



Symptomatic Heterotopic Gastric Mucosa in Distal Esophagus

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Abstract

Introduction Heterotopic gastric mucosa (HGM) in esophagus is commonly noted as an inlet patch at endoscopy. We describe a rare patient with symptomatic distal esophageal HGM.

Case Report A 40-year-old male presented with retrosternal pain and marked odynophagia for the last 4 weeks without any history of ingestion of antibiotics, foreign body, or corrosive. Endoscopy showed abrupt circumferential transition to salmon pink mucosa at 35 cm from incisors. From 35 to 41 cm, there were areas of polypoid edematous thickening with few superficial ulcers of 1 to 3 mm. Squamous epithelium was visualized at narrow band imaging from 41 cm to the Z-line at 43 cm with no hiatus hernia. Biopsy showed gastric-type mucosa with parietal cells without dysplasia. Serology for cytomegalovirus and human immunodeficiency virus was negative. He was managed with proton pump inhibitors (PPIs) and prokinetics and improved symptomatically. Follow-up endoscopy at 3 months demonstrated healing of ulcers with persistence of HGM and pseudopolyps. He remains well on maintenance with PPI at 1-year follow-up.

Keywords

- heterotopic gastric mucosa
- distal esophagus
- retrosternal pain

Conclusion Symptomatic HGM in distal esophagus is rare and can be differentiated from Barrett's esophagus histologically and by presence of squamous epithelium between HGM and stomach. Inflammatory mass lesions may develop and mimic esophageal malignancy. Symptoms are largely due to acid production and usually respond to PPI.

Introduction

Heterotopic gastric mucosa (HGM) is commonly noted in the proximal esophagus as a salmon-colored inlet patch.¹ Most patients are asymptomatic, but few may complain of globus sensation in the throat, hoarseness, dysphagia, or chronic cough.² The presence of HGM in the distal esophagus is rare.³ We describe a patient with symptomatic distal esophageal HGM.

Case Report

A 40-year-old male reported to us with complaints of acute onset of spasmodic retrosternal pain, heartburn, and marked odynophagia for 4 weeks. There was no history of ingestion of antibiotics, foreign body, or corrosive. He did not give history of fever, anorexia, or weight loss. Clinical examination was unremarkable. Blood counts, biochemistry, and ultrasonography of abdomen were normal. Endoscopy showed abrupt

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circumferential transition to salmon pink mucosa at 35 cm from the incisors. From 35 cm to 41 cm, there were areas of edema and polypoid thickening with few superficial ulcers of 1 to 3 mm. Squamous epithelium was visualized at narrow band imaging (NBI) from 41 cm to the Z-line at 43 cm with no hiatus hernia (►Fig. 1). Biopsy from 35 to 41 cm showed gastric mucosa with parietal cells without dysplasia (►Fig. 2A). Serology for cytomegalovirus and human immunodeficiency virus were negative. Contrast-enhanced computed tomography scan chest abdomen showed circumferential mural thickening of 7 mm involving distal esophagus with associated polypoid thickening of 26 × 6 mm along right lateral wall (►Fig. 2B). He was managed with proton pump inhibitors (PPI) and prokinetics and improved symptomatically over 2 weeks. Follow-up endoscopy at 3 months (►Fig. 3) demonstrated healing of ulcers with persistence of HGM and polypoid lesion. He remains well on maintenance PPI at 1 year.

Discussion

Prevalence of HGM in proximal esophagus ranges between 1 and 10%, but it can rarely be found in the distal esophagus.⁴ The pathophysiology of its origin is not well understood.

According to the congenital hypothesis, the process of replacement of columnar with squamous epithelium commences from mid-esophagus at 24 weeks of gestation and extends to the proximal and distal ends.⁵ The proximal esophagus is the last to achieve squamous epithelization, thus explaining the high frequency of HGM in the proximal esophagus as compared with distal esophagus. The second hypothesis attributes HGM to chronic acid exposure due to gastroesophageal reflux, akin to Barrett's esophagus.⁶ Chronic irritation and injury result in inflammation that leads to reactivation or proliferation of remnant columnar mucosa and inhibits the proliferation of stem cells leading to epithelium transformation into a columnar type.

The presence of HGM in the distal esophagus is rare. Similar case is reported by Lupu et al³ in a 16-year-old female with multiple pseudopolypoid formations of gastric heterotopia that are located in the distal part of the esophagus. Endoscopy combined with histology confirmed the presence of pure oxyntic mucosa.

