DOI: 10.7860/NJLM/2024/69874.2862

Case Report

Pathology Section

Coexistence of Gastric Hyperplastic Polyp and Polypoid Foveolar Hyperplasia: A Case Report

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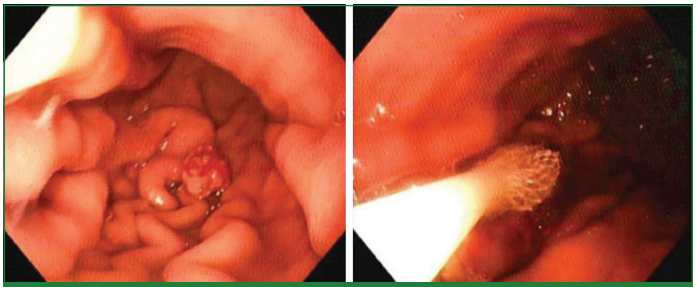
ABSTRACT

Benign gastric epithelial lesions, known as gastric hyperplastic polyps, include concomitant inflammatory alterations and elongated or convoluted foveolae and cystically dilated glands. It is believed that polypoid foveolar hyperplasia, a regenerative lesion associated with both acute and chronic mucosal damage, as well as, chronic gastritis, may serve as a precursor to gastric hyperplastic polyps. In the presented case, a 64-year-old female with a history of *Helicobacter pylori (H. pylori)*-associated chronic gastritis complained of abdominal pain, heartburn and regurgitation for one year. During endoscopy, three polyps were identified, one at the gastroesophageal junction, another below the gastroesophageal junction, and a third at the distal body of the stomach, of which only two polyps were sent for HPE. Gross inspection revealed that both soft-tissue masses were globular, with the cut-section of the polyp from the stomach's distal body showing haemorrhagic areas. Under the microscope, the polyp from the stomach's distal body revealed hyperplastic foveolar epithelium that was distorted, branching, elongated and dilated, situated in an oedematous, inflammatory stroma. The polyp just below the gastroesophageal junction displayed characteristics of polypoid foveolar hyperplasia, including elongated pits devoid of dilation features. The final diagnosis for the polyp from the distal body of the stomach was gastric hyperplastic polyp, and the polyp from just below the gastroesophageal junction was diagnosed as polypoid foveolar hyperplasia. Malignant transformation affects hyperplastic polyps, but foveolar polypoid hyperplasia remains unaffected, underscoring the importance of careful and diligent examination to distinguish between the two conditions.

Keywords: Corkscrew-like pattern, Malignant transformation, Tortuous foveolae

CASE REPORT

A 64-year-old female patient with Hypertension and Type II Diabetes Mellitus, who was on Proton Pump Inhibitors for 5 years, anti-diabetic and anti-hypertensive medications, presented with abdominal soreness, heartburn, regurgitation for one year associated with two episodes of black stool, to the surgical gastroenterology department. This individual had a history of anaemia and chronic gastritis, with audible bowel sounds and a soft, non tender abdomen during a physical examination. Her Glycated Haemoglobin (HbA1c) was 6.6%, and haemoglobin was 7.1 g/dL, resulting in a 1-pint Packed Red Blood Cell (PRBC) transfusion being performed prior to the endoscopic polypectomy. During the endoscopy, three polyps were discovered: a 1 cm polyp at the distal body of the stomach [Table/Fig-1], one 0.5 cm polyp at the gastroesophageal junction and another 0.5 cm polyp, below the gastroesophageal junction [Table/Fig-2]. The provisional diagnoses for the polyps were gastric polyps.

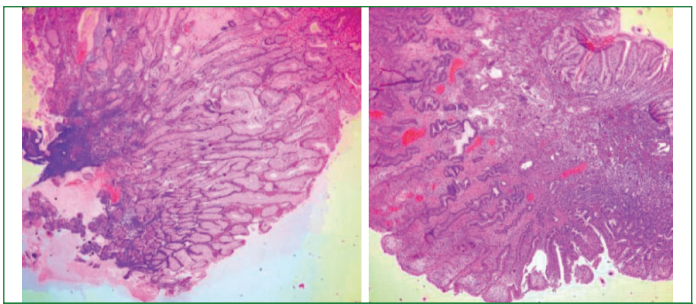


[Table/Fig-1]: Polyp at the distal body of the stomach. **[Table/Fig-2]:** A polyp below the gastroesophageal junction, inside the endoscopy basket. (Images from left to right)

Following the endoscopic polypectomy, the polyps from the distal stomach body and below the gastroesophageal junction were taken to the Pathology laboratory. Upon gross examination, both soft tissue masses received were globular. The cut-section of the polyp from the distal body of the stomach revealed haemorrhagic areas.

