CASE REPORT

Endoscopic Submucosal Dissection of Gastric High-Grade Foveolar Dysplasia With Normal Background Mucosa

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Most gastric neoplastic lesions appear in patients with gastric premalignant conditions. Here, we present the case of a 75-year-old woman with no prior history of *Helicobacter pylori* infection, with a big gastric adenoma resected by endoscopic submucosal dissection. Histopathological examination revealed high-grade foveolar dysplasia. Interestingly, surrounding mucosa was normal, without signs of *H. pylori* infection or gastric preneoplastic conditions. The presented case emphasizes that high-risk gastric lesions may be present within a normal stomach without endoscopic signs of *H. pylori* infection or premalignant conditions. This underscores the importance of careful examination in regular practice of esophagogastroduodenoscopy, even in low-risk patients.

Keywords: Endoscopic Resection; Gastrointestinal Endoscopy; *Helicobacter pylori*; Stomach Neoplasms

Introduction

Gastric cancer is one of the leading causes of cancer-related deaths worldwide.[1] Most of the mortality due to gastric cancer is related to diagnosis in advanced stages with poor prognosis.[2] Gastric adenocarcinoma is the most common type of gastric cancer, especially the intestinal type. The relation with *Helicobacter pylori* (*H. pylori*) infection and gastric adenocarcinoma has been well-documented, and International Agency for Research on Cancer has declared *H. pylori* as a type I carcinogen since 1994.[3] After *H. pylori* infection, inflammatory driven changes of the gastric mucosa may lead to chronic atrophic gastritis with or without intestinal metaplasia and dysplasia (ie, low- or high-grade),[4] with an increased risk to progress to gastric adenocarcinoma.[5–7] Furthermore, eradication of *H. pylori* after treatment of gastric dysplasia has been associated with a reduction in the risk of recurrence.[8]

Endoscopic surveillance of gastric premalignant conditions has been proposed by international guidelines in high-risk populations.[9–12] This strategy may lead to early detection of gastric cancer. However, less attention has been given to gastric dysplasia emerging from normal gastric mucosa without *H. pylori* infection or gastric preneoplastic conditions, which may be related to sporadic gastric cancer cases in lower risk areas.

Here, we present the case of a gastric adenoma with high-grade dysplasia emerging from a normal background gastric mucosa, without signs of *H. pylori* infection or gastric preneoplastic conditions.

Case Report

A 75-year-old female patient was referred to Kingston Health Sciences Centre Endoscopy Unit for the finding of gastric lesion in the anterior wall of proximal body in an esophagogastroduodenoscopy (EGD) performed for foregut symptoms. Her medical records included hypothyroidism, obesity, type 2 diabetes, gastroesophageal reflux, and she had active smoking habit (50 packs/year). There was no family history of gastric cancer, and *H. pylori* blood serology was negative.

A detailed EGD with appropriate gastric cleaning and insufflation was performed. A ∼60 mm wide flat elevated lesion (0-IIa) was found on the greater curvature and anterior wall of proximal body using white light endoscopy. Magnifying endoscopy using blue light imagine revealed regular papillary surface mucosal pattern with slightly irregular thin vascular arrangement in some parts of the lesion and a small area with loss of the surface mucosal pattern and irregular and branched thin vascular pattern (Figure 1). Background gastric mucosa surrounding the lesion showed regular arrangement of collecting venules and normal foveolar appearance.

Endoscopic submucosal dissection was performed (Figure 2). Briefly, margins of the lesion were marked using soft coagulation. The lesion was elevated with submucosal injection with saline solution. Complete circumferential mucosal incision was performed using a Flush Knife BTS

