

Clinicopathological characteristics of gastric polyps in an *Helicobacter pylori*-endemic region: a real-world cohort study

Başak Çakal*, Yunus Halil Polat

Department of Gastroenterology, Ankara Training and Research Hospital, University of Health Sciences, Ankara, Türkiye

Cite this article as: Çakal B, Polat YH. Clinicopathological characteristics of gastric polyps in an *Helicobacter pylori*-endemic region: a real-world cohort study. *Intercont J Int Med.* 2026;4(4):91-95. doi:10.51271/ICJIM-0084

Received: 23.09.2026

Accepted: 23.10.2026

Published: 28.10.2026

ABSTRACT

Aims: Gastric polyps represent a heterogeneous group of mucosal lesions influenced by inflammatory, environmental, and pharmacologic factors. The distribution of polyp subtypes varies geographically, particularly in regions with high *Helicobacter pylori* (Hp) prevalence, where inflammation-driven lesions predominate. This study aimed to evaluate the clinicopathological characteristics of gastric polyps and their association with Hp infection and proton pump inhibitor (PPI) use in a real-world cohort.

Methods: In this retrospective cross-sectional study, 8,659 patients who underwent esophagogastroduodenoscopy (EGD) between January 2023 and January 2024 were screened, and 109 patients with histopathologically confirmed gastric polyps were included. Demographic features, endoscopic findings, histopathological subtypes, Hp status, background mucosal pathology, and PPI use were analyzed.

Results: The overall prevalence of gastric polyps was 1.25%. The mean age was 61.4±12.2 years, with a female predominance (73.4%). Hyperplastic polyps were the most common subtype (71.6%), followed by adenomatous (14.7%) and fundic gland polyps (6.4%). Hp positivity was significantly associated with hyperplastic polyps ($p=0.011$), while adenomatous polyps were significantly associated with chronic active gastritis (CAG) ($p=0.016$). All fundic gland polyps occurred in patients with long-term PPI use and were strongly associated with Hp negativity ($p<0.001$). No fundic gland polyps were observed in patients with concurrent Hp infection or chronic atrophic gastritis.

Conclusion: Gastric polyp development reflects the underlying gastric microenvironment shaped by inflammatory and pharmacologic factors. In Hp-endemic regions, hyperplastic polyps predominate and are strongly associated with Hp, whereas fundic gland polyps constitute a distinct entity linked to long-term PPI use and a lower prevalence of Hp infection.

Keywords: *Helicobacter pylori*, polyps, stomach, proton pump inhibitors

INTRODUCTION

Proliferative lesions originating from the gastric mucosa or submucosa and projecting into the lumen are defined as gastric polyps.^{1,2} Gastric polyps are detected in approximately 0.8–6% of upper gastrointestinal endoscopic procedures and are frequently identified incidentally during gastroscopy.^{1,3} Although most polyps are asymptomatic, they may present with gastrointestinal bleeding, anemia, abdominal discomfort, or, rarely, gastric outlet obstruction.^{4,5}

The histopathological distribution of gastric polyps varies geographically and appears to be influenced by environmental factors, *Helicobacter pylori* (Hp) prevalence, and proton pump inhibitor (PPI) use.^{1,2,4,6,7} Hyperplastic polyps and adenomatous polyps constitute the majority of cases, whereas fundic gland polyps are more commonly observed in Western populations with lower Hp prevalence and widespread PPI use.^{1,6,7} In contrast, hyperplastic and adenomatous/neoplastic gastric polyps appear to be relatively more prevalent in regions with a higher prevalence of Hp infection.⁸

Hp plays a central role in gastric carcinogenesis through chronic inflammation, mucosal atrophy, intestinal metaplasia, and

epithelial proliferation. Persistent Hp-induced gastritis leads to alterations in gastric epithelial turnover and may contribute to the development of hyperplastic and adenomatous polyps.⁹ Moreover, Hp eradication has been associated with regression of certain hyperplastic polyps, suggesting a potential causal relationship.¹⁰

Despite numerous epidemiological studies, data regarding the clinicopathological characteristics of gastric polyps in Hp-prevalent regions remain limited, and the interaction between Hp infection and specific polyp subtypes has not been fully elucidated. Therefore, the present study aimed to evaluate the demographic features, anatomical distribution, histopathological characteristics of gastric polyps, and their association with Hp infection in our population.