The histopathological examination of the polyp from the distal body of the stomach revealed fragments of a gastric polyp showing large areas of ulceration, oedema with focal regeneration. The polyp exhibited hyperplastic foveolar epithelium that was dilated, distorted, branched and elongated, within an oedematous, inflammatory stroma with an abundant blood supply. Additionally, there were tiny, haphazardly arranged smooth muscle bundles [Table/Fig-3].

The histopathological examination of the polyp from just below the gastroesophageal junction showed features of polypoid foveolar hyperplasia (lesion less than 10 mm) composed of elongated pits without dilatation features. There was oedema in the lamina propria. There was no evidence of malignancy in either specimen [Table/Fig-4]. The final impression of the polyp from the distal body of the stomach was gastric hyperplastic polyp, and the polyp from just below the gastroesophageal junction was diagnosed as polypoid foveolar hyperplasia.



[Table/Fig-3]: Polyp with elongated, distorted, branched, and dilated hyperplastic foveolar epithelium (H&E, 10x).
[Table/Fig-4]: Polyp with elongated pits and a characteristic corkscrew appearance (H&E, 10x). (Images from left to right)

DISCUSSION

The second most prevalent type of gastric polyps is called hyperplastic polyps. In areas of malignant transformation, p53

overexpression and Tumor Protein p53 (TP53) mutation are observed [1,2,3]. Hyperplastic polyps are most commonly found in patients in their sixth and seventh decades of life, comprising between 15% and 75% of all gastric polyps [4]. These are localised, non neoplastic expansions of the stomach mucosa associated with inflammatory changes [2]. They exhibit elongated or tortuous (hyperplastic) foveolae, an oedematous lamina propria, distended vessels and cystically dilated glands [5]. Reactive lesions, known as gastric hyperplastic polyps, result from the body's healing and regenerative responses to mucosal damage, unlike neoplastic polyps in the colon [4]. The formation of a hyperplastic polyp is believed to be a consequence of this damage process and the mucosal healing response triggered by inflammation. These progress through stages of polypoid foveolar hyperplasia and foveolar hyperplasia sequentially [5].

Hyperplastic polyps often occur in the setting of intestinal metaplasia (37%), *H. pylori* gastritis (25%), chemical/ reactive gastropathy (21%), and autoimmune gastritis (12%). These conditions are typically associated with underlying gastritis [6]. Additionally, less common conditions linked with hyperplastic polyps include Cytomegalovirus (CMV) gastritis, lymphocytic gastritis, amyloidopathy, Zollinger-Ellison syndrome and Gastric Antral Vascular Ectasia (GAVE) [2,7].

In areas of malignant transformation, p53 overexpression and Tumour Protein p53 (TP53) mutation are observed [1,2,3].

Macroscopically, gastric hyperplastic polyps may appear as large, lobulated, pedunculated, and eroded polyps or small, dome-shaped polyps typically <1 cm in size [1]. They can develop anywhere in the stomach, such as the body, fundus and cardia, and also at gastroenteric anastomoses, with or without antrum involvement. They are usually solitary and predominantly antral (60%), but can also originate from other locations. Twenty percent of patients have multiple polyps, and cases with more than fifty polyps are referred to as hyperplastic polyposis [6].

Under the microscope, the typical characteristics include dilated, hyperplastic foveolae that are elongated, twisted, branching, and have an abundance of mucinous cytoplasm. They are situated in an oedematous, inflammatory stroma that is highly vascular. Globoid cells may predominate in the glands. The glands may appear corkscrew-shaped or twisted, and the foveolar cells are densely packed. The lamina propria can occasionally be fibrotic, either with or without persistent inflammatory infiltrates. The polyp's surface may appear worn down and show signs of regeneration due to nuclear expansion and cytoplasmic mucin loss [4]. Tiny, randomly spaced smooth muscle bundles may also be present.