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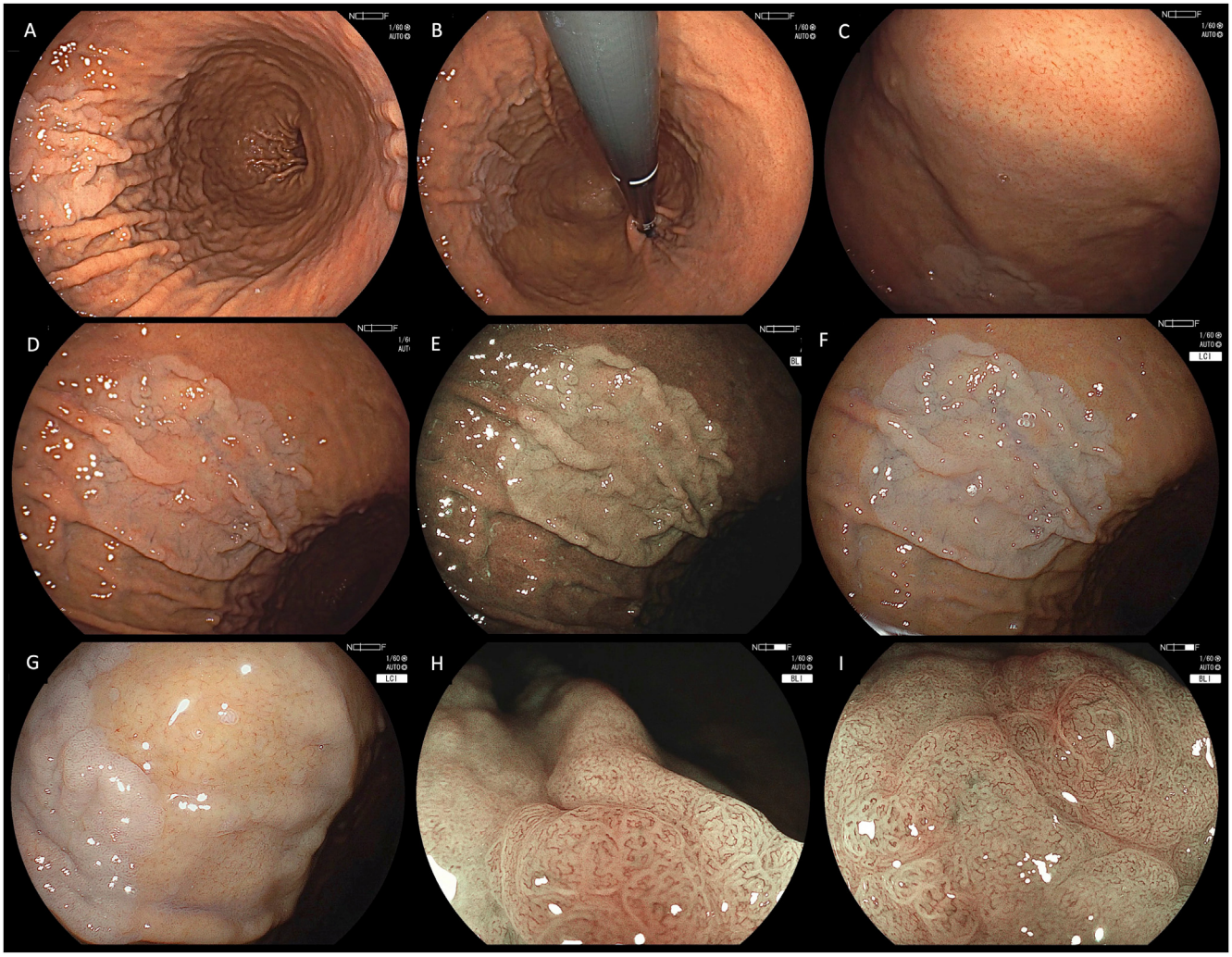

Figure 1. Endoscopic findings of gastric foveolar-type epithelial dysplasia. (A) White light endoscopy (WLE) forward view of proximal gastric body with a whitish flat elevated lesion (0-IIa) on the greater curvature and anterior wall. (B) Retroflex view of the lesion in the greater curvature with white light. (C) Regular arrangement of collecting venules (RAC) and normal foveolar mucosa surrounding lesion observed with WLE. (D) WLE, (E) blue light imagine (BLI), and (F) linked color imaging (LCI) of flat elevate lesion (0-IIa) surrounded by normal background mucosa. (G) Margins of the lesion and normal background oxyntic mucosa with RAC observed with LCI. (H) Magnifying endoscopy using BLI shows a regular papillary surface pattern with regular vascular arrangement and (I) small area with loss of the surface pattern and irregular and branched thin vascular pattern.

