dL</td>
<td>(40-96)</td>
</tr>
<tr>
<td>Ht 33.4 %</td>
<td>(40.0-48.0)</td>
<td>LDL-C 58.7 mg/dL</td>
<td>(60.1-119.9)</td>
</tr>
<tr>
<td>MCV 92.3 fl</td>
<td>(78.0-110.0)</td>
<td>TSH 1.77 µU/mL</td>
<td>(0.45-4.22)</td>
</tr>
<tr>
<td>MCH 30.7 pg</td>
<td>(28.0-35.0)</td>
<td>FT3 2.5 pg/mL</td>
<td>(2.5-3.6)</td>
</tr>
<tr>
<td>MCHC 33.2 %</td>
<td>(31.0-36.0)</td>
<td>FT4 1.0 ng/mL</td>
<td>(0.6-1.3)</td>
</tr>
<tr>
<td>PLT 23.7×10⁶ /µL</td>
<td>(11.7-32.9×10⁶)</td>
<td>CRP 0.05 mg/dL</td>
<td>(0.20)</td>
</tr>
</tbody>
</table>

Blood chemistry:

- TP 5.5 g/dL (6.7-8.3) Na 139 mEq/L (138-146)
- Alb 2.9 g/dL (4.0-5.0) K 3.5 mEq/L (3.6-4.9)
- T-Bil 0.3 mg/dL (0.2-1.2) Cl 97 mEq/L (99-106)
- ALT 39 IU/L (3-42) Ca 8.1 mg/dL (8.7-10.3)
- LDH 212 IU/L (119-229) IP 3.1 mg/dL (2.5-4.7)
- ALP 277 IU/L (115-359) Cu 73 µg/dL (78-131)
- TP 10 IU/L (11-47) Mg 1.9 mEq/L (1.5-2.8)
- Lipase 45 IU/L (37-125) PT 122.2 % (70-130)
- Fe 59 µg/dL (63-192) APTT 33.4 sec (25-35)
- BUN 15.6 mg/dL (8-22)
- CRE 0.79 mg/dL (0.6-1.1)
- UA 5.9 mg/dL (3.6-7.0)

Coagulating system:

- Coagulating system
- PT 122.2 % (70-130) APTT 33.4 sec (25-35)

Figure 1. Image of the barium meal study. Upper gastrointestinal barium radiography showed early passage of contrast medium into the colon. The presence of a fistula is shown (*). C: colon, D: duodenum, GR: gastric remnant.

Figure 2. Images of the second upper gastrointestinal endoscopy performed with gastrografin. A. Upper gastrointestinal endoscopy showed an ulceration just over the anastomosis. The arrow indicates the direction to the duodenum. B. The existence of a fistula communicating with the duodenum and transverse colon was confirmed. C: colon, D: duodenum, F: fistula, GR: gastric remnant, U1: ulceration.
the stomal ulcer into the colon under a chronic H. pylori-positive condition.

The patient was admitted to our hospital, and elective surgical correction was performed. The intraoperative findings indicated the presence of severe adhesions between the transverse colon, including the fistula. The stomach with the proximal duodenal bulb and a part of the transverse colon, including the fistula. The stomach was anastomosed in a functional end-to-end manner. As shown in Fig. 3B, the fistulous opening was located just over the anastomosis. A pathological examination found no evidence of inflammatory bowel disease or malignant transformation within the fistula. The fistulou communication was considered to have been caused by a peptic stomal ulcer (Fig. 3C, D). The patient’s postoperative course was uneventful, with the duration of hospitalization being only 19 days. Laboratory data obtained three months after the operation showed improvements in TP (7.1 g/dL), Alb (4.5 g/dL), Hb (13.2 g/dL), Zn (77 μg/dL) and Cu (93 μg/dL). The patient’s general status has greatly improved, and he is currently doing well.

Discussion

After the first presentation by Sanderson in 1863 (5), 125 cases of benign DCF were reviewed by Benn et al. in 1997 (1). We searched the medical literature using PubMed with the key words “duodenocolic fistula” from 1997 through 2012 and identified a total of 20 cases (2, 6-24). The etiology was benign in 10 cases (Crohn’s disease (CD), 4 (6-9); foreign body ingestion, 1 (10); ulcerative colitis, 1 (11); duodenal ulcer, 1 (12); tuberculosis, 1 (13); abscess cavity, 1 (14); stent migration, 1 (15)) and malignant in 10 cases (colon cancer, 8 (2, 16-22); lymph node metastasis from colon cancer, 2 (23, 24)). The frequencies of the causal factors in the 136 cases of benign DCF, including our case, are listed in Table 2.

