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【 CASE REPORT 】

A Rare Case of Gastric Ulcer Penetrating the Pancreas that was Successfully Managed by Conservative Therapy

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Abstract:
A 62-year-old woman was admitted to our hospital with septic shock due to left submandibular osteomyelitis and cellulitis. Her condition improved following tooth extraction, drainage, and the administration of antibiotics. However, on the 4th day of hospitalization, she went into hemorrhagic shock after defecating a massive tarry stool. Emergency esophagogastroduodenoscopy (EGD) was performed. We found a giant ulcer at the antral greater curvature of the stomach. Computed tomography (CT) revealed that the gastric ulcer had penetrated the pancreas. She had no signs of peritonitis and had a bad general condition. She was therefore managed solely by conservative therapy. She recovered within days.

Key words: peptic ulcer, gastric ulcer, pancreas, penetration, conservative therapy

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Introduction

Peptic ulcer disease (PUD) usually occurs in the stomach and proximal duodenum. The serious complications of PUD include hemorrhage, perforation, and gastric outlet obstruction (1). However, cases involving a gastric ulcer penetrating the pancreas are very rare, with only two reported cases, both of which were treated surgically. We herein report a rare case of a patient with a gastric ulcer penetrating the pancreas that was successfully treated by conservative therapy.

Case Report

In January, 2015, a 62-year-old woman was admitted to our hospital with consciousness disturbance. Based on the findings of whole body computed tomography (CT), we diagnosed her with septic shock due to left submandibular osteomyelitis and cellulitis. She also exhibited disseminated intravascular coagulation. A blood test revealed the following: hemoglobin (Hb), 11.1 g/dL; white blood cell (WBC) count, 13,000/mm^3; platelet (PLT) count, 3.3×10^4/mm^3; C-reactive protein (CRP), 39.9 mg/dL; prothrombin time (PT)-INR, 1.37; and fibrin degradation product (FDP), 319.3 μg/mL. She regularly underwent hemodialysis for chronic kidney failure and was taking clopidogrel. She had no medical history of peptic ulcer and had not been taking nonsteroidal anti-inflammatory drugs (NSAIDs), proton pump inhibitors, or histamine receptor antagonists. Her condition improved after tooth extraction, the drainage of the affected region, and the administration of antibiotics. However, she suddenly went into hemorrhagic shock after defecating a massive tarry stool on the 4th day of hospitalization. The patient was lucid, and her blood pressure, pulse rate, and temperature were 75/38 mmHg, 80 bpm, and 37.4℃, respectively. No abdominal pain or tenderness was observed. A blood test revealed the following findings: Hb, 6.0 g/dL; WBC count, 6,700/mm^3; PLT count, 3.5×10^4/mm^3; CRP, 9.0 mg/dL; PT-INR, 1.11; and FDP, 32.3 μg/mL. Emergency esophagogastroduodenoscopy (EGD), which was performed to identify the source of the hemorrhage, revealed a giant ulcer with oozing hemorrhage at the antral greater curvature of the stomach (Fig. 1, 2a). While looking for the bleeding point, we found a hole at the bottom of the ulcer, which suggested perforation or penetration (Fig. 2b). Emergency CT did not

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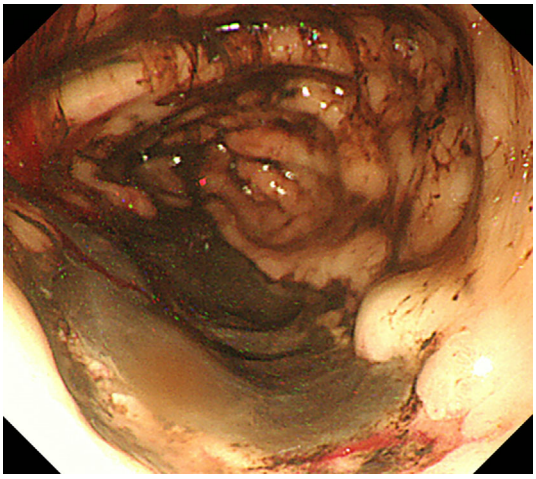

Figure 1. A giant ulcer was found at the antral greater curvature of the stomach.