METHODS

Ethics

This study was approved by the Ethics Committee of Ankara Training and Researches Hospital (Date: 26.03.2025, Decision

*Corresponding Author: Başak Çakal, basakcakal@hotmail.com



No: E-25-456). All procedures were carried out in accordance with the ethical rules and the principles of the Declaration of Helsinki.

Study Population

This retrospective cross-sectional study was conducted at the Department of Gastroenterology, Ankara Training and Research Hospital, between January 2023 and January 2024. Due to the retrospective design and use of anonymized data, the requirement for informed consent for study participation was waived.

During the study period, 8,659 patients who underwent esophagogastroduodenoscopy (EGD) for various clinical indications were retrospectively screened. Among these, 109 patients with histopathologically confirmed gastric polyps were included in the study. Lesions considered as polyp precursor changes, such as isolated foveolar hyperplasia without true polyp formation, were excluded. Patients with incomplete medical records were also excluded.

Demographic characteristics, presenting symptoms, history of PPI use, Hp status, presence of chronic active gastritis (CAG), atrophic gastritis, intestinal metaplasia, polyp localization, number, diameter, and histopathological subtype were recorded from electronic medical records. PPI use was defined as regular use of a PPI for at least 1 year prior to endoscopy.

Endoscopic Procedure

All endoscopic procedures were performed after a minimum fasting period of 8 hours. Patients were monitored throughout the procedure, which was carried out under local oropharyngeal anesthesia using 10% lidocaine spray with supplemental oxygen support.

All detected gastric polyps were removed endoscopically. Depending on the size and morphology of the lesion, polypectomy was performed using biopsy forceps or snare excision with electrocautery. In cases of multiple polyps, the diameter of the largest lesion was recorded for analysis. No procedure-related complications were observed.

To assess the background gastric mucosa and evaluate the presence of Hp infection, multiple biopsies were obtained from at least one site each from the antrum and corpus in all patients.

Histopathological Evaluation

All biopsy and polypectomy specimens were fixed in formalin, embedded in paraffin, and evaluated by experienced gastrointestinal pathologists. Histopathological classification of gastric polyps was based on routine pathological evaluation using standard histologic criteria and categorized as fundic gland polyps, hyperplastic polyps, and adenomatous polyps.¹¹

Gastric mucosal biopsies were evaluated in accordance with the Updated Sydney System.¹² Chronic gastritis was defined by mononuclear inflammatory cell infiltration in the lamina propria. CAG was defined by the presence of neutrophilic infiltration in addition to chronic inflammatory changes. Atrophic gastritis was diagnosed based on the loss of appropriate gastric glands, with or without accompanying intestinal metaplasia. Intestinal metaplasia was identified by the presence of goblet cells replacing the native gastric

epithelium. Hp infection was assessed histopathologically in gastric mucosal biopsies obtained from both the antrum and corpus.

Statistical Analysis

The data analyses were performed using Statistical Package for the Social Sciences v. 21 software (IBM SPSS for Windows version 21, IBM Corporation, Armonk, NY, USA). All continuous variables with normal distribution were defined as mean $\pm$ standard deviation, and non-normally distributed variables as median (min-max) values. Categorical variables were stated as number (n) and percentage (%). The comparisons of independent continuous variables between hyperplastic polyp and other polyps were performed using the Independent Samples t-test. The Mann-Whitney U test was applied to compare independent continuous variables between adenomatous polyps, fundic polyps, and other polyps. A binary logistic regression model including Hp status, gender, and the Hp status $\times$ gender interaction term was performed to assess whether the association between Hp infection and hyperplastic polyps differed by gender. The relationships between categorical variables were analyzed using Chi-square analysis or Fisher's Exact test. A value of $p < 0.05$ was considered statistically significant.