Previously, the most common cause of DCF was peptic ulcers; however, only one case caused by peptic ulcers has
Table 2. Causes and Frequencies of Benign Duodenocolic Fistula (DCF) in Published Cases

<table>
<thead>
<tr>
<th>Causes</th>
<th>1997</th>
<th>1997-2012</th>
</tr>
</thead>
<tbody>
<tr>
<td>Crohn's disease</td>
<td>29</td>
<td>4 (6-9)</td>
</tr>
<tr>
<td>Duodenal ulcer</td>
<td>30</td>
<td>1 (12)</td>
</tr>
<tr>
<td>Tuberculosis</td>
<td>15</td>
<td>1 (13)</td>
</tr>
<tr>
<td>Duodenal diverticulum</td>
<td>15</td>
<td>0</td>
</tr>
<tr>
<td>Iatrogenic (including stent migration)</td>
<td>5</td>
<td>1 (15)</td>
</tr>
<tr>
<td>Ulcerative colitis</td>
<td>4</td>
<td>1 (11)</td>
</tr>
<tr>
<td>Foreign body penetration</td>
<td>3</td>
<td>1 (10)</td>
</tr>
<tr>
<td>Diverticulosis coli</td>
<td>4</td>
<td>0</td>
</tr>
<tr>
<td>Appendicitis</td>
<td>3</td>
<td>0</td>
</tr>
<tr>
<td>Stomal ulcer</td>
<td>0</td>
<td>1 (this case)</td>
</tr>
<tr>
<td>Amebiasis</td>
<td>1</td>
<td>0</td>
</tr>
<tr>
<td>Abscess cavity</td>
<td>0</td>
<td>1 (14)</td>
</tr>
<tr>
<td>Pseudotumor (hydatid cyst)</td>
<td>1</td>
<td>0</td>
</tr>
<tr>
<td>Idiopathic/Spontaneous</td>
<td>15</td>
<td>0</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td>125</td>
<td>11 (references)</td>
</tr>
</tbody>
</table>

been reported in the past 16 years. Due to the development of effective therapy for peptic ulcers using antacid drugs (H2RA and PPI) and H. pylori eradication regimens, the incidence of such fistulas has decreased dramatically. CD is currently the most common cause of DCF (24%, 33/136). Several reports have described the most frequent causal factors to be colonic malignancy and inflammatory bowel disease, especially CD (2, 25-27). Malignant causes include carcinoma of the colon, gallbladder and duodenum and metastatic esophageal carcinoma (23, 28-32).

As far as we were able to determine based on a search of published reports, our case is the only case of peptic stomal ulcers developing after gastrectomy as a cause of DCF. Thirty cases of duodenal ulcers reported by Benn et al. (1, Table 2) may contain one case of a stomal ulcer developing after gastrectomy; however, the references were not indicated, and we were unable to obtain the records.

The typical clinical symptoms of DCF are diarrhea, weight loss, abdominal pain, nausea and fecal vomiting (33, 34). These symptoms are extremely similar to those of gastrojejunocolic fistulas; diarrhea and weight loss are reported to be present in 80-82% of such patients (35). In addition, our patient had dysgeusia. Dysgeusia occurring in patients with DCF most likely results from malabsorption of trace elements, especially Zn, due to the reduction of intestinal transit time. In fact, our patient exhibited a low level of Zn (59 μg/dL), and his symptoms of dysgeusia improved together in association with an increase in the Zn level after the therapeutic operation.

The presence of DCF is usually diagnosed on radiological studies. Barium enemas are generally believed to be the most reliable, with a sensitivity of 85-95% (1, 33). Furthermore, a high yield in diagnosis (53-87%) is achieved with barium meal studies (33). Barium meals detect fistulas less frequently than enemas (36). On the other hand, the diagnostic sensitivity of gastrointestinal endoscopy is only 33-60%. As for colonoscopy, a diagnosis is made in only 0-25% of cases (33). In our case, the initial gastrointestinal endoscopy and colonoscopy did not reveal the presence of DCF. A barium meal study suggested the existence of a fistula, and a second upper gastrointestinal endoscopy with gastrografin imaging confirmed the diagnosis of DCF. Gastrografin is a water-soluble contrast medium that is safer to use than barium in cases with a fear of leakage in the digestive tract. We propose that performing proper preparation of residual matter and combination studies with endoscopy and contrast medium is useful for making the diagnosis of DCF.

In the 1980s, the excision of fistulas, direct closure and serosal patching for the surgical treatment of DCF were reported (37, 38). Recently, due to the development of total parenteral and enteral nutritional support and subsequent improvements in these therapies, one-stage en bloc resection has become the preferred and accepted method for the treatment of DCF (39). Although performing surgery is the only effective way to eliminate the devastating sequelae of such fistulas, a few reports have demonstrated the efficacy of octreotide (23) and cover stent placement (16), especially in cases of inoperable malignancy.

The eradication of H. pylori following endoscopic surgery for early gastric carcinoma is recommended (40). Several reports have described the mucosa of the gastric remnant showing significant decreases in inflammation and improvements in glandular atrophy following H. pylori eradication (41, 42). These improvements are believed to reduce carcinogenesis in the gastric remnant and prevent the recurrence of peptic stomal ulcers (43), although there are only very limited studies concerning the eradication of H. pylori in the gastric remnant following surgery. In our patient, the stomach partially remained following the surgery; therefore, we are planning to perform eradication therapy.

In patients with chronic watery diarrhea of obscure origin following gastrectomy, DCF is a possible cause of the diarrhea. Performing proper bowel preparation and combination studies with endoscopy and contrast imaging is essential for making an accurate diagnosis of DCF.

The authors state that they have no Conflict of Interest (COI).

References

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