



# An Intramucosal Mucinous Adenocarcinoma Arising From A Gastric Hyperplastic Polyp

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## Abstract

Hyperplastic polyps constitute 75% of all gastric polyps. Most of them are innocent lesions and are smaller than 1 cm. They develop as a consequence of an exaggerated mucosal response to tissue injury and inflammation on the basis of chronic gastritis. A 69-year-old male was admitted to the Gastroenterology Clinic with complaints of abdominal pain and vomiting. Gastroscopy showed a polyp that was 2 cm in its greatest dimension. A histopathologic examination of this lesion revealed adenocarcinoma infiltration in the mucosa. Subtotal gastrectomy was performed. Macroscopically, a polyp with a stalk was seen in the antrum; microscopically, it was consisted with a hyperplastic polyp. In the polyp, a few foci of infiltrative glands embedded in the mucinous matrix were noticed in the lamina propria, and the patient was diagnosed as intramucosal mucinous adenocarcinoma arising from a gastric hyperplastic polyp. Because of the risk of carcinoma development, which is rare, all hyperplastic polyps should be completely excised, and a histopathologic examination should be carefully performed.

**Keywords:** Hyperplastic polyp, stomach, mucinous adenocarcinoma

## Introduction

Hyperplastic polyps are lesions that are mostly smaller than 1 cm; they are *sessile* and innocuous. They develop as a result of tissue damage and an exaggerated mucosal regeneration. Intestinal metaplasia and dysplasia at a rate of 1–20% may be seen in hyperplastic polyps. However, the risk of carcinoma development is less than 2%. The risk increases in multiple polyps (1). Here we present a case of an intramucosal mucinous adenocarcinoma that developed in a solitary hyperplastic polyp localized in the stomach antrum. There were extensive atrophic gastritis, intestinal metaplasia, and, interestingly, gastritis cystica profunda areas in the non-tumorous stomach.

## Case Report

A 69-year-old male was admitted to a gastroenterology clinic with complaints of abdominal pain and vomiting. Laboratory test results were within normal limits, and there was no significant feature on performing abdominal ultrasonography. A polypoid mass with a diameter of 2 cm was observed in the antrum small curvature on gastroscopy, which was performed when a wall thickening was detected in the antrum by abdominal CT. An adenocarcinoma showing mucin production in biopsied superficial mucosal tissues was detected, and subtotal gastrectomy was performed in the patient. A pedunculated polyp (2×1 cm) in the antrum small curvature and flattening of pili in other stomach areas were observed on performing macroscopic examinations. In the histopathologic examination of the polyp, intestinal metaplasia areas, hyperplasia and cystic dilatation in the gastric foveolae and smooth muscle bundles and inflammation in the lamina propria were detected; morphological findings were compatible with hyperplastic polyps (Figure 1). Small infiltrative glandular structures within the mucinous stroma were detected in the several small foci in the polyp (Figure 2). Signet ring cells also attracted attention partly in the infiltration. The mucinous material in the background histologically showed an Alcian blue (+) staining (Figure 3). On conducting an immunohistochemical study, MUC 2 (+), MUC5AC focal (+), and MUC 6 (-) were found in tumor cells and MUC5AC (+), MUC-2 (-), and MUC 6 were found in non-tumor polyp areas. The Ki 67 proliferation index was significantly high in the tumor area (Figure 5). The patient was diagnosed with an intramucosal mucinous adenocarcinoma arising from the hyperplastic polyp. Extensive intestinal metaplasia and atrophic gastritis were detected in the non-tumor stomach. In addition, small cystic glands seen in many non-tumoral areas in the submucosa were considered as gastritis cystica profunda (Figure 6). No angiolymphatic invasion and metastases were detected in a total of 10 lymph nodes around the small and large curvature. This case has been presented with the consent of the patient.