RESULTS

Gastric polyps were identified in 109 of 8,659 upper gastrointestinal endoscopies performed during the one-year study period, yielding an overall prevalence of 1.25%. The study cohort comprised 80 (73.4%) females and 29 (26.6%) males, with a mean age of 61.4 ± 12.2 years. The mean polyp diameter was 9.4 ± 6.6 mm (range, 2-50 mm). Regarding anatomical localization, polyps were most frequently found in the antrum ($n=41$, 37.6%), followed by the corpus ($n=33$, 30.3%), cardia ($n=27$, 24.8%), and fundus ($n=8$, 7.3%). When the patients were separated into two groups as <60 years and ≥ 60 years, the highest frequency of polyps was in the group aged ≥ 60 years (64 polyps, 58.7%). The most common presenting complaint was dyspepsia ($n=66$, 60.5%), followed by abdominal pain ($n=18$, 16.5%), anemia ($n=14$, 12.8%), and gastrointestinal bleeding ($n=5$, 4.5%). The demographic and baseline characteristics of patients with gastric polyps are summarized in [Table 1](#).

In gender-based analyses, no statistically significant difference was observed between female and male patients in terms of polyp localization or histological subtype. Polyp localization did not differ significantly by gender ($p=0.652$). Hyperplastic polyps were observed in 75.0% of females and 62.1% of males ($p=0.186$). Adenomatous polyps were detected in 12.5% of females and 24.1% of males ($p=0.148$), while fundic gland polyps were detected in 7.5% of females and 3.4% of males ($p=0.672$). A binary logistic regression model including Hp status, gender, and the Hp status $\times$ gender interaction term was performed to evaluate whether the association between Hp infection and hyperplastic polyps differed by gender. The interaction term was not statistically significant ($p=0.659$), indicating no evidence that gender modified the association between Hp infection and hyperplastic polyp occurrence.

Histopathologically, hyperplastic polyps were the most common subtype (71.6%), followed by adenomatous polyps

Table 1. Demographic characteristics of gastric polyps

Polyp type	Age	Gender (F/M)	Size (mm)	Cardia	Fundus	Corpus	Antrum	Multiple	Polypectomy
Hyperplastic (n=78)	61.3±12.2	60/18	9.1±6.8	21 (26.9%)	5 (6.4%)	21 (26.9%)	31 (39.7%)	14 (17.9%)	59 (75.6%)
Adenomatous (n=16)	63.9±10.8	10/6	11.2±6.2	3 (18.8%)	-	5 (31.3%)	8 (50%)	2 (12.5%)	16 (100%)
Fundic (n=7)	52.1±12.0	6/1	6.9±2.6	1 (14.3%)	3 (42.9%)	3 (42.9%)	-	2 (28.6%)	5 (71.4%)
NET (n=4)	58.8±10.3	2/2	8.3±3.9	-	-	3 (75%)	1 (25%)	-	1* (25%)
Adenocarcinoma (n=2)	78-81	-/2	10-30	2 (100%)	-	-	-	1 (50%)	**
GIST (n=1)	78	1/-	10	-	-	1 (100%)	-	-	***
Hamartomatous (n=1)	61	1/-	10	-	-	-	1 (100%)	1 (100%)	1 (100%)

F: Female, M: Male, NET: Neuroendocrine tumor, GIST: Gastrointestinal stromal tumor *While gastric surgical procedure was performed in one patient after polypectomy, direct gastric surgical procedure was performed in three patients without polypectomy. ** Gastric surgical procedure was performed without polypectomy in two patients. *** Gastric surgical procedure was performed without polypectomy in one patient.

(14.7%) and fundic gland polyps (6.4%). A single polyp was detected in 89 patients (81.7%), while 20 patients (18.3%) had multiple (≥ 2) polyps. The characteristics of the cases with a single polyp and multiple polyps are given in [Table 2](#).

Table 2. Comparison of data of cases with single or multiple polyps

	Single (n=89)	Multiple (n=20)	p
Age	63 (27-85)	64.5 (45-85)	0.184
Gender (F/M)	65 (73%) / 24 (27%)	15 (75%) / 5 (25%)	0.857
Localization	33 (37.1%) Antrum 27 (30.3%) Corpus 23 (25.8%) Cardia 6 (6.7%) Fundus	8 (40%) Antrum 6 (30%) Corpus 4 (20%) Cardia 2 (10%) Fundus	0.919
Chronic active gastritis	30 (33.7%)	9 (45%)	0.341
Intestinal metaplasia	25 (28.1%)	3 (15%)	0.226
Hp presence	41 (46.1%)	8 (40%)	0.622

F: Female, M: Male, Hp: *Helicobacter pylori*

Although not statistically significant, multiple polyps were more frequent among patients with hyperplastic polyps (n=14, 17.9%). While no statistically significant difference was observed in age distribution between hyperplastic and adenomatous polyps and other polyp types, fundic gland polyps were significantly more prevalent among younger patients compared to other polyp subtypes (p=0.048).

