Jordan Medical Journal

ORIGINAL ARTICLE

Case Report: A Duodenal Polyp with Gastric Foveolar Metaplasia and Dysplasia

Khairat Battah[1], Arwa Battah[2], Mohammad AlKhreisat[3], Ruba Malkawi[4]

[1] Department of Pathology, Microbiology, and Forensic Medicine, Faculty of Medicine, Al-Balqa Applied University, Al Salt, Jordan.

[2] Nephrology Department, Rutgers University, University Hospital, Newark

[3] Department of Special Surgery, Faculty of Medicine, Al-Balqa Applied University, Al Salt, Jordan

[4] College of Pharmacy, Jadara University, Irbid, Jordan.

*Corresponding author:

Kh.Battah@bau.edu.jo

Received: August 5, 2024

Accepted: September 29, 2024

DOI:
https://doi.org/10.35516/jmj.v59i3.2866

Abstract

Duodenal polyps are a common finding during endoscopic investigations, typically representing benign conditions such as regenerative or reactive changes and gastric heterotopia. Some regenerative changes can present as metaplasia, particularly of the gastric foveolar epithelial type. However, adenomatous changes associated with gastric foveolar epithelium are rare. This article presents a case of a gentleman whoexperiened dyspepsia and dysphagia. Endoscopy revealed a polypoid lesion that exhibited low-grade dysplasia with gastric foveolar metaplasia upon histopathologic examination. This case supports the hypothesis that metaplastic epithelium in the duodenum can serve as a precursor to neoplastic processes, underscoring the importance of vigilant surveillance and the necessity for further molecular investigations.

Keywords: Histopathology, gastric foveolar metaplasia, dysplasia

INTRODUCTION

Gastric foveolar metaplasia (GFM) and heterotopic gastric mucosa (HGM) in the duodenum are notable for their distinct histological features and potential clinical implications. GFM generally results from various types of injuries, such as peptic damage, H. pylori infection, or chronic inflammation, and is considered a reactive or regenerative change [1]. In contrast, HGM, which is histologically identical to normal oxyntic gastric mucosa, has traditionally been viewed as a congenital anomaly [1].

GFM is a common finding in duodenal biopsies; however, dysplasia associated with GFM is limited in the literature. Recent research indicates that a subset of GFM in the duodenum, originating from unknown causes,

may precede adenomatous changes and carry neoplastic potential [2]. This subset may exhibit a tendency for neoplastic transformation similar to that observed in metaplastic epithelium elsewhere in the gastrointestinal tract [2], potentially acting as precursors to gastric foveolar adenomas (GFA) and gastric-type adenocarcinomas [3,4].

Herein, we report a case of a gentleman who presented with upper gastrointestinal tract symptoms. The patient underwent an upper endoscopy, which identified two abnormalities: an extensive hyperplastic change in the first part of the duodenum (D1) and a flat polyp in the duodenal bulb (D2). Histological examination revealed low-grade dysplasia with GFM in the first lesion and HGM in the second lesion.

Case Presentation

A 58-year-old gentleman presented with a history of dysphagia and dyspepsia for one month. The patient had no current or past history of H. pylori infection, no history of prolonged non-steroidal anti-inflammatory drug (NSAID) use, and no significant past medical history or previous gastrointestinal surgeries. There was no family history of gastrointestinal cancers. A urea breath test for H. pylori was negative. Laboratory tests, including CBC and liver function tests, were within normal limits. The patient subsequently underwent an upper endoscopy. During the procedure, two abnormalities were identified. The first lesion exhibited polypoid changes measuring approximately 2.5 cm in the first part of the duodenum (D1), and the second was a flat polyp measuring 3 cm in the duodenal bulb (D2). The background duodenal and gastric mucosa appeared normal. Biopsies were taken from both lesions to determine their histopathological nature and decide the appropriate management.

Pathological Findings

Three formalin-fixed small pieces of tissue were received from the abnormal area in D1. Four formalin-fixed small pieces of tissue were received from the polyp in D2.

Biopsies from D1 showed glandular mucosa with columnar epithelium similar to foveolar cells in the gastric mucosa, characterized by an apical mucin cap (Figures 1, 2A). The lamina propria displayed mild mixed inflammation. Notably, there were areas with villous or irregularly papillary structures lined by cells exhibiting nuclear stratification and occasional enlargement. These features are consistent with low-grade dysplasia (Figures 1, 2B).

Histological examination of biopsies from the duodenal bulb revealed gastric glands composed of a surface gastric foveolar epithelium with pyloric-type glands and some chief and parietal cells (Figure 3). These findings are consistent with HGM in the duodenal bulb.