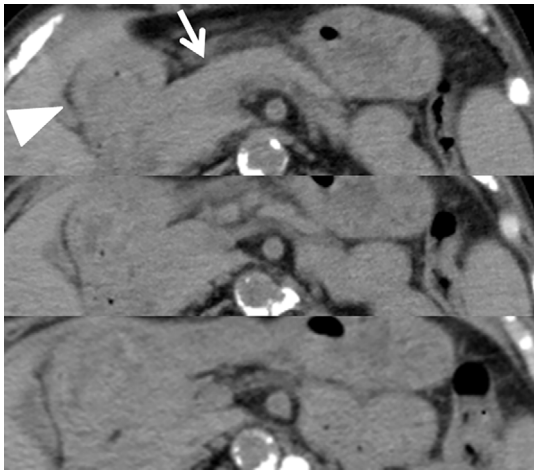

Figure 3. Computed tomography suggested that there was no free air, and showed that the fat density between the antral greater curvature of the stomach (arrowhead) and pancreas (arrow) had disappeared.

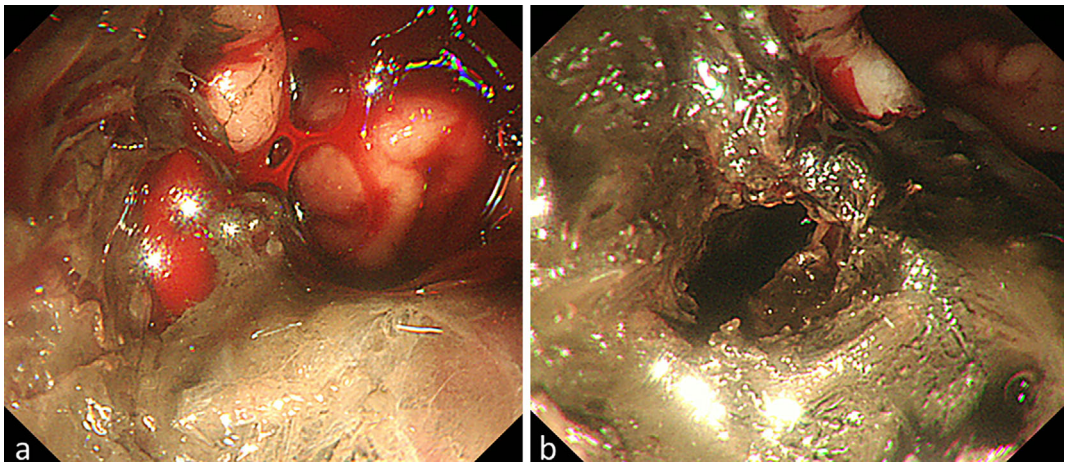

Figure 2. Emergency esophagogastroduodenoscopy revealed oozing hemorrhage from the ulcer (a) and a hole at the bottom of the ulcer (b), suggesting perforation or penetration.

indicate the presence of free air, and the fat density between the antral greater curvature of the stomach and the pancreas had disappeared (Fig. 3). We therefore determined that the gastric ulcer had penetrated the pancreas, and not perforated the peritoneal cavity. We treated her conservatively because she did not show any signs of peritonitis and her general condition was bad. Her treatment included omeprazole (20 mg twice a day, intravenously) and erythrocyte transfusion (four units, daily). The episodes of tarry stool gradually decreased, and EGD confirmed complete hemostasis 3 days after the initial intervention (7th day of hospitalization). No rebleeding was observed thereafter, and the ulcer gradually diminished (Fig. 4). The endoscopic findings showed no atrophy (the criteria of Kimura and Takemoto C-0) and no intestinal metaplasia in the stomach. A histological examination revealed no evidence of malignancy or *Helicobacter pylori*. Finally, scar healing was confirmed by EGD.

Discussion

The serious complications of PUD are hemorrhage, perforation, and gastric outlet obstruction (1). Using a Japanese administrative database, perforation occurred in 9% of PUD cases (2). Perforation usually affects the anterior wall of the duodenum (60%), the antral curvature (20%), or the lesser curvature (20%) of the stomach (1). In-hospital death of perforated gastric ulcer was reported at 7% in Japan (3).