Histopathological background findings revealed chronic gastritis in 55 patients (50.5%), CAG in 39 patients (35.8%), and chronic atrophic gastritis in 14 patients (12.8%). While all adenomatous polyps underwent polypectomy, 19 hyperplastic (24.4%) and 2 (28.6%) fundic polyps were followed up due to endoscopic biopsy results and small dimensions (<5 mm). Gastric surgical procedures were performed in two

adenocarcinomas, one gastrointestinal stromal tumor, and four neuroendocrine tumor cases.

A history of Hp infection was present in 49 patients (45%), while 25 patients (22.9%) had a history of PPI use for more than one year. The most common types of polyps and their association with Hp are shown in [Table 3](#). Hp positivity was significantly more frequent in patients with hyperplastic polyps compared to other histological subtypes (p=0.011). In contrast, the majority of adenomatous polyps (n=10, 62.5%) were detected in patients with CAG, demonstrating a statistically significant association (p=0.016). All patients with fundic gland polyps had a history of long-term PPI use. A strong association was observed between PPI use and fundic gland polyp development (p<0.001). Notably, none of the patients with fundic gland polyps had concomitant CAG (p=0.048) or Hp infection (p=0.016).

DISCUSSION

Gastric polyps represent a heterogeneous group of lesions arising from overlapping pathogenic pathways, including mucosal inflammation, atrophy and metaplasia, as well as pharmacologically induced epithelial changes. In this real-world cohort, our findings indicate that gastric polyps are not isolated entities but rather reflections of the underlying gastric microenvironment. Within this framework, Hp plays a central role by promoting epithelial proliferation, particularly in hyperplastic polyp formation, whereas CAG was more closely associated with adenomatous transformation in our cohort, and PPI-induced hypergastrinemia contributes to the development of fundic gland polyps in Hp-negative settings. Overall, our findings suggest that gastric polyps should be interpreted within a multidimensional pathogenic framework integrating inflammatory, structural, and iatrogenic factors.

Table 3. Characteristics of patients by type of gastric polyp

	Hyperplastic polyp (n=78)	Other type (n=31)	p	Adenomatous polyp (n=16)	Other type (n=93)	p	Fundic polyp (n=7)	Other type (n=102)	p
Age (year)	61.3 ± 12.2	61.9 ± 12.3	0.800	64 (42-83)	63 (27-85)	0.396	53 (37-69)	63 (27-85)	0.048
Gender (F)	60 (76.9%)	20 (64.5%)	0.186	10 (62.5%)	70 (75.3%)	0.359	6 (85.7%)	74 (72.5%)	0.672
Multiple	14 (17.9%)	6 (19.4%)	0.864	2 (12.5%)	18 (19.4%)	0.731	2 (28.6%)	18 (17.6%)	0.610
Size (mm)	9.0±6.8	10.3±6.2	0.376	10 (3-25)	8 (2-50)	0.119	6 (3-10)	9 (2-50)	0.293
Chronic active gastritis	29 (37.2%)	10 (32.3%)	0.629	10 (62.5%)	29 (31.2%)	0.016	0 (0%)	39 (38.2%)	0.048
Intestinal metaplasia	18 (23.1%)	10 (32.3%)	0.322	6 (37.5%)	22 (23.7%)	0.351	1 (14.3%)	27 (26.5%)	0.675
Hp presence	41 (52.6%)	8 (25.8%)	0.011	6 (37.5%)	43 (46.2%)	0.516	0	49 (48%)	0.016

Data are presented as mean±standard deviation, median (minimum–maximum), or \%(n) (%), as appropriate. p: P-value, F: Female, Hp: *Helicobacter pylori*