DISCUSSION

The duodenum, especially its second portion, is susceptible to developing small intestinal polyps [5]. Duodenal polyps are classified according to WHO criteria into ampullary or non-ampullary polyps [6]. Non-ampullary polyps are mostly non-neoplastic and often represent metaplastic foveolar epithelium with hyperplasia/regeneration or proliferations of Brunner glands [5]. Less common duodenal polyps in the non-ampullary region include heterotopic and neoplastic lesions. Among the neoplastic lesions, intestinal-type adenomas outnumber the adenomas with a gastric epithelial phenotype, the latter including pyloric gland adenomas and gastric foveolar adenomas (GFA) [6,7].

GFM in the duodenum is typically a reactive or reparative condition resulting from chronic inflammation, peptic injury, or H. pylori infection [5]. Similar to other gastrointestinal metaplasias, such as intestinal metaplasia in the esophagus and stomach, GFM may have a subset that proceeds adenomatous changes [3,4]. In fact, Matsubara et al. found mutations in GNAS and/or KRAS in nearly half of duodenal GFM and about one-third of HGM cases [4].

Rubio CA found that duodenal adenomas with gastric foveolar epithelium are not necessarily associated with H. pylori infection [2], suggesting other pathogenic pathways for metaplastic and dysplastic changes. The pathogenesis of dysplasia in the context of GFM and HGM is not fully understood. However, it is hypothesized that as the duodenal epithelium is exposed to high levels of bile acids along with pancreatic digestive enzymes, this may contribute to chronic mucosal injury, leading to metaplastic changes and potentially initiating neoplastic transformation.

The number of GFAs reported in the literature is limited. Most cases present as small protruding polyps around 1 cm in size and are histologically characterized by columnar cells that resemble gastric foveolar epithelium, featuring elongated, stratified nuclei and an apical mucin cap, often displaying a tubulovillous structure [3,8]. These adenomas can arise from a background of foveolar metaplasia or gastric heterotopia [3]. This reported case demonstrates similar histopathological features, along with inflammatory changes in the lamina propria, indicating a reactive or possibly reparative process alongside a spectrum of metaplastic and hyperplastic changes to low-grade adenomatous changes, consistent with literature reports [3,4].

The risk of transformation of GFAs to gastric-type adenocarcinoma is related to the size of the adenoma, the villous component, and the presence of high-grade dysplasia [6]. The literature varies regarding the grade of dysplasia typically associated with GFA in the duodenum. Some studies, such as those by Kővári B et al., suggest that GFAs have a higher tendency to harbor high-grade dysplasia [5]. In contrast, other studies found that GFAs in the duodenum typically harbor low-grade dysplasia and that none of the adenomas in these studies transformed into adenocarcinoma on follow-up [9]. In this case, the low-grade dysplasia observed highlights the need for careful monitoring and follow-up.

CONCLUSION

This case underscores the importance of recognizing foveolar metaplasia in the duodenum, as there is a potential for dysplastic changes. Although the occurrence of such changes in foveolar metaplasia is rare, this case emphasizes the need for careful histopathological examination and close follow-up. Further molecular studies could provide additional insights into the pathogenesis and potential malignant transformation associated with this condition.

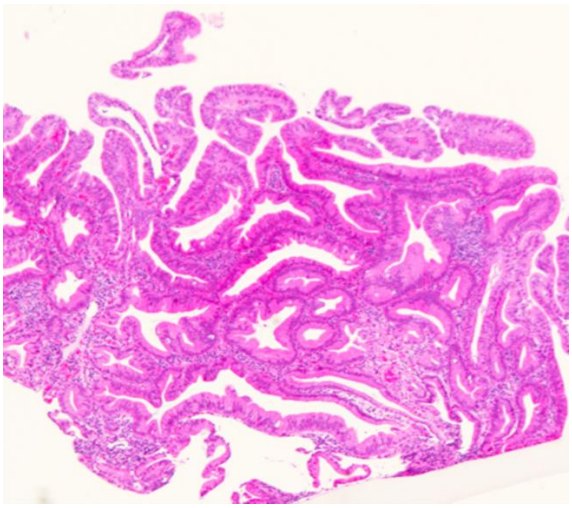

Figure 1: Duodenal adenoma demonstrating villous and irregular papillary structures lined by cells exhibiting nuclear stratification, consistent with low-grade dysplasia. Hematoxylin- eosin stain. x4.