(Fujifilm, Japan). Clip and band traction method was applied using clips with elastic band attached to the proximal and distal sides of the lesion to better expose the submucosa. Submucosal dissection was performed with a Flush Knife BTS and PreciseSECT (ERBE, Germany). *En bloc* resection was achieved. There were no signs of deep mural injury among the resection ulcer, and satisfactory hemostasis was accomplished. Resected specimen measured 85 × 60 mm. Histopathological examination revealed high-grade foveolar dysplasia (Figure 3). Lateral margins were clear with a final size of the lesion of 60 × 55 mm. No signs of carcinoma were found. Surrounding gastric mucosa was normal without *H. pylori* infection, chronic atrophic gastritis, or intestinal metaplasia.

Discussion

Gastric adenomas are defined as any protruding lesion from the gastric mucosa with the presence of epithelial dysplasia of any grade. Strikingly, gastric adenomas are much more infrequent than the colon adenomas. Gastric epithelial dysplasia can be classified, according to its histologic characteristics and mucous expression patterns, into intestinal type (the most common) or foveolar type (also known as gastric type).[13] *Abraham et al* described in a series of 61 gastric adenomas that intestinal-type dysplasia was more commonly associated to *H. pylori* infection and intestinal metaplasia in the background mucosa, contrasted with foveolar-type dysplasia, which was more commonly associated to normal background gastric mucosa.[14] The relative risk of progression to

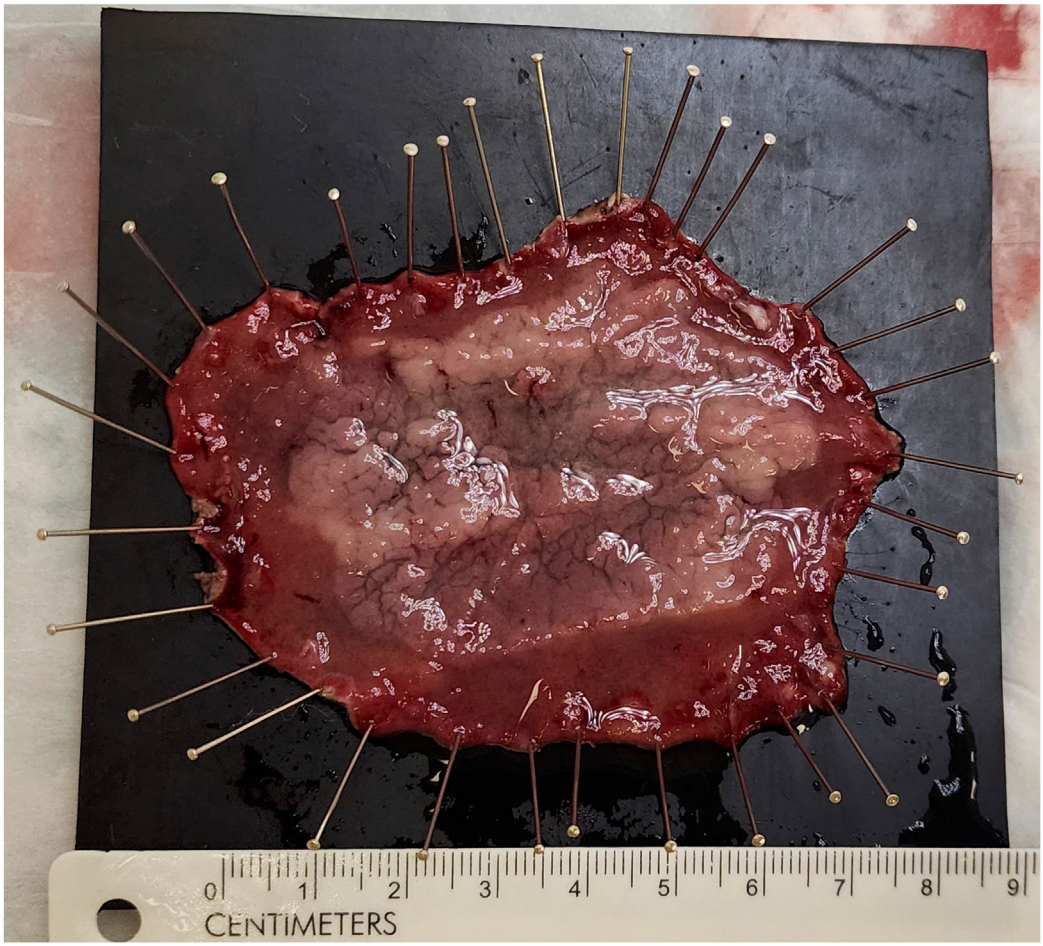

Figure 2. Extended endoscopic submucosal dissection specimen.