In this retrospective cohort of patients undergoing EGD, gastric polyps were identified in 1.25% of cases, a prevalence consistent with previously reported rates of 0.8%–7.7%.^{11,13} Hyperplastic polyps were the predominant subtype, followed by adenomatous and fundic gland polyps.^{8,14} This distribution aligns with data from regions with high Hp prevalence, where inflammation-driven mucosal alterations predominate. The high frequency of hyperplastic polyps in our cohort likely reflects sustained mucosal injury and regenerative activity associated with chronic gastritis. These lesions are generally considered reactive rather than truly neoplastic and are thought to arise in the setting of chronic mucosal injury, reflecting exaggerated foveolar epithelial repair, regeneration, and hyperproliferation.¹⁵ Consistent with this mechanism, we observed a significant association between Hp infection and hyperplastic polyps. Hp colonization induces CAG characterized by neutrophilic infiltration, oxidative stress, and sustained release of proinflammatory cytokines, including IL-1 β and TNF- α , which disrupt epithelial homeostasis and promote compensatory proliferation.^{16,17} Over time, this process leads to architectural distortion, foveolar hyperplasia, and polyp formation. Importantly, evidence from meta-analyses demonstrates regression of hyperplastic polyps following Hp eradication, supporting a causal relationship rather than a mere association.^{10,18} Accordingly, our findings further support current recommendations advocating Hp testing and eradication in patients with hyperplastic gastric polyps.

In our cohort, adenomatous polyps were significantly associated with CAG, whereas no significant associations were observed with intestinal metaplasia or Hp infection. Although gastric adenomas are traditionally linked to atrophic and metaplastic changes, our findings suggest that, in real-world settings, active inflammatory changes may also accompany these lesions.^{8,19} CAG is characterized by persistent epithelial injury, neutrophilic infiltration, and increased cellular turnover, which may promote a microenvironment conducive to dysplastic transformation.^{15,20} Ongoing epithelial injury and regeneration may promote cell proliferation and oxidative DNA damage within an inflammatory mucosal microenvironment, thereby facilitating neoplastic progression. In addition, inflammatory mediators and reactive oxygen species generated in inflamed gastric mucosa may further contribute to DNA damage and malignant transformation.²⁰ However, given the cross-sectional design of our study, this association should be interpreted cautiously and does not imply a direct causal relationship.

Fundic gland polyps were observed exclusively in patients with long-term PPI use, demonstrating a strong association, while none were associated with Hp infection. This distinct distribution suggests a pathophysiological mechanism fundamentally different from inflammation-driven polyp formation. Chronic acid suppression induced by PPI therapy leads to sustained hypergastrinemia, which exerts trophic effects on the oxyntic mucosa and is associated with characteristic histologic changes, including parietal cell protrusion and cystic dilatation of fundic glands.²¹ The absence of Hp infection in patients with fundic gland polyps is consistent with prior studies reporting an inverse relationship between Hp-associated gastritis and the development of these lesions.^{22,23} Hp-related gastritis, particularly when

accompanied by glandular atrophy, may reduce functional parietal cell mass and thereby attenuate gastrin-mediated trophic effects on the fundic mucosa, potentially limiting polyp formation.²⁴ These findings are in line with large observational and meta-analytic studies demonstrating an increased prevalence of fundic gland polyps with prolonged PPI exposure.^{23,25,26} Clinically, this underscores the importance of periodic reassessment of long-term PPI therapy, particularly in patients without clear indications.

A key strength of our study is its real-world design, capturing a heterogeneous patient population with varying degrees of inflammation and medication exposure. Unlike controlled trials or highly selected cohorts, our findings reflect the complex interplay of multiple factors simultaneously influencing gastric mucosal pathology. This heterogeneity likely explains the variability in polyp distribution and behavior reported across studies. A female predominance was observed in our cohort. However, gender-based analyses did not demonstrate statistically significant differences in polyp localization or histological subtype. In addition, the Hp status $\times$ gender interaction term was not statistically significant in the logistic regression model, suggesting that the association between Hp infection and hyperplastic polyp occurrence did not differ significantly between female and male patients. Therefore, the observed female predominance should be interpreted cautiously and may reflect selection or referral bias related to the retrospective, endoscopy-based, single-center design rather than a definite biological predisposition.

