Case Report

Perforated Peptic Ulcer of the Jejunum with Ectopic Gastric Mucosa

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Ectopic gastric mucosa (EGM) of the jejunum is rare. We report an 81-year-old man with perforated peptic ulcer of the jejunum originating from EGM. Emergency segmental resection of the jejunum was performed with satisfactory immediate results. Diagnosis after surgery was established by histopathology. In addition, sections were examined immunohistochemically using antibodies against MUC5AC, MUC6 and MUC2. Hematoxylin-eosin-stained sections showed glandular differentiated tissue interposed between normal small bowel epithelium. EGM was positive for MUC5AC and MUC6, and negative for MUC2. Histological examination of the resected specimen confirmed that the perforation was caused by a peptic jejunal ulcer associated with EGM. To our knowledge, this is the first reported case of perforated peptic ulcer of the jejunum with EGM in an elderly patient.

ACTA MEDICA NAGASAKIENSIA 54: 45 - 48, 2009

Keywords: Jejunum; Peptic ulcer; Ectopic gastric mucosa

Introduction

Nontraumatic perforation of the small bowel is rare. Ectopic gastric mucosa (EGM) is commonly present in Meckel's diverticulum.1 Approximately 30 cases of EGM located in the small bowel beyond the ligament of Treitz and not associated with Meckel's diverticulum, have been reported.2 The acquired variety of EGM is common in the ileum or jejunum where mucosal regeneration occurs due to inflammatory lesions such as regional enteritis.3 The usual clinical picture is that of acute intestinal obstruction, perforation of intestinal ulcer, or intestinal bleeding and anemia.4 We report a case of perforated peptic ulcer caused by EGM in the jejunum. Search of the June 1952-March 2009 PubMed database for the keyword "ectopic gastric mucosa" showed no such case has been published so far in the English literature.

Case report

An 81-year-old man was admitted to the casualty with a clinical picture of acute abdomen. He gave a history of gastric ulcer-like symptoms. On admission, arterial blood pressure was 120/80 mmHg, heart rate 100 bpm, and body temperature 37.1 °C. Results of laboratory tests were all within the normal ranges except for leukocyte count, which was 12 × 10⁹/ml. Physical examination was suggestive of peritonitis. A CT scan of the abdomen taken subsequently showed swelling of the mesenteric lymph nodes and thickening of the jejunal wall with free intra-abdominal fluid and air on the mesenteric border of the mid jejunum (Figure 1). At laparotomy, a moderate amount of purulent ascites was seen in the abdominal cavity. A perforation was found in the jejunum approximately 100 cm distal from the ligament of Treitz, and a 60cm-long jejunal segment was resected. En bloc resection of the affected jejunum was performed because the preoperative CT scan was suggestive of jejunal perforation by malignancy (especially Hodgkin's lymphoma), although subsequent pathological examination showed EGM with typical signs of a perforated peptic ulcer, with no malignancy. Macroscopic examination showed a perforated jejunal ulcer and two unperforated ulcers (Figure 2). No cancerous lesions were found during the operation, but the resected jejunal segment showed a few areas of inflammatory thickening of the intestinal wall. The resected specimens (intestine and mesenteric lymph nodes) contained no malignancies. The mesenteric lymphadenopathy was caused by chronic inflammation associated with the peptic ulcers.

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Received July 21, 2009; Accepted August 17, 2009
The small bowel epithelium with EGM contained fundic glands with both parietal and chief cells as well as pyloric glands (Figure 3). The gastrointestinal epithelium could be classified into the following four phenotypes based on the staining patterns for MUC2, MUC5AC, and MUC6 (Table 1). Immunohistochemical staining for MUC5AC, MUC6, and MUC2 yielded different results for the normal small bowel epithelium and EGM; the normal small bowel epithelium was positive for MUC2 and negative for MUC5AC and MUC6 (Figure 4A) while EGM was positive for MUC5AC and MUC6 and negative for MUC2 (Figure 4B).

