The Development of Gastric Outlet Obstruction due to a Lumen-occupying Protruding Duodenal Ulcer Mimicking a Submucosal Tumor

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Abstract:
Even in the era of Helicobacter pylori eradication and proton pump inhibitors, peptic ulcer remains an important disease. Stricture due to a duodenal ulcer in the healing stage is a well-known etiology of benign gastric outlet obstruction. However, a duodenal ulcer-induced submucosal tumor-like change with gastric outlet obstruction is a very rare manifestation. We herein present a rare case of a patient with deteriorating symptoms of gastric outlet obstruction caused by an unusual manifestation of a lumen-occupying protruding duodenal ulcer mimicking a submucosal tumor.

Key words: gastric outlet obstruction, peptic ulcer, duodenal ulcer, submucosal tumor

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Introduction
Gastric outlet obstruction (GOO) has both benign and malignant etiologies. Since the emergence of eradication therapy for Helicobacter pylori and proton pump inhibitors for the treatment of peptic ulcer, malignant disease has become the leading etiology of GOO (1). Nevertheless, GOO caused by numerous benign etiologies remains important, and peptic ulcer is the leading cause of benign GOO (2). The diagnostic criteria for duodenal ulcer-related GOO are gastric retention and severe stenosis of the duodenal bulb on esophagogastroduodenoscopy (EGD), because an endoscope cannot be advanced into the duodenal bulb due to severe stricture (3). Although peptic ulcers typically appear as mucosal defects with various degrees of excavation and/or mucosal scarring, there are few reports of gastric ulcers with features that mimic a submucosal tumor (SMT) (4-6). We herein report a case of GOO that developed due to a lumen-occupying protruding duodenal ulcer mimicking a submucosal tumor.

Case Report
A 70-year-old man was referred to our department with a one-year history of a deteriorating abdominal fullness after meal ingestion with no significant medical history of peptic ulcer or malignancy. The patient had no history of using non-steroidal anti-inflammatory agents or other ulcer inducing drugs. The patient smoked approximately 40 cigarettes per day. The laboratory findings on admission were as follows: white blood cell count, 5.7×10^3/μL; hemoglobin 13.1 g/dL; C-reactive protein, 0.1 mg/dL; albumin, 4.22 g/dL; serum carcinoembryonic antigen, 1.7 ng/mL (upper limit of normal, ULN: 5.0 ng/mL), and serum carbohydrate antigen 19-9, 2.0 U/mL (ULN: 37.0 U/mL). The serum Helicobacter pylori (H. pylori) antibody titer was 3.0 U/mL (ULN: 10.0 U/mL). Contrast enhanced computed tomography demonstrated an extended stomach with food residue and an enhanced lesion that protruded into the duodenal bulb (Fig. 1). EGD revealed an expansive space-occupying lesion in the duodenal bulb (Fig. 2). The lesion included a reddish mu-
cosa with indentation. Double-contrast fluoroscopy under EGD using a J-turn position in the duodenal bulb revealed a filling defect that was acting like a ball valve. A forceps biopsy from the duodenal mucosa showed no evidence of malignancy and a non-specific pathological diagnosis. Positron emission tomography (PET) demonstrated an intensive abnormal uptake [maximum standardized uptake value (SUV) 6.1] at the duodenal bulb (Fig. 3), which was suggestive of malignancy. Next, we conducted an endoscopic ultrasonography-guided fine-needle biopsy (EUS-FNB) us-

**Figure 1.** Contrast enhanced computed tomography (CE-CT). An image of a CE-CT demonstrating an enhanced lesion protruding into the lumen of a duodenal bulb. The stomach is extended with food residue.

**Figure 2.** Endoscopic views of the duodenal lesion. Esophagogastroduodenoscopy (EGD) demonstrating a space-occupying lesion at the duodenal bulb. A pyloric ring was expanded and obstructed with this lesion. EGD in the duodenal J-turn position demonstrating an opposite hemisphere of the lesion mimicking a submucosal tumor. The lesion was capped with a reddish normal mucosa with a central depression.
The patient was scheduled to undergo surgery with curative intent for the following reasons: 1) the GOO status was deteriorating, 2) the maximum SUV in the PET was 6.1, and 3) there possibility of a false-negative cytopathological EUS-FNB result was a concern. Laparotomy revealed severe adhesion between the gallbladder and the hepatoduodenal ligament. An intraoperative frozen section analysis revealed no malignant tissue. Thus, in addition to a distal gastrectomy, cholecystectomy, distal bile duct resection, and Roux-en-Y reconstruction with hepaticojejunostomy was performed. One of the resected specimens showed a duodenal ulcer associated with marked tissue granulation and severe chronic active inflammatory infiltration in the submucosal and muscular layers. There was no evidence of malignancy in the other resected organs (Fig. 5). The patient’s postoperative course was uneventful and there was no evidence of ulcer recurrence.

Discussion

We described a rare case of deteriorating GOO caused by a benign duodenal ulcer mimicking a submucosal tumor. Peptic ulcers are the leading cause of benign GOO (2). Severe stenosis of the duodenal bulb revealed by EGD is one of the diagnostic criteria for duodenal ulcer-related GOO (3). In the present case, the deteriorating GOO was caused by a lumen-occupying protruding duodenal ulcer that was behaving like a ball valve. We proceeded with a differential diagnosis according to the characteristics observed in the EUS image (7). The duodenal mass demonstrated echogenicity and was contiguous to the duodenal submucosal layer (third layer). The muscular layer (fourth layer) at the base of the mass was thickened; thus, the primary differential diagnosis was ectopic pancreas (7, 8). The first EGD biopsy specimens did not provide a specific pathological diagnosis. We therefore performed EUS-FNB to obtain submucosal tissue for a cytopathological diagnosis. In a previous article, EUS-guided tissue acquisition provided a diagnostic accuracy of 92.7% for endoscopic biopsy-negative gastrointestinal tumors (9). However, the sensitivity was 87.3% and...
submucosal protrusion of a gastric ulcer (4-6), the submucosal protrusion of a duodenal ulcer easily occupies the duodenal lumen resulting in GOO (as described above). In rare cases, the manifestation results in intussusception (12).

With the exception of heavy cigarette consumption, the present patient had no risk factors for duodenal ulcer. In recent articles, a smoking habit, the intake of aspirin, and H. pylori infection were identified as independent risk factors for duodenal ulcer formation (odds Ratio: 2.84, 4.28, and 3.56, respectively) (13). Furthermore, cigarette smoking aggravates ulcer formation and delays ulcer healing in the gastrointestinal mucosa (14). We deduced that severe protracted inflammation in the submucosal layer due to smoking might eventually lead to the development of submucosal protrusion that could mimic SMT in the duodenal bulb. We also considered that severe adhesion around the duodenal bulb might develop as a result of micro-perforation of the ulcer. Severe inflammation could explain the intensive abnormal uptake at the duodenal bulb on PET.

In conclusion, severe stricture caused by a duodenal ulcer is a well-known cause of GOO. However, we should keep in mind that duodenal ulcers rarely have unusual manifestations of GOO caused by a lumen-occupying protrusion that mimics a submucosal tumor.

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

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