CLINICAL PRACTICE UPDATES

AGA Clinical Practice Update on Management of Gastric Polyps: Expert Review

Anna M. Buchner,[1] Robert J. Huang,[2] Gregory Y. Lauwers,[3] and Hashem B. El-Serag[4]

[1]*Division of Gastroenterology and Hepatology, University of Pennsylvania, Philadelphia, Pennsylvania;* [2]*Division of Gastroenterology and Hepatology, Stanford University, Stanford, California;* [3]*Department of Pathology, H. Lee Moffitt Cancer Center and Research Institute, Tampa, Florida; and* [4]*Section of Gastroenterology and Hepatology, Department of Medicine, Baylor College of Medicine, Houston, Texas*

DESCRIPTION: **This Clinical Practice Update (CPU) expert review will advise clinicians on the diagnosis and management of gastric mucosal polyps. Gastric polyps are raised epithelial lesions of the gastric mucosa that can arise from various mucosal alterations and perturbations, including mucosal hyperplasia, adenoma, fundic gland proliferation, and enterochromaffin-like cell proliferation. Current guidance on the management of gastric polyps remains limited. This CPU provides a framework for understanding the natural history and epidemiology of gastric polyps and advises on best practices for the endoscopic detection and classification of gastric polyps, the endoscopic resection of gastric polyps, and endoscopic surveillance following resection. Because gastric polyps often occur within a field of altered gastric mucosa (eg, mucosal atrophy, pseudo-pyloric and intestinal metaplasia), we will advise on best practices for the sampling and surveillance of mucosal pathology giving rise to gastric polyps. This CPU is intended to complement other documents issued by the American Gastroenterological Association (AGA) Institute on gastric