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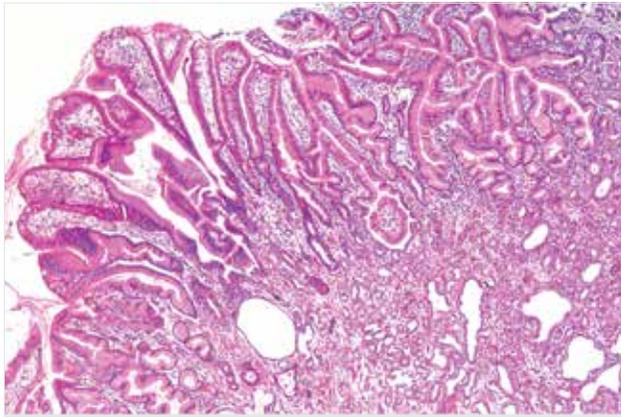
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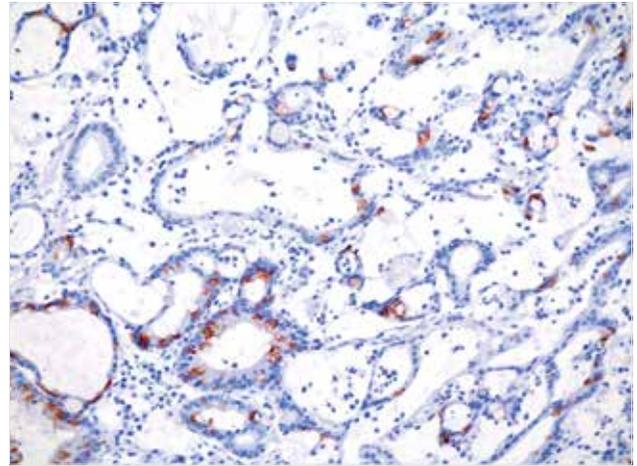
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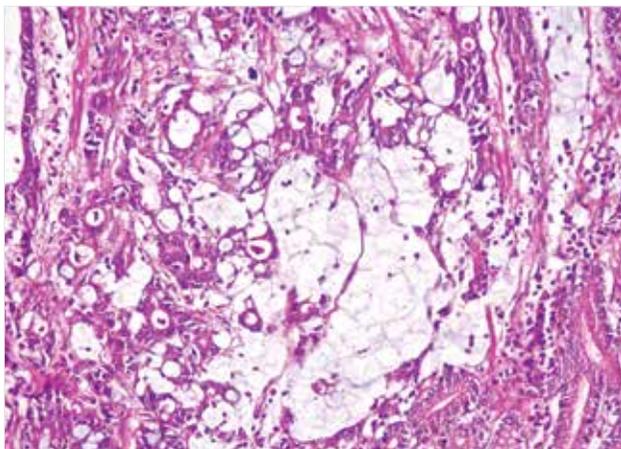
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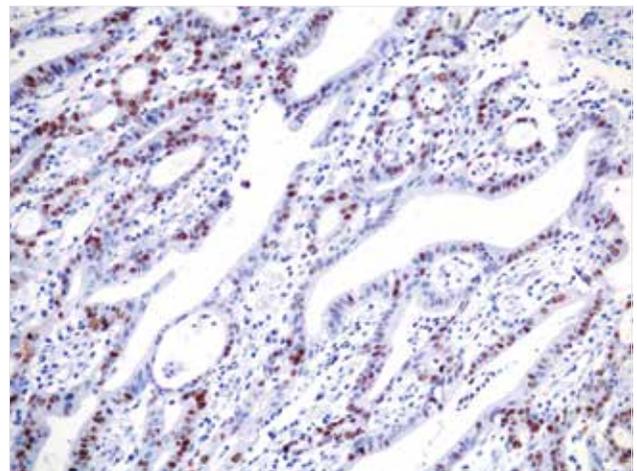
**Figure 1.** Hyperplastic polyp characterized by hyperplasia and cystic dilatation in gastric foveolae and by smooth muscle bundles in the lamina propria. HE  $\times 40$



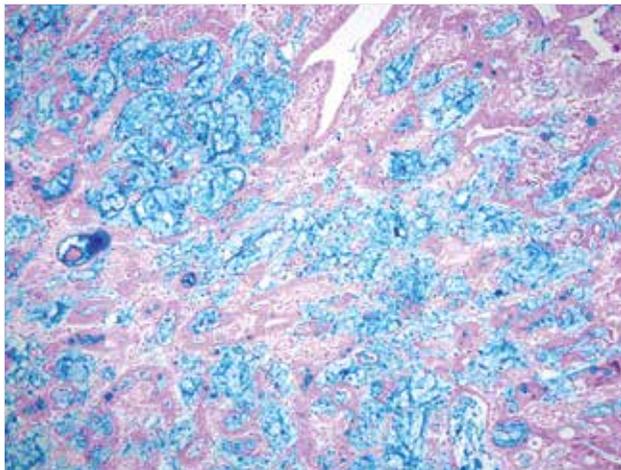
**Figure 4.** MUC 2 positivity in the tumor. HE  $\times 100$