Between 1% and 20% of hyperplastic polyps exhibit dysplasia, and 2% of them may contain a carcinoma center, especially in polyps larger than 1 cm [1]. Typically, lesions larger than 5 cm exhibit high-grade dysplasia. Patients over the age of 50 are more prone to developing dysplasia and cancer [5].

Polypoid foveolar hyperplasia is believed to have originated from gastric hyperplastic polyps, and their microscopic architecture is slightly different from the latter [8]. Most cases were classified as polypoid foveolar hyperplasia because they lacked the stromal and epithelial diagnostic features of a classic hyperplastic polyp and were less than 1 cm in their largest dimension at the time of endoscopic sampling [1].

Polypoid foveolar hyperplasia, a regenerative lesion akin to hyperplastic polyps, is associated with various forms of acute and chronic mucosal damage, including chronic gastritis. It often appears adjacent to gastrojejunostomy stomas or at the mucosal margins of carcinomas, ulcers, and surface erosions. Moreover, bile reflux, alcohol use, CMV infection, and the use of non steroidal anti-inflammatory medications may be linked to it. The size of polypoid foveolar hyperplasia can either expand, shrink, or remain constant [5].

Polypoid foveolar hyperplasia typically presents as a sessile lesion that is smaller than 10 mm in size [1]. These polyps can be single or numerous, and they are most commonly found in the antrum and body [5].

The hallmark microscopic feature of polypoid foveolar hyperplasia is simple hyperplasia of the foveolar epithelium with elongated pits; dilatation, cystic alteration, or notable architectural deformation of the epithelium are absent. The presence of luminal serration and elongation of the foveolar epithelium gives it a corkscrew-like pattern. The foveolae are usually densely packed with minimal intervening stroma. There are variations in the quality of the epithelium. It may sometimes appear mucin-depleted, reactive, with larger nuclei, prominent nucleoli and mitotic figures. In other instances, the epithelium appears mature, and cytoplasmic mucin is preserved. Intestinal metaplasia to varying degrees is also possible, although less common than in hyperplastic polyps. Although there may be a slight lymphoplasmacytic infiltration in the lamina propria, smooth muscle hypertrophy is usually absent unless there is concurrent bile reflux [5].

Although gastric hyperplastic polyps are considered benign neoplasms of the stomach mucosa, there is a possibility that they could become malignant. While prior studies had reported lower rates of dysplastic changes, histological analysis has indicated intestinal metaplasia and dysplastic features in a significant proportion of up to 19% of resected Gastric Hyperplastic Polyp (GHP) underscoring their substantial risk for neoplastic progression [9]. Furthermore, studies have demonstrated that eradicating *Helicobacter pylori* reduced the likelihood of gastric hyperplastic polyps recurring after endoscopic excision, providing evidence of the benefits of *Helicobacter pylori* eradication in patients with gastric hyperplastic polyps post-endoscopic resection [10].

CONCLUSION(S)

This instance of polypoid foveolar hyperplasia and gastric hyperplastic polyp emphasises the significance of careful histological evaluation. While considered common, it is essential to diagnose these entities that have the potential to significantly improve a patient's quality of life. Furthermore, since malignant transformation affects gastric hyperplastic polyps but not foveolar polypoid hyperplasia, it is imperative to distinguish between a gastric hyperplastic polyp and a polypoid foveolar hyperplasia. Therefore, histopathology serves as an essential component of their therapy, serving as a means of ruling out malignancy.

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PLAGIARISM CHECKING METHODS: [Jain H et al.]
- Plagiarism X-checker: Feb 03, 2024
- Manual Googling: Mar 26, 2024
- iThenticate Software: Apr 19, 2024 (9%)

ETYMOLOGY: Author Origin

EMENDATIONS: 6

AUTHOR DECLARATION:
- Financial or Other Competing Interests: None
- Was informed consent obtained from the subjects involved in the study? No
- For any images presented appropriate consent has been obtained from the subjects. No

Date of Submission: **Feb 02, 2024**
Date of Peer Review: **Mar 20, 2024**
Date of Acceptance: **Apr 20, 2024**
Date of Publishing: **Jul 01, 2024**