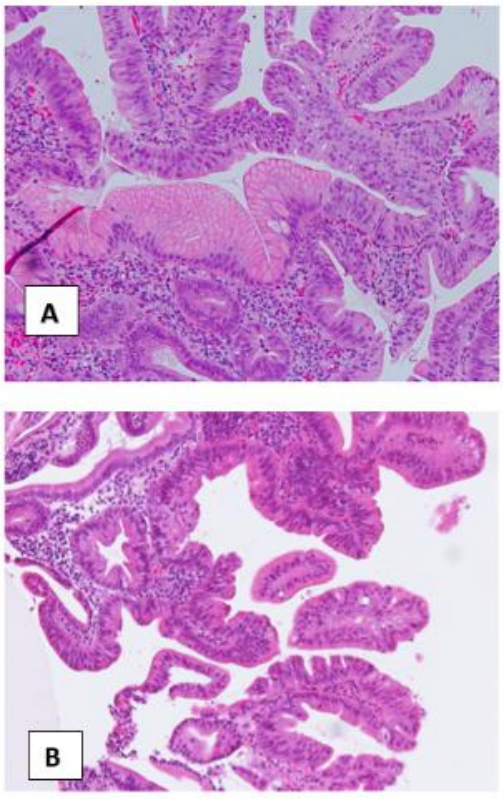

Figure 2: A. Higher power view demonstrating glandular mucosa with columnar cells and characteristic apical mucin cap similar to gastric foveolar cells. Hematoxylin- eosin stain, x10.

B. Higher power view demonstrating villous structure with elongated and stratified with some pleomorphism. Hematoxylin- eosin stain, x10.

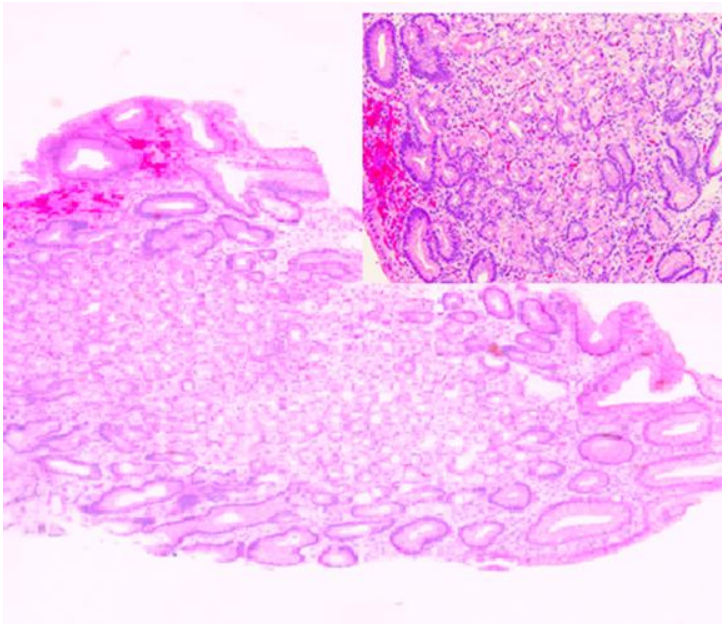

Figure 3: Gastric heterotopia characterized by gastric glands composed of a surface gastric foveolar epithelium with pyloric type glands and some chief and parietal cells (inset).

REFERENCES

1. Riddell R, Jain D. Lewin, Weinstein, and Riddell's Gastrointestinal Pathology and Its Clinical Implications. Vol 2. 2nd ed. Philadelphia, PA: Wolters Kluwer/Lippincott Williams & Wilkins Health; 2014:1331–1335.
2. Rubio CA. Gastric duodenal metaplasia in duodenal adenomas. J Clin Pathol. 2007 Jun;60(6):661-3. doi: 10.1136/jcp.2006.039388. Epub 2006 Jul 12. PMID: 16837629; PMCID: PMC1955048.
3. Mitsuishi T, Hamatani S, Hirooka S, Fukasawa N, Aizawa D, Hara Y, Dobashi A, Goda K, Fukuda T, Saruta M, Urashima M, Ikegami M. Clinicopathological characteristics of duodenal epithelial neoplasms: Focus on tumors with a gastric mucin phenotype (pyloric gland-type tumors). PLoS One. 2017 Apr 4;12(4):e0174985. doi: 10.1371/journal.pone.0174985. PMID: 28376132; PMCID: PMC5380350.
4. Matsubara, A., Ogawa, R., Suzuki, H., Oda, I., Taniguchi, H., Kanai, Y., Kushima, R. and Sekine, S., 2015. Activating GNAS and KRAS mutations in gastric foveolar metaplasia, gastric heterotopia, and adenocarcinoma of the duodenum. British journal of cancer, 112(8), pp.1398-1404.
5. Kővári B, Kim BH, Lauwers GY. The pathology of gastric and duodenal polyps: current concepts. Histopathology. 2021 Jan;78(1):106-124. doi: 10.1111/his.14275.
6. Digestive System Tumours, WHO Classification of Tumours 2019, 5th Edition, Volume 1, 118- 120.
7. Khor TS, Brown I, Kattampallil J, Yusoff I, Kumarasinghe MP. Duodenal adenocarcinoma arising from a pyloric gland adenoma with a brief review of the literature. BMJ Case Rep. 2010 Dec 29; 2010: bcr1020103385. doi: 10.1136/bcr.10.2010.3385. PMID: 22802482; PMCID: PMC3028104.
8. Chen ZM, Scudiere JR, Abraham SC, Montgomery E. Pyloric gland adenoma: an entity distinct from gastric foveolar type adenoma. Am J Surg Pathol. 2009 Feb; 33(2): 186-93. doi: 10.1097/PAS.0b013e31817d7ff4. PMID: 18830123.