adenocarcinoma between intestinal and foveolar-type dysplasia has not been well established. It has been suggested that high-grade dysplasia and adenocarcinoma within the lesion are more commonly found among intestinal-type adenomas in Western populations.[14] In contrast, Eastern studies have shown a higher risk of adenocarcinoma among foveolar-type dysplasia.[15]

Magnifying endoscopy and virtual chromoendoscopy can help to characterize focal gastric lesion to assess the risk of high-grade dysplasia or adenocarcinoma and define the best resection strategy. These tools are regularly used for the assessment of neoplastic gastric lesions. However, very scarce studies have evaluated its role in the characterization of morphological subtype of the dysplasia among gastric lesions. *Kang et al* compared the endoscopy characteristics of intestinal *vs* foveolar dysplasia subtype in a series of gastric dysplasia.[16] Concordantly to our report, they described a papillary, regular surface pattern in foveolar dysplasia, with a lower frequency of *H. pylori* infection in the background mucosa compared to intestinal dysplasia subtype.

The presented case report emphasizes that gastric high-risk lesions, particularly high-grade foveolar dysplasia, may be present in an apparently normal stomach without endoscopic signs of *H. pylori* infection. This underscores the importance of careful cleaning and examination of the entire stomach in the regular practice of EGD, even in low-risk patients. Missing these types of lesions may lead to interval gastric cancer (ie, a gastric adenocarcinoma that appears after a normal EGD within the past 3 years).[17] In this context, several clinical guidelines have made practical recommendations and proposed quality indicators to improve the quality of diagnostic EGD and reduce the risk of missing lesions.[18–20] Currently, proper sedation, premedication with mucolytic or defoaming agents, stomach cleaning, careful examination, complete photo documentation, sufficient observation time, application of virtual chromoendoscopy, characterization of suspicious lesions, and biopsy collection are considered essential parts of a high-quality EGD.

In conclusion, high risk lesions such as foveolar high-grade dysplasia may be seen in patients with apparently normal gastric mucosa. Therefore, a detailed and high-quality EGD must be attained even in low-risk patients.