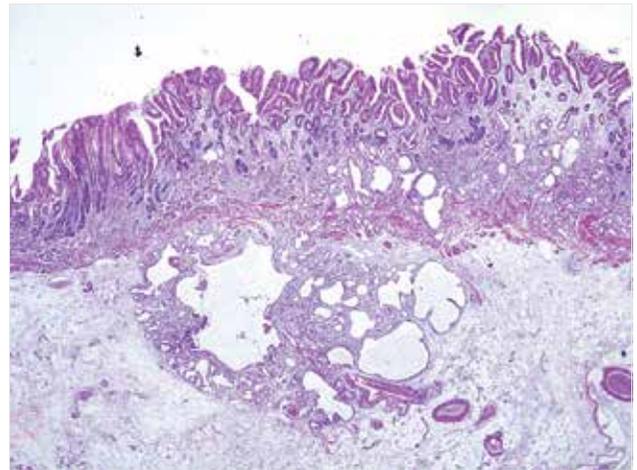
**Figure 2.** Tumor infiltration forming cribriform structures within the mucinous stroma. HE  $\times 200$



**Figure 5.** High Ki 67 proliferation index in the tumor. HE  $\times 200$



**Figure 3.** Alcian blue positivity in the mucinous material in the background. HE  $\times 100$



**Figure 6.** Gastritis cystica profunda areas in the non-tumor stomach tissue. HE  $\times 40$

## Discussion

Hyperplastic polyps constitute approximately 75% of all gastric polyps. They are mostly seen in 6-7 decades in both genders at equal frequency (1) and are usually asymptomatic. Those that are large-sized and localized in the gastroesophageal junction or pylorus can lead to obstruction (1). Most of these polyps develop as a result of tissue damage

and an exaggerated mucosal response to inflammation on the basis of chronic gastritis. *Helicobacter pylori* gastritis and chemical gastritis that develop secondary to bile reflux or due to Billroth II resections are associated with the development of hyperplastic polyps (1).

Intestinal metaplasia and dysplasia at a rate of 1–20% may be seen in hyperplastic polyps. However, the risk of carcinoma develop-

ment is less than 2%. The risk of dysplasia in polyps larger than 2 cm and carcinoma development in multiple polyps increases. The risk of dysplasia and carcinoma development is higher over 50 years of age (1). With the more frequent application of endoscopic polypectomy and mucosal resection, the incidence of dysplasia and carcinoma in hyperplastic polyps has also increased (2). Yao et al. (3) have indicated that the P53 mutation plays a role in malignant transformation in hyperplastic polyps. It has been suggested that the mucin profile of a carcinoma arising from a hyperplastic polyp is a gastric type in most cases (3). In our case, the intestinal phenotype is at the forefront in the tumor because there are MUC-2 (+) and MUC5AC focal (+) in tumoral areas and MUC5AC (+) and MUC-2 (-) in non-tumor polyp areas.

The development of a well- and moderately differentiated adenocarcinoma has mostly been reported on the basis of a hyperplastic polyp (2, 4, 5, 6). There have been few cases on a signet ring cell carcinoma and poorly differentiated adenocarcinoma arising from a hyperplastic polyp (7, 8). However, no cases on a mucinous adenocarcinoma arising from a hyperplastic polyp have been encountered in the English literature.

Gastritis cystica profunda is a lesion that is mostly seen in gastroenterostomy anastomosis regions, displaced in the gastric submucosa, hyperplastic, and composed of cystic dilated gastric glands. Chronic inflammation and ischemia are considered to be responsible for the pathogenesis. It may rarely develop also during chronic gastritis without any history of gastric surgery. Although it is a benign lesion, it is argued that it may carry malignant potential (9). The fact that the etiopathogenesis resembles hyperplastic polyps may explain the presence of two lesions in our case.

The risk of carcinoma in hyperplastic polyps increases with size. However, carcinoma cases that developed in hyperplastic polyps smaller than 2 cm in diameter, even in millimeters, have been reported in the literature (7, 10).

## Conclusion

Hyperplastic polyps need to be completely removed, and the entire polyp should be carefully examined histopathologically due to the risk of malignancy development, which is low.

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**Peer-review:** Externally peer-reviewed.

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