9. Chen ZM, Scudiere JR, Abraham SC, Montgomery E. Pyloric gland adenoma: an entity distinct from gastric foveolar type adenoma. Am J Surg Pathol. 2009 Feb; 33 (2): 186-93. doi: 10.1097/PAS.0b013e31817d7ff4. PMID: 18830123.
10. Katrina Collins, Saverio Ligato; Duodenal Epithelial Polyps: A Clinicopathologic Review. Arch Pathol Lab Med 1 March 2019; 143 (3): 370–385. doi: https://doi.org/10.5858/arpa.2018-0034-RA.
11. Riddell R, Jain D. Lewin, Weinstein, and Riddell's Gastrointestinal Pathology and Its Clinical Implications. Vol 2. 2nd ed. Philadelphia, PA: Wolters Kluwer/Lippincott Williams & Wilkins Health; 2014:1331–1335.
12. Ramzi Mulki, Vaidehi Avadhani, Parit Mekaroonkamol, Mo1304 FOVEOLAR GASTRIC METAPLASIA OF THE DUODENUM: AN ENDOSCOPIC MIMCKER OF DUODENAL AND AMPULLARY ADENOMAS, Gastrointestinal Endoscopy, Volume 89, Issue 6, Supplement, 2019, Pages AB481-AB482, ISSN 0016-5107.

Carbone R, Rovedatti L, Lenti MV, Furlan D, Errichiello E, Gana S, Luinetti O, Arpa G, Alvisi C, De Grazia F, Valente EM, Sessa F, Paulli M, Vanoli A, Di Sabatino A. Histologic heterogeneity and syndromic associations of non-ampullary duodenal polyps and superficial mucosal lesions. Dig Liver Dis. 2021 Dec;53(12):1647-1654. doi: 10.1016/j.dld.2021.03.011. Epub 2021 Apr 1. PMID: 33814312.

تقرير حالة: ورم إثني عشري مع تحول نسيجي فوفيولار (الحؤول النقري) مع تغيرات ورمية

خيرات بطاح[1]، أروى بطاح[2]، محمد الخريسات[3]، رُبى مَلكاوي[4]

قسم علم الأمراض، الأحياء الدقيقة، والطب الشرعي، كلية الطب، جامعة البلقاء التطبيقية، السلط، الأردن.

قسم أمراض الكلى، جامعة روتجرز، مستشفى الجامعة، نيوارك.

قسم الجراحة الخاصة، كلية الطب، جامعة البلقاء التطبيقية، السلط، الأردن.

كلية الصيدلة، جامعة جدارا، إربد، الأردن.

Received: August 5, 2024

Accepted: September 29, 2024

DOI: https://doi.org/10.35516/jmj.v59i3.2866

الملخص

خلفية الدراسة والأهداف : تُعتبر الاورام الإثنا عشرية اكتشافًا شائعًا خلال الفحوصات التنظيرية، وغالبًا ما تمثل حالات حميدة مثل التغيرات التجديدية أو التفاعلية والتباين المعدي. قد تظهر بعض التغيرات التجديدية كتحول نسيجي، خاصةً من نوع الخلايا الفوفيولاريه (النقرية) المعدية.

منهجية الدراسة : إلا أن التغيرات الورمية المرتبطة بالظهارة الفوفيولارية المعدية نادرة. يعرض هذا المقال حالة لرجل كان يعاني من عسر الهضم وصعوبة في البلع.

النتائج : أظهرت نتائج التنظير الداخلي وجود سليلة تظهر تغيرات ورمية منخفضة الدرجة مع تحول نسيجي فوفيولاري معدي عند الفحص النسيجي. تدعم هذه الحالة الفرضية التي تشير إلى أن الظهارة المتحولة في الإثنا عشر يمكن أن تكون مقدمة لتحولات ورمية،

االاستنتاجات : يؤكد على أهمية المراقبة اليقظة والحاجة إلى مزيد من الدراسات الجزيئية.

الكلمات الدالة: علم الأنسجة، التحول النسيجي الفوفلاري المعدي، التغيرات الورمية