Penetration is pathologically similar to perforation, except that the ulcer does not erode into the peritoneal cavity, but into another organ such as the liver or pancreas (4). Fujihara et al. reported that antral and duodenal ulcers can penetrate the pancreas (5). Indeed, in our patient, the ulcer was located at the antral greater curvature of the stomach, and had penetrated the pancreas.

Free peritoneal perforation resulting in chemical and bacterial peritonitis represents a surgical emergency (1). At the

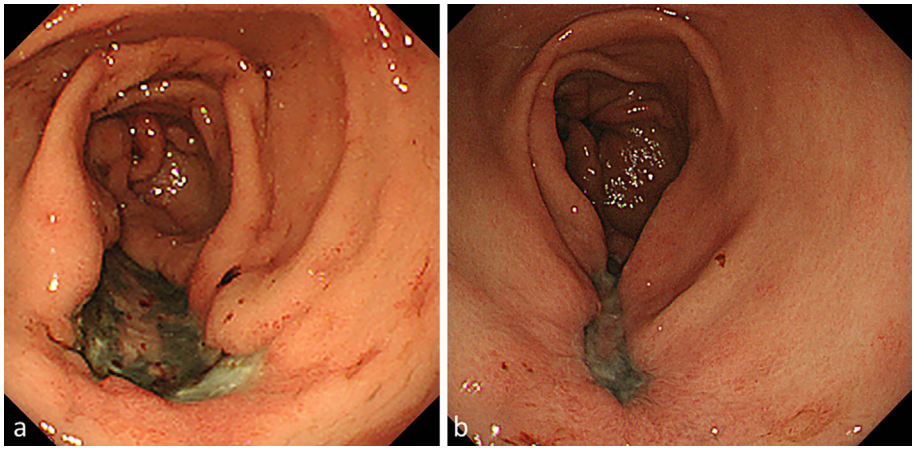

Figure 4. Esophagogastroduodenoscopy images from 16th (a) and 31th (b) day of hospitalization suggested the gradual diminution of the gastric ulcer following conservative therapy without surgery.

time we performed emergency EGD on our patient, we doubted that perforation had occurred, as there was no free air on CT and the patient had neither abdominal pain nor tenderness. Therefore, we did not diagnose the patient as having peritonitis. Furthermore, because of her bad general condition, she was unlikely to have tolerated a surgical procedure at the time, which is why we opted to treat her conservatively. Although daily erythrocyte transfusion was necessary because of the ongoing hemorrhage related to penetration, the patient recovered without operation in a matter of days.

The prevalence of *Helicobacter pylori*-negative and NSAID-negative PUD (idiopathic PUD) has been reported 0.9-2.6% in Japan based on data from the 1990s. Recently, there has been a marked decline in *Helicobacter pylori* infection rates among general population in Japan. Then, the prevalence of idiopathic PUD was 12% in (2012-2013) Japan (6). Our patient had not been taking NSAIDs, showed no atrophy (according to the criteria of Kimura and Takemoto C-0) and no intestinal metaplasia of the stomach. A histological examination revealed no evidence of *Helicobacter pylori*; thus, she was thought to be negative for *Helicobacter pylori*. The reason for the gastric ulcer formation was unclear, but it might have been influenced by the use of clopidogrel.

Gastric ulcers rarely penetrate the pancreas. In August 2016, a PubMed search using the keywords "peptic ulcer", "gastric ulcer", and "penetration" retrieved only two reports of gastric ulcer penetrating the pancreas; both cases were treated surgically (5, 7). One case (5) involved localized peritonitis although there was no free air; thus, surgical treatment was necessary. In the other case (7), a physical examination revealed little more than tenderness of the epigastrium; thus, the patient was first treated conservatively. However, surgical treatment was needed because the ulcer remained unhealed after one month of conservative therapy. In the case of our patient, there was no free air, there were no signs of peritonitis, and her general condition was bad. We therefore treated her conservatively. Conservative therapy may be possible in cases in which a gastric ulcer penetrates the pancreas that do not involve peritonitis.

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

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