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Bile Ductular Proliferation in the Gastric Wall after Gastric Ulcer Penetration into the Liver

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Liver penetration is one of the most serious complications of peptic ulcer diseases but is rarely encountered. A 57-year-old man was admitted to our hospital for further evaluation of a gastric malignancy. One month before admission, he was diagnosed with an adenocarcinoma by endoscopic biopsy at other hospital. A subtotal gastrectomy was performed to confirm malignancy and relieve the epigastric pain. Histologically, single glands were located between the ulcer base and attached liver tissue and stained positively for cytokeratin 19. The pathologic diagnosis was a gastric ulcer and bile ductular proliferation in the liver and gastric wall. Here, this report is the first Korean case of liver penetration of a gastric ulcer.

Key Words: Stomach ulcer; Penetration; Liver

Peptic ulcer diseases (PUDs) are common and can be complicated by inflammation, ulceration, or perforation.[1,2] Organ penetration can occur into the pancreas, gastrohepatic omentum, biliary tract, or liver. But, penetration of the liver is rare and may lead to severe complications, such as upper gastrointestinal hemorrhage and abscess formation.

Here, we report the case of a 57-year-old man with a bile ductular proliferation in the gastric wall after a perforated gastric ulcer penetrated the liver.

CASE REPORT

A 57-year-old man was admitted to our hospital for further evaluation of a gastric malignancy. One month before admission, he was diagnosed with an adenocarcinoma by endoscopic biopsy at another hospital. The patient presented with intermittent epigastric pain and had been receiving medication for a gastric ulcer at a private clinic for 4 years. He was a smoker with a 15 pack-year smoking history and over the previous 2-3 years had sonumed more than half a bottle of Soju 15 times/mo. A physical examination revealed no specific findings. In particular, the abdomen was soft, nontender, and nondistended. The following laboratory parameters were of note: hemoglobin, 9.4 g/dL; hepatitis B surface (HBs) antigen(-), anti-HBs(+), and anti-hepatitis C virus(-). Tumor marker and liver function tests were within normal limits. Abdominal computed tomography revealed wall thickening of the gastric mid-body and no lymph node enlargement in adjacent areas (Fig. 1). The initial endoscopic examination revealed a large ulcer in the posterior wall of the gastric body. The histopathological findings were ulcer debris, typical giant cells and histiocytes and basophilic material. A second endoscopic biopsy was performed and the subsequent histopathological findings matched those of the first biopsy. He underwent a radical subtotal gastrectomy with a Billroth type II anastomosis to rule out malignancy and relieve the epigastric pain.

The gastrectomy specimen showed a large ulcer, measuring 3×2 cm, in the lesser curvature of the gastric body, and liver tissue was attached to the serosal surface of the ulcer. A histological examination showed that single glands were located between the ulcer base and the attached liver tissue (Fig. 2). The attached liver tissues showed ductular proliferation (Fig. 3A), and single glands were scattered in the gastric wall (Fig. 3B). Cells of single glands exhibited oval-shaped nuclei with occasional nucleoli.

No mitotic figures were observed. Immunohistochemically, cytokeratin (CK)7 and CK19 (Fig. 3C) were positive in single glands, but CK20 was negative. The Ki-67 proliferation rate was very low. A paraffin block of a remnant specimen was prepared to search for tumor cells.

DISCUSSION

Perforation occurs in 5-10% of patients with PUD, and 60% of gastric ulcer perforations occur along the lesser curvature. Unusual presentations of a penetrating gastric ulcer include pneumopericardium,[3] subcutaneous emphysema,[4] splenic abscess, tension pneumothorax, gastropleural fistula, gastrobronchial fistula, gastroenteral fistula,[5] and penetration of the heart and liver.

The hepatic-related complications of a perforated gastric ulcer are liver abscess, upper gastrointestinal hemorrhage, subcap-

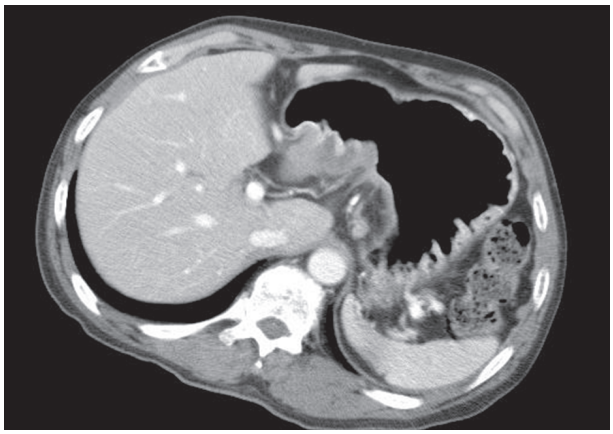

Fig. 1. Computed tomography scan shows focal gastric wall thickening with enhancement at the posterior wall of the mid-body of the stomach.

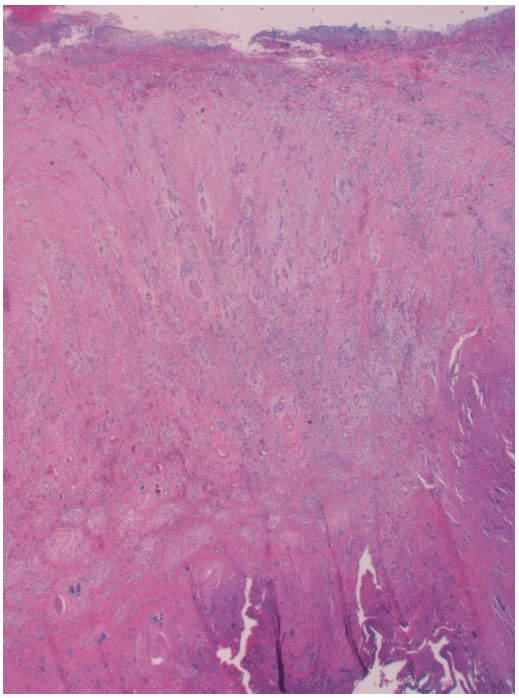

Fig. 2. Single glands are located between the ulcer base and the attached liver tissue.