Limitations

There are several limitations to the present study. First, this was a retrospective, single-center study with a relatively small sample size; therefore, our findings should be interpreted as real-world regional observations rather than a global assessment. In addition, the number of male patients and some subgroup sizes were limited, restricting the reliability of gender-stratified analyses. These findings should therefore be confirmed in larger multicenter cohorts with prespecified gender-based analyses. Second, the number of patients with long-term PPI use was limited, and the exact duration of PPI exposure could not be determined. Finally, long-term follow-up data were not available; therefore, we could not evaluate whether gastric polyps regressed after Hp eradication.

CONCLUSION

As a result, gastric polyps represent a heterogeneous group of lesions whose development is strongly influenced by the balance between inflammatory (Hp-driven) and pharmacologic (PPI-induced) factors. In Hp-endemic populations, hyperplastic polyps predominate and are closely linked to chronic gastritis, whereas fundic gland polyps, on the other hand, represent a distinct entity associated with long-term acid suppression and Hp negativity. These findings emphasize that gastric polyps should not be evaluated in isolation but in the context of the underlying gastric mucosal environment, and that management strategies should be guided by the combined assessment of histopathological features, Hp status, and medication exposure.

ETHICAL DECLARATIONS

Ethics Committee Approval

This study was approved by the Ethics Committee of Ankara Training and Researches Hospital (Date: 26.03.2025, Decision No: E-25-456).

Informed Consent

This retrospective study used pre-existing anonymized patient data. No additional intervention was performed, and there was no direct patient contact. The study was approved by the Ethics Committee, and the requirement for written informed consent was waived by the ethics committee.

Peer Review Process

This manuscript was subject to external peer review.

Conflict of Interest

The authors declare no conflicts of interest related to this study.

Financial Disclosure

The authors received no financial support for the conduct or publication of this research.

Author Contributions

Concept: BC; Design: BC; Control: BC; Data Collection and/or Processing: BC, YHP; Analysis and/or Interpretation: BC, YHP; Literature Review: BC; Article Writing: BC; Critical Review: BC.