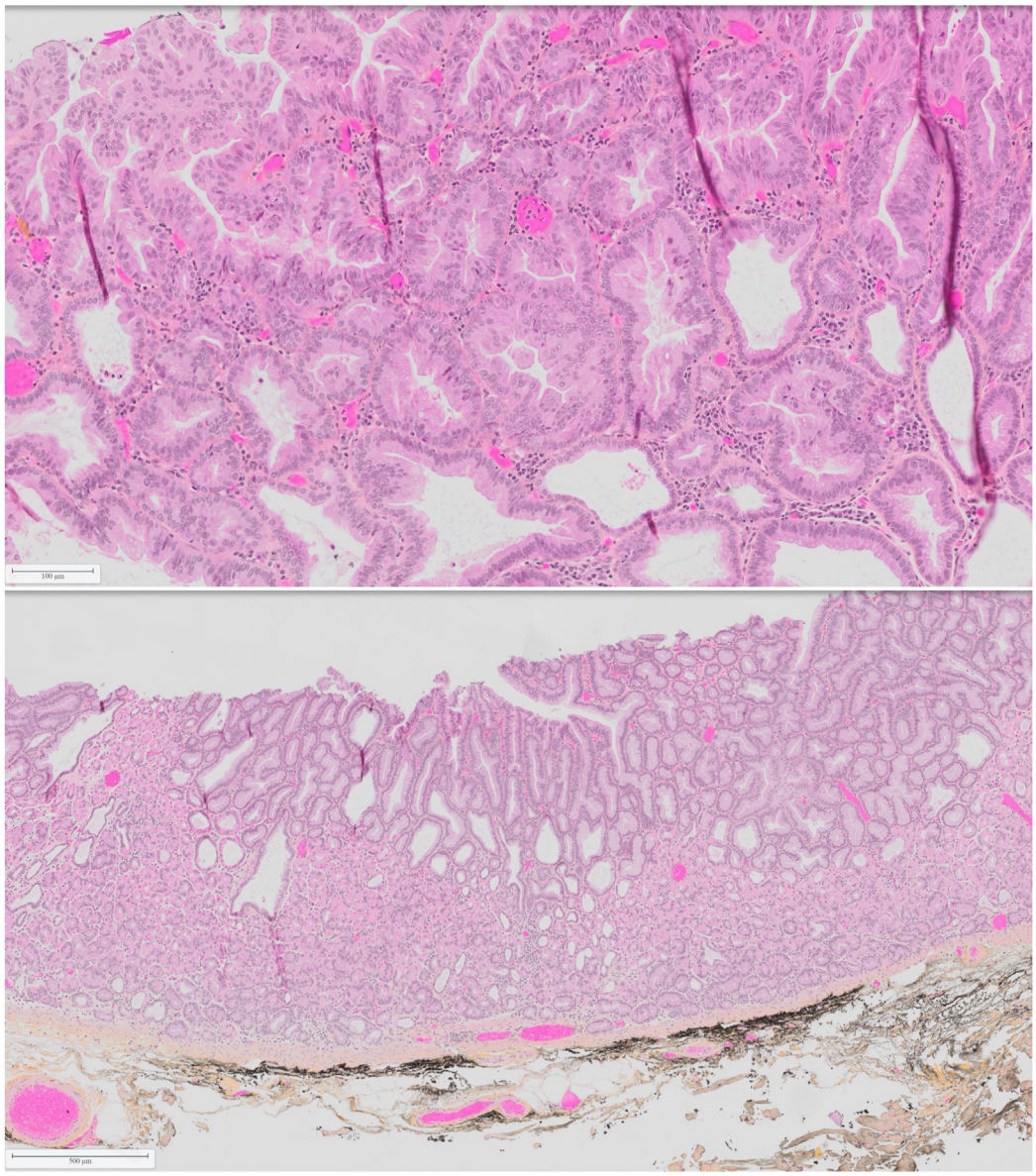

Figure 3. Histopathological examination of the resected specimen (60 × 55 mm). Hematoxylin and eosin (H&E) staining of resected specimen revealed high-grade foveolar dysplasia with negative lateral margins and no signs of carcinoma (above). Surrounding gastric mucosa was normal without *Helicobacter pylori* infection (below).

References

1. Bray F, et al. CA Cancer J Clin 2024;74:229–263.
2. Balakrishnan M, et al. Curr Gastroenterol Rep 2017; 19(8):36.
3. International Agency for Research on Cancer. IARC Monogr Eval Carcinog Risks Hum 1994;61:1–241.
4. Correa P. Cancer Res 1988;48:3554–3560.
5. Rugge M, et al. Am J Gastroenterol 2018; 113(11):1621–1628.
6. Lee JWJ, et al. Gut 2022;71(5):854–863.
7. Latorre G, et al. Gut 2024;73(10):e18.
8. Yoo HW, et al. Gastroenterology 2024;166(2):313–322.e3.
9. Banks M, et al. Gut 2019;68(9):1545–1575.
10. Gupta S, et al. Gastroenterology 2020;158(3):693–702.
11. Shah SC, et al. Gastroenterology 2021; 161(4):1325–1332.e7.
12. Pimentel-Nunes P, et al. Endoscopy 2019; 51(4):365–388.
13. Lauwers GY, et al. Gut 1999;45(5):784–790.
14. Abraham SC, et al. Am J Surg Pathol 2002; 26(10):1276–1285.
15. Park do Y, et al. Am J Surg Pathol 2008;32:524–533.
16. Kang HM, et al. World J Gastroenterol 2014; 20(42):15771–15779.
17. Kim TJ, et al. Clin Gastroenterol Hepatol 2023; 21(5):1205–1213.e2.
18. Bisschops R, et al. Endoscopy 2016;48:843–864.
19. Kim SY, et al. Clin Endosc 2022;55(3):319–331.
20. Emura F, et al. Dig Endosc 2020;32(2):168–179.

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The corresponding author, on behalf of all authors, certifies that patient consent was obtained to publish this case and accompanying images.

Reporting Guidelines:
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