Most patients with HGM are asymptomatic, and no treatment is required. Less than 10% of patients may complain of chest or throat pain, dysphagia, globus sensation, shortness of breath, chronic cough, and hoarseness.^{1,2} Symptoms may



Fig. 1 (A) Transition from normal to salmon pink mucosa at 35 cm. (B) Polypoid lesion and superficial ulcers (38-41 cm). (C) Normal squamous epithelium for 2 cm above Z-line (41-43 cm).

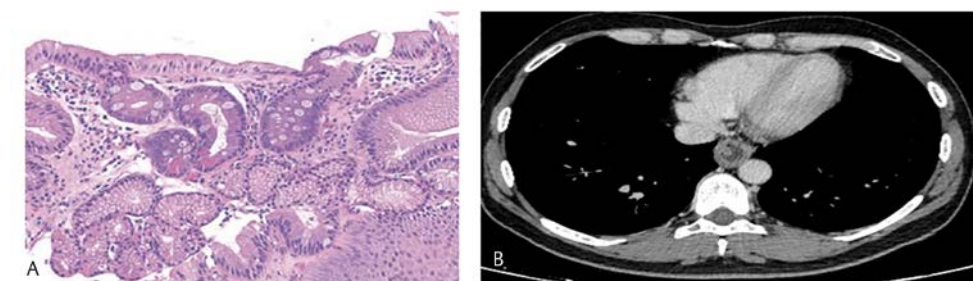


Fig. 2 (A) Gastric-type columnar mucosa with parietal cells without dysplasia (H&E stain). (B) CECT scan showing circumferential mural thickening of distal esophagus with polypoid thickening of right lateral wall.

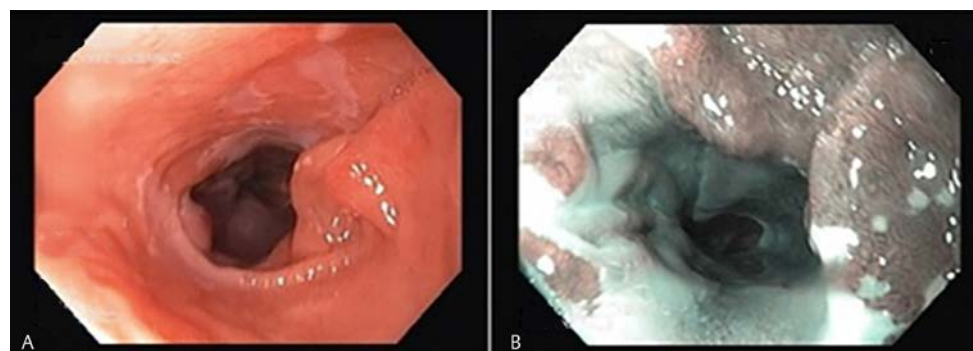


Fig. 3 (A) Healing of ulcers and persistence of polypoid lesion. (B) No change in squamous epithelium (41-43 cm).

be due to production of acid due to the presence of parietal cells or mucus or both. Five categories of HGM have been described by von Rahden et al (► **Table 1**).⁷

Compared with Barrett's esophagus that has intestinal metaplasia of the squamous epithelium, HGM histologically consists of mucus-secreting columnar cells, chief cells, and parietal cells.⁷ In our patient, histopathologic examination revealed parietal cells, without intestinal metaplasia or dysplasia. Also, the columnar epithelium in Barrett's esophagus extends from the stomach to esophagus in continuation.⁸ In our patient, as visualized by NBI, columnar epithelium in distal esophagus was clearly separated from that in the stomach by a 2 cm patch of squamous epithelium. We noted thickening of mucosa with formation of pseudopolyps but, unlike Barrett's esophagus, HGM is not known to be a premalignant condition.⁹ Although *Helicobacter pylori* are known to have strong affinity for gastric-type mucosa, we could not demonstrate the same in our patient.⁹ Treatment with acid suppression, as seen by us, improves symptoms in patients with HGM. Use of ablative modalities such as argon plasma coagulation or radio frequency has been reported in patients refractory to treatment with acid suppression.¹⁰

Conclusion

Symptomatic HGM in distal esophagus is rare and can be differentiated from Barrett's esophagus histologically and by presence of squamous epithelium between HGM and

stomach. Inflammatory mass lesions may develop and mimic esophageal malignancy. Symptoms are largely due to acid production and usually respond to PPI.

Conflict of Interest

None declared.

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Table 1 Clinicopathological classification for heterotopic gastric mucosa

Type	Description of symptoms	Findings
I	Asymptomatic	None
II	Symptomatic without benign complications	None/nonspecific
III	Symptomatic with benign complications	Strictures/webs/fistula/ bleeding
IV	Dysphagia	Intraepithelial dysplasia
V	Dysphagia	Malignant transformation