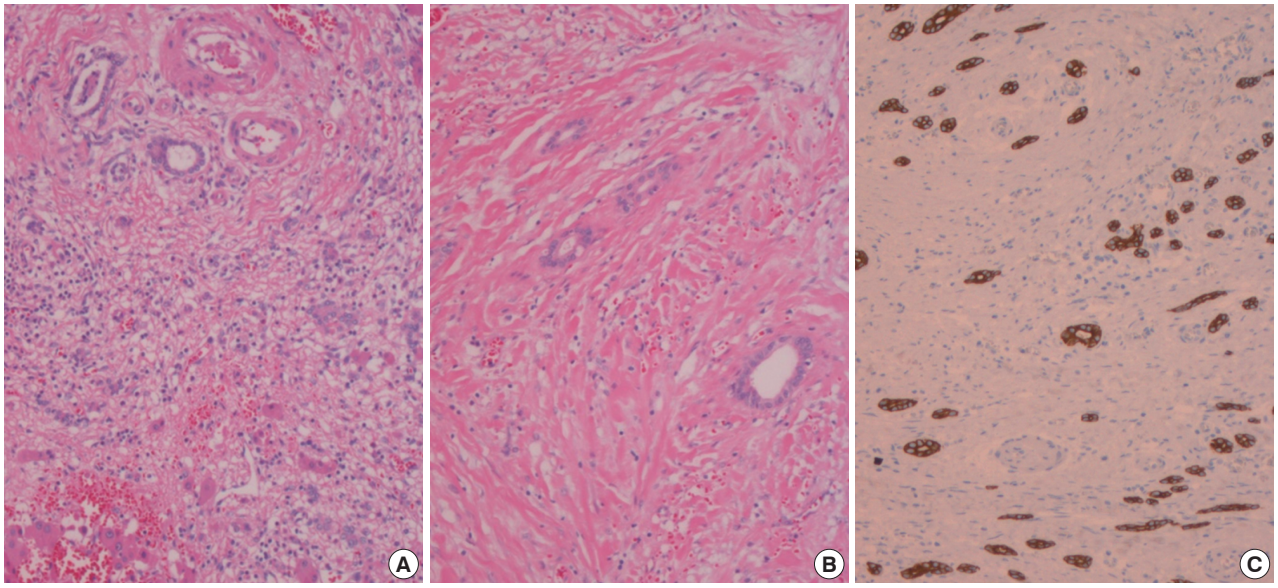

Fig. 3. Histologically, the attached liver tissue shows ductular proliferation (A), and single glands are distributed in the gastric wall (B). Immunohistochemically, single glands in the gastric wall stain positively for cytokeratin 19 (C).

sular liver abscess,[6] and liver rupture. Kayacetin and Kayacetin[7] reported one case and reviewed 13 others of liver penetration by a peptic ulcer. A diagnosis was made in all cases by the presence of liver tissue on histological examinations of endoscopic biopsies, and males predominated (78.6%). Ulcers were located in the lesser curvature of the stomach (42.9%), in the anterior (21.4%) and posterior (7.1%) walls of the antrum, and in the anterior (14.3%) and posterior (14.3%) walls of duodenal bulbs. In no case was the ulcer recognized clinically or radiologically prior to endoscopy.

In present case, no liver was observed in two endoscopic biopsies. The gastrectomy specimen revealed single glands located between the ulcer base and attached liver tissue, and no atypism was observed in the cells of single glands. Immunohistochemically, single glands in the gastric wall and ductular proliferation in the attached liver tissue stained positively for CK19, but the Ki-67 proliferation rate was very low; thus, we concluded that the single glands in the gastric wall originated from the bile duct.

Slides of the remnant specimen were prepared, but no cancer cells were found, so the slide with the diagnosis of an adenocarcinoma from the other hospital was reviewed. The slide showed clustered gastric glands and single cells in the lamina propria with ulcer debris, which suggested reparative change. Accordingly, if a gastric malignancy is suspected, a histopathological examination of the endoscopic biopsies is essential, and distinguishing malignancy from reparative or reactive changes should be undertaken meticulously.

In all cases mentioned above, a diagnosis was established by the histological presence of liver tissue in endoscopic biopsies. However, our case showed no hepatic tissue, but rather ductular proliferation of both the gastric wall and the attached liver tissue. These findings suggest protracted contact with gastric acid. However, the attached liver tissue was not severely inflamed and did not show inflammatory atypia, and a liver function test was within normal limits, suggesting that local hepatic injury does not perturb liver functions and that the diagnostic value of liver function tests in cases of liver penetration by an ulcer are limited.

In summary, liver penetration by a gastric ulcer is uncommon. In such cases, a histopathological examination of the endoscopic biopsy material is essential for diagnosis and for excluding a primary malignancy. Histopathological findings showed the presence of hepatic tissue or ductular proliferation in the specimen.

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