ACG CASE REPORTS JOURNAL

IMAGE | ESOPHAGUS

Dysphagia Caused by *Helicobacter pylori*-Associated Inlet Patch Ulcer

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CASE REPORT

A 52-year-old man without any significant medical history presented with a 2-week history of progressive dysphagia (solid and liquid) without any globus sensation and weight loss. Physical examination was unremarkable. An esophagogastroduodenoscopy revealed an inlet patch (2 cm in diameter) with a linear ulcer in the proximal esophagus (Figure 1). The rest of the esophageal mucosa seems normal without any evidence of stricture, mass, extrinsic compression, and esophagitis. Initial biopsies obtained from the inlet patch and stomach were placed into 2 separate rapid urease test kits (CLOtest; Ballard Medical Products, Draper, UT). Rapid urease test kit colors changed from yellow to red within 30 minutes, which was consistent with *Helicobacter pylori* infection. Histopathologic evaluation of biopsies obtained from the inlet patch revealed gastric oxyntic cells and goblet cells adjacent to normal esophageal mucosa with stratified squamous epithelium with the presence of *H. pylori* (Figure 2). The patient received a triple therapy regimen (rabeprazole, metronidazole, and amoxicillin) for 14 days. The patient's dysphagia completely improved, and the follow-up urea breath test was negative for *H. pylori* infection. The patient did not have any recurrent dysphagia during the second-year follow-up.

Inlet patch is an island of heterotopic gastric mucosa located at the proximal esophagus. The reported endoscopic prevalence of inlet patch is widely variable (range: 0.18%–14.5%).[1,2] Most inlet patches are usually asymptomatic and often overlooked by endoscopists. Reported symptoms include cough, globus sensation, sore throat, hoarseness, excessive throat clearing, heartburn, dysphagia, and regurgitation.[1,3] They are likely due to acid and mucus production. *H. pylori* colonization of the inlet patch is common and is closely correlated with *H. pylori* density in the stomach.[4] *H. pylori* colonization of the inlet patch may potentially predispose to similar disorders associated with gastric colonization, such as peptic ulcer disease.[1] In our case, eradication of *H. pylori* infection of esophageal heterotopic gastric mucosa cured dysphagia symptoms due to inlet patch ulcer. Therefore, careful

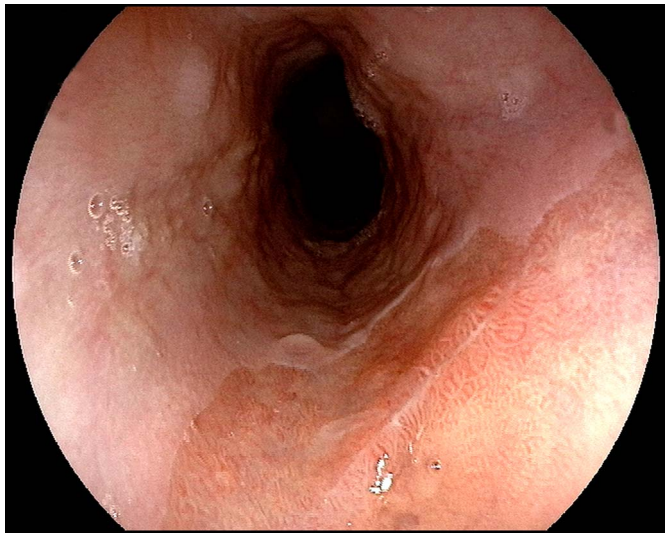


Figure 1. Inlet patch (2 cm in diameter) with a linear ulcer.

ACG Case Rep J 2020;7:e00405. doi:10.14309/crj.0000000000000405. Published online: June 15, 2020

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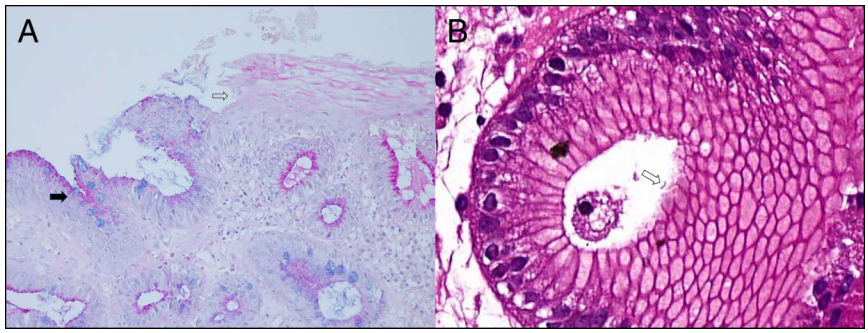


Figure 2. Biopsy obtained from the inlet patch revealed (A) gastric oxyntic cells and goblet cells (solid arrow) adjacent to normal esophageal mucosa with stratified squamous epithelium (open arrow) and (B) *Helicobacter pylori* (arrow).

examination of the cervical esophagus is crucial during the evaluation of a patient with dysphagia. Since recent studies revealed that narrow-band imaging facilitates the detection of inlet patches, we suggest using narrow-band imaging when examining the esophagus.[1]

DISCLOSURES

Author contributions: Both authors contributed equally to this manuscript. T. Attila is the article guarantor.

Financial disclosure: None to report.

Informed consent was obtained for this case report.

Received January 22, 2020; Accepted April 8, 2020

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