The patient had an uneventful postoperative course with no further complications. Before discharge, upper and lower GI endoscopies showed normalization of the remaining gastrointestinal tract. At that stage, a Tc99m-pertechnetate scintiscan was negative (figure not shown). At the last follow-up examination, the patient remains free of disease 32 months after discharge.
Table 1. Classification of the gastrointestinal epithelium according to the staining patterns for MUC2, MUC5AC, and MUC6

<table>
<thead>
<tr>
<th>MUCSAC and MUC6: negative</th>
<th>Intestinal pattern (Goblet cells)</th>
<th>Unclassified pattern</th>
</tr>
</thead>
<tbody>
<tr>
<td>MUC5AC and/or MUC6: positive</td>
<td>Gastrointestinal pattern</td>
<td>Pyloric gland (MUC6-positive)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Foveolar gland (MUC5AC-positive)</td>
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</tbody>
</table>

Discussion

EGM has been found in different locations, such as the tongue, esophagus, larynx, lungs, gallbladder, pancreas, urinary bladder, small bowel, colon and rectum. The esophagus, duodenum, and Meckel's diverticulum are the most common sites for the congenital variety, but it is rarely found in the ileum or jejunum. EGM of the intestinal tract is an occasional, incidental, gross or microscopic finding at surgery or autopsy. Nontraumatic perforation of the small bowel is rare. In our case, emergency surgery was performed because of a perforated peptic ulcer in the jejunum. The patient had persistent gastric ulcer-like symptoms before admission. EGM can also be found in gastric cysts, enteric duplications, duplication cysts, in an otherwise normal small bowel, or in Barrett's esophagus. Symptoms and complications are presumably related to heterotopic hydrochloric acid secretion by the EGM, which may induce chronic inflammation and ulceration.

Despite modern imaging techniques, diagnosis is difficult and often made intraoperatively in Meckel's diverticulum. CT is most accurate in the differential diagnosis; however, incorrect diagnosis may be due to unawareness of the possible existence of the EGM, which is quite rare.

The gastric epithelium is usually positive for MUC5AC (gastric foveolar epithelium) and MUC6 (gastric pyloric gland) but negative for MUC2. On the other hand, goblet cells of the intestinal epithelium are generally positive for MUC2 but negative for MUC5AC and MUC6. In our case, immunohistochemical examination revealed that EGM was positive for MUC5AC and MUC6, but negative for MUC2. These findings indicate that the majority of the cells of EGM identified on H&E staining of the resected specimen were mature for mucin expression as well as for normal gastric mucosa. Therefore, peptic ulceration is probably due to the EGM. Also, with increasing age, degradation of the alkaline exocrine of the pancreas may cause ulceration without neutralizing the acids. The ulcerations were found in the jejunum approximately 100 cm distal from the ligament of Treitz. EGM is considered a congenital condition; however, our patient was an 81-year-old man with a perforated peptic ulcer of the jejunum associated with EGM. Interestingly, we may thus conjecture that EGM is acquired in our case.

The 99mTc-pertechnetate, a radionuclide, loosely binds plasma protein and accumulates in functional gastric mucosa. The utility of 99mTc-pertechnetate scintigraphy in the diagnosis of EGM is well established, particularly, in the case of Meckel's diverticulum. Therefore, to identify the presence of the remnant EGM, the patient underwent 99mTc-pertechnetate scintigraphy postoperatively. In our case, no positive findings could be obtained by a 99mTc-pertechnetate scintiscan after surgery. Therefore, the patient was followed-up without further treatment.

In conclusion, we reported a rare case of perforated peptic ulcer arising from EGM in the jejunum. Surgeons must be aware of ectopic gastric mucosa, especially in patients with nontraumatic perforation of the small bowel. Careful clinical and histopathological examinations as well as immunohistochemical staining are recommended for the diagnosis of peptic ulcer of the small bowel.

References