REFERENCES

- Morais DJ, Yamanaka A, Zeitune JMR, Andreollo NA. Gastric polyps: a retrospective analysis of 26,000 digestive endoscopies. *Arq Gastroenterol.* 2007;44(1):14-17. doi:10.1590/s0004-28032007000100004
- Carmack SW, Genta RM, Schuler CM, Saboorian MH. The current spectrum of gastric polyps: a 1-year national study of over 120,000 patients. *Am J Gastroenterol.* 2009;104(6):1524-1532. doi:10.1038/ajg.2009.139
- Çiyiltepe H, Çetin DA, Gündeş E, et al. Endoscopic and histopathological features of the upper gastrointestinal system polyps: evaluation of 12,563 procedures. *Turk J Surg.* 2019;35(2):98-104. doi:10.5578/turksurg.4155
- Sonnenberg A, Genta RM. Prevalence of benign gastric polyps in a large pathology database. *Dig Liver Dis.* 2015;47(2):164-169. doi:10.1016/j.dld.2014.10.004
- Costa D, Ramai D, Tringali A. Novel classification of gastric polyps: the good, the bad and the ugly. *World J Gastroenterol.* 2024;30(31):3640-3653. doi:10.3748/wjg.v30.i31.3640
- Archimandritis A, Spiliadis C, Tzivras M, et al. Gastric epithelial polyps: a retrospective endoscopic study of 12974 symptomatic patients. *Ital J Gastroenterol.* 1996;28(7):387-390
- Jalving M, Koornstra JJ, Wesseling J, Boezen HM, de Jong S, Kleibeuker JH. Increased risk of fundic gland polyps during long-term proton pump inhibitor therapy. *Aliment Pharmacol Ther.* 2006;24(9):1341-1348. doi:10.1111/j.1365-2036.2006.03127.x
- Reyes-Placencia D, Cantú-Germano E, Latorre G, Espino A, Fernández-Esparrach G, Moreira L. Gastric epithelial polyps: current diagnosis, management, and endoscopic frontiers. *Cancers.* 2024;16(22):3771. doi:10.3390/cancers16223771
- Markowski AR, Markowska A, Guzinska-Ustymowicz K. Pathophysiological and clinical aspects of gastric hyperplastic polyps. *World J Gastroenterol.* 2016;22(40):8883-8891. doi:10.3748/wjg.v22.i40.8883
- Nam SY, Lee SW, Jeon SW, Kwon YH, Lee HS. *Helicobacter pylori* eradication regressed gastric hyperplastic polyp: a randomized controlled trial. *Dig Dis Sci.* 2020;65(12):3652-3659. doi:10.1007/s10620-020-06065-0
- Buchner AM, Huang RJ, Lauwers GY, El-Serag HB. AGA clinical practice update on management of gastric polyps: expert review. *clin gastroenterol hepatol off clin pract. Clin Gastroenterol Hepatol.* 2026;24(4):893-905. doi:10.1016/j.cgh.2026.01.007
- Dixon MF, Genta RM, Yardley JH, Correa P. Classification and grading of gastritis. The updated Sydney System. International workshop on the histopathology of gastritis, Houston 1994. *Am J Surg Pathol.* 1996;20(10):1161-1181. doi:10.1097/00000478-199610000-00001
- Cheesman AR, Greenwald DA, Shah SC. Current management of benign epithelial gastric polyps. *Curr Treat Options Gastroenterol.* 2017;15(4):676-690. doi:10.1007/s11938-017-0159-6
- Yacoub H, Bibani N, Sabbah M, et al. Gastric polyps: a 10-year analysis of 18,496 upper endoscopies. *BMC Gastroenterol.* 2022;22(1):70. doi:10.1186/s12876-022-02154-8
- Yang H, Yang W-J, Hu B. Gastric epithelial histology and precancerous conditions. *World J Gastrointest Oncol.* 2022;14(2):396-412. doi:10.4251/wjgo.v14.i2.396
- White JR, Winter JA, Robinson K. Differential inflammatory response to *Helicobacter pylori* infection: etiology and clinical outcomes. *J Inflamm Res.* 2015;8:137-147. doi:10.2147/JIR.S64888
- Han L, Shu X, Wang J. *Helicobacter pylori*-mediated oxidative stress and gastric diseases: a review. *Front Microbiol.* 2022;13:811258. doi:10.3389/fmicb.2022.811258
- Ouyang Y, Zhang W, Huang Y, et al. Effect of *Helicobacter pylori* eradication on hyperplastic gastric polyps: a systematic review and meta-analysis. *Helicobacter.* 2021;26(5):e12838. doi:10.1111/hel.12838
- Kövári B, Kim BH, Lauwers GY. The pathology of gastric and duodenal polyps: current concepts. *Histopathology.* 2021;78(1):106-124. doi:10.1111/his.14275
- Zhang S, Shen Y, Liu H, et al. Inflammatory microenvironment in gastric premalignant lesions: implication and application. *Front Immunol.* 2023;14:1297101. doi:10.3389/fimmu.2023.1297101
- Kim GH. Proton pump inhibitor-related gastric mucosal changes. *Gut Liver.* 2021;15(5):646-652. doi:10.5009/gnl20036
- Notsu T, Adachi K, Mishihiro T, Ishimura N, Ishihara S. Fundic gland polyp prevalence according to *Helicobacter pylori* infection status. *J Gastroenterol Hepatol.* 2020;35(7):1158-1162. doi:10.1111/jgh.14934
- Gao W, Huang Y, Lu S, Li C. The clinicopathological characteristics of gastric polyps and the relationship between fundic gland polyps, *Helicobacter pylori* infection, and proton pump inhibitors. *Ann Palliat Med.* 2021;10(2):2108-2114. doi:10.21037/apm-21-39
- Kale K, Renavikar PS, Amarapurkar AD. Immunological insights into gastric polyps: pathogenesis, inflammation and neoplastic potential. *Clin Transl Immunol.* 2026;15:e70092. doi:10.1002/cti2.70092
- Sawaid IO, Samson AO. Proton pump inhibitors and cancer risk: a comprehensive review of epidemiological and mechanistic evidence. *J Clin Med.* 2024;13(7):1970. doi:10.3390/jcm13071970
- Tran-Duy A, Spaetgens B, Hoes AW, de Wit NJ, Stehouwer CDA. Use of proton pump inhibitors and risks of fundic gland polyps and gastric cancer: systematic review and meta-analysis. *Clin Gastroenterol Hepatol.* 2016;14(12):1706-1719.e5. doi:10.1016/j.cgh.2016.05.018