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You Just Got Burned! What Is Wrong With This Gastric Pouch?

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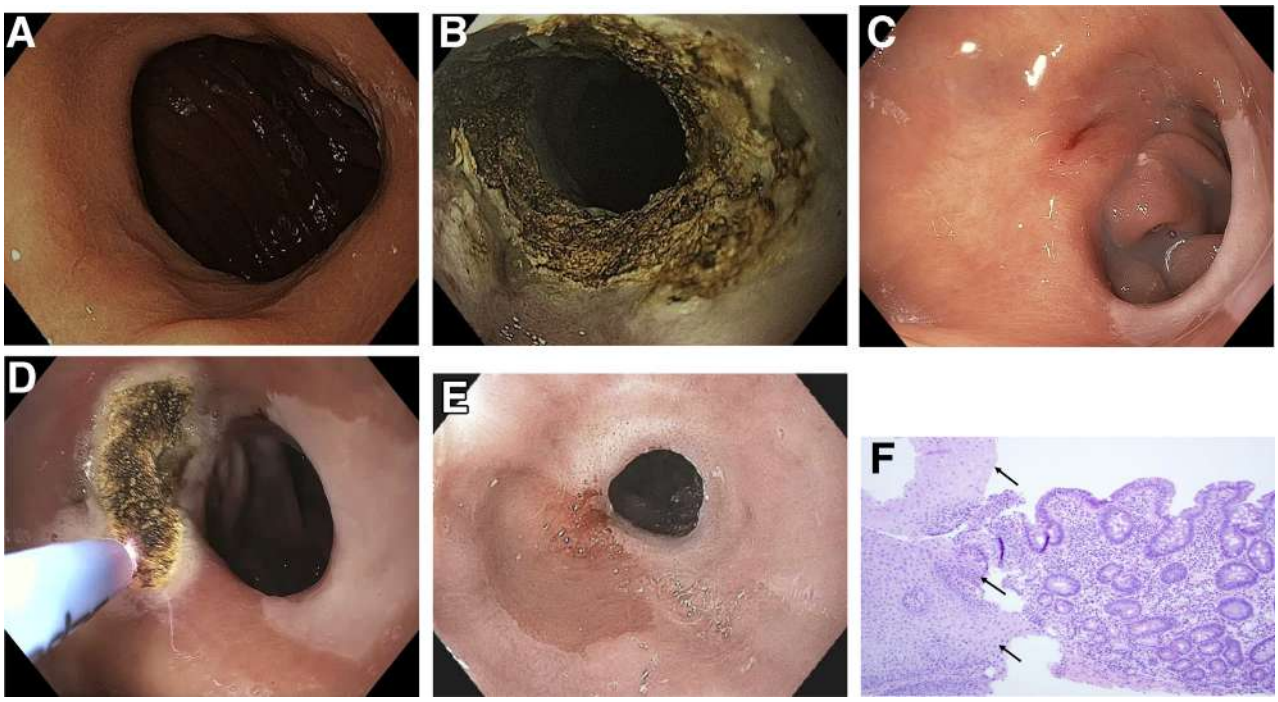

Question: We describe 3 unique presentations of patients with a prior history of Roux-en-Y gastric bypass who were referred for endoscopic treatment of weight regain. All of the patients had failed prior attempts at lifestyle modifications and pharmacologic weight loss treatment. On physical examination, their body mass indices ranged from 27 to 30 kg/m^2, but examination and complete laboratory evaluation, including thyroid-stimulating hormone, were otherwise normal. During the first upper gastrointestinal esophagogastroduodenoscopies, the pouch and the gastrojejunal anastomosis (GJA) were characterized by healthy appearing mucosa. In all 3 cases, the pouch size measured between 3 and 5 cm and the GJA was dilated, with diameters of >20 mm (Figure *A*). Laser resurfacing of the stoma by argon plasma coagulation (APC) at 0.8 L/min and 70 watts was successfully performed in a 1-cm concentric ring fashion around the gastric side of the GJA (Figure *B*). Three months after the initial APC, the patients returned for reevaluation. The pouch mucosa again seemed to be normal, but there was persistent, albeit improved, dilation of the GJA. Repeat APC sessions were performed without any adverse events.

On their next follow-up esophagogastroduodenoscopies, all patients were noted to have an abnormal color of approximately 25% of their gastric pouch epithelium, which seemed to be more similar to the esophageal epithelium (Figure *C*). The GJAs remained dilated, measuring >14 mm, and their third APC sessions were performed (Figure *D*). The patients did well after the procedures, with significant weight loss. A 1-year follow-up endoscopy showed a 10-mm GJA, but the gastric pouch was now approximately 80% to 100% covered with this abnormal epithelium (Figure *E*). A forceps biopsy was performed for histologic evaluation (Figure *F*).

What is the histopathologic diagnosis of this finding?

Look on page 2141 for the answer and see the *Gastroenterology* website (www.gastrojournal.org) for more information on submitting your favorite image to Clinical Challenges and images in GI.

Conflicts of interest
The authors disclose the following:
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Answer to: Image 2 (Page 2139): Squamous Metaplasia

Weight regain can accompany reemergence of obesity-related comorbidities and, thus, early intervention is important. Although diet, exercise, and behavior modifications are fundamental, they can have limited efficacy. Thus, endoscopic management is important, with specific evaluation for gastrogastric fistulae, pouch dilation, and GJA dilation, all of which can be successfully intervened upon endoscopically. For GJA dilation in particular, APC has been used with promising results.[1]

In the normal GI tract, the esophagus is lined with squamous epithelium, and the stomach is lined with columnar epithelium. One of the most well-known and well-documented scenarios in which the typical mucosal lining is replaced by abnormal mucosa is Barrett's esophagus (BE). BE is defined by the replacement of the normal distal squamous epithelial lining with columnar epithelium with a minimum length of 1 cm (tongues or circumferential) containing specialized intestinal metaplasia on histopathologic examination. It is well-documented that treatment of BE with thermal ablation and acid suppression therapy results in reepithelialization of the esophagus with neosquamous mucosa.[2] In contrast with this, in our patients, after we burned the gastric columnar mucosa with APC to treat their dilated GJA, the gastric pouch mucosa has been replaced with squamous epithelium, which we have termed "reverse BE." To our knowledge, there are no reports of this condition in the literature, nor do we know the precise cause. There is a series of patients without a history of bariatric surgery who developed squamous metaplasia in the proximal gastric cardia.[3] The authors hypothesized that this condition may be due to chronic mucosal injury owing to hiatal hernia, reflux, caustic ingestion, chronic gastritis, or pyloric stenosis. We suggest two potential mechanisms for this condition in our patients: (1) extending the ablation to the Z-line on the medial aspect of the pouch may allow for the distal extension of squamous mucosa during the healing process; and (2) acid suppression therapy with proton pump inhibitors after the procedure, in combination with a postoperative decrease in acid production, allows for a shift in the cell proliferation and differentiation in the pouch. Notably, although there are well-defined guidelines for surveillance of BE, owing to the risk of progression to esophageal adenocarcinoma,[2] it is unclear what clinical significance this reverse BE may have in the future. It is important to continue to monitor these patients and clarify the natural history of this finding.

References

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