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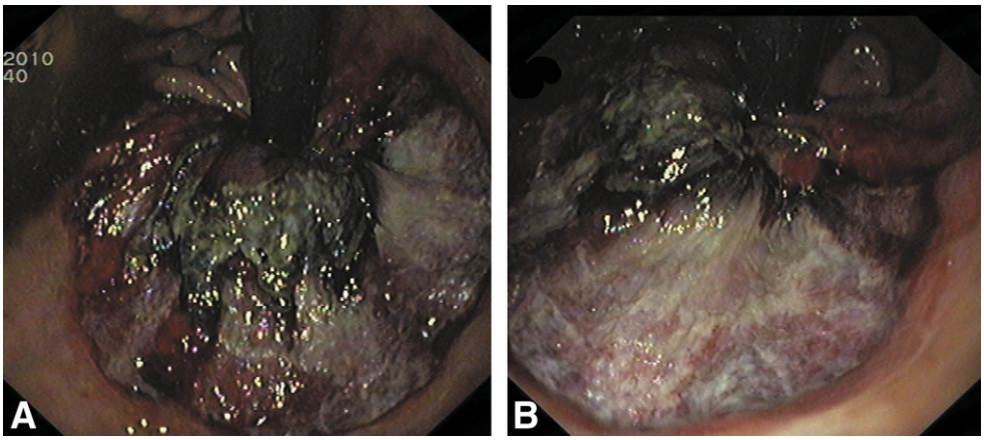
Gastric necrosis caused by gastric banding



A 48-year-old woman, with a history of type 2 diabetes, stage 5 chronic kidney disease, and gastric banding that had been performed 8 months previously because of morbid obesity, was admitted with progressive vomiting over a period of 3 weeks, after stoma calibration. Upper endoscopy revealed ulceration and extensive gastric necrosis of the gastric body near the band impression (**A, B**). Biopsy specimens were taken, and histology showed hemorrhagic gastric necrosis. Laparoscopic removal of the gastric band was performed with anterior gastric wall seromuscular suturing. During the surgical procedure, gastric necrosis was confirmed at the stenotic depression corresponding to the lap band.

DISCLOSURE

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Commentary

Ischemic gastric necrosis is a rare occurrence in large measure because of the stomach's abundant blood supply, which includes 5 major arteries (right and left gastric, right and left gastroepiploic, and the short gastrics) as well as numerous minor and collateral vessels. Studies in cadavers have shown that complete gastric wall vascular filling is possible with only 1 patent major artery, and in animal models, it is only after ligation of all the major arteries and most of the collateral circulation that gastric necrosis can be produced. In healthy volunteers with normal splanchnic vasculature, however, transient gastric ischemia may develop during maximal exercise as judged by intragastric P_{CO_2} tonometry. Nonetheless, despite this great wealth of arterial circulation and the stomach's resistance to ischemia, gastric necrosis has been seen with occlusive splanchnic arterial disease, excessive distention of the stomach either with air or after ingestion of massive amounts of food and liquid, with intrathoracic herniation, volvulus, and in the Prader-Willi syndrome. Ischemia that accompanies gastric dilation is thought to be due to venous insufficiency, which occurs at an intraluminal pressure of more than 14 mm; as little as 3 L of fluid can distend a normal stomach to this point of tension, and with 4 L of fluid, intragastric pressure of 120 to 150 mm Hg can occur and result in gastric rupture. Chronicity of gastric dilation is an important additional factor, and intragastric volumes up to 15 liters have been reported in eating disorders such as bulimia. Gastric ischemia also can result from ingestion of certain medications such as nonsteroidal anti-inflammatory drugs, from therapeutic embolization, and, as this case teaches us, from laparoscopic gastric banding, although why there was an 8-month interval between banding and the presentation of gastric

ischemia eludes me. There are no pathognomonic symptoms of gastric ischemic necrosis, and without rapid diagnosis, what begins as mild epigastric tenderness, vomiting, or diarrhea rapidly eventuates in acute peritonitis, septic shock, and death. In modern facilities, diagnosis is usually made on CT scanning (gastric pneumatosis, pneumoperitoneum, and portal venous gas), at endoscopy, or in the surgical theater. An interesting cycle has unfolded here and one that is not unique to this case. We try to solve problems and in doing so create other problems that need solving. Fortunately for this woman, her story seems to have a happy ending . . . .

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Hepatobiliary cystadenoma of the liver prolapsing into the extrahepatic bile duct (with video)

A 28-year-old woman was admitted to our hospital with upper abdominal pain. Palpebral conjunctivae showed slight icterus, and abdominal examination revealed a soft but slightly tender abdomen in the epigastric region without hepatomegaly. Laboratory data indicated obstructive jaundice without tumor marker elevation. US, CT (**A**), and magnetic resonance imaging (**B**) showed bilateral dilatation of the intrahepatic bile ducts and a cyst measuring 7.3 cm × 7.0 cm, with thin, internal, septal structures in segment IV of the liver. ERCP revealed a huge filling defect in the common bile duct (CBD) (**C**). EUS and intraductal US (**D**) revealed a multilocular cystic lesion in the CBD. Peroral videocholangioscopy showed a smooth surface of the cystic lesion (**E**) (Video 1, available online at www.giejournal.org). The patient underwent resection of the left medial section of the liver, with en bloc excision of the extrahepatic bile duct and gallbladder. A large, cystic mass with well-defined tumor margins was observed in the liver. The sausage-shaped mass prolapsed into the extrahepatic bile duct (**F**), and the cut surface of the resected specimen revealed septal formation and cystic spaces (**G**). The smooth-walled lining continued into the cystic mass in segment IV of the liver via segment IV of the bile duct. Histologic examination revealed multiple loculations lined by a columnar biliary epithelium with ovarian-like stroma (**H** [H&E, orig. mag. X4], **I** [H&E, orig. mag. X100]). The tumor was histologically diagnosed as hepatobiliary cystadenoma of the liver, with a part of the cyst prolapsing into the CBD.

DISCLOSURE

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Commentary

Hepatobiliary cystadenomas (HBCs) are extremely rare hepatic neoplasms. Only about 200 cases have been reported since the first HBC resection was described in the late 19th century. Although its cause remains largely speculative (perhaps from obstruction of a congenitally aberrant biliary tree), its hosts are almost certainly women, most of whom remain asymptomatic for decades. The tumor is generally intrahepatic and right-sided, can grow to a massive size (up to 30 cm), and is prone to the usual complications: bleeding, infection, rupture, and rarely malignant degeneration. You might be wondering why the authors reported the (normal) serum tumor markers; HBCs may be the one cystic lesion for which markers (CA 19-9 [carbohydrate antigens] and CEA [carcinoembryonic antigen]) have diagnostic value (but as you can see, the often dramatic radiographic images are not subtle). Complete resection is the treatment of choice and should be pursued in the fit operative patient because the potential for malignant degeneration cannot be reliably ascertained. HBCs are analogous to mucinous cystic lesions of the pancreas, and the ovarian-like stromal lining explains not only the sex specificity but suggests origins in Müllerian remnants that got lost somewhere in the right upper quadrant during embryogenesis. This case is striking in its rarity, its affliction of a young patient, and its prolapsing presentation that gave access to a diverse array of high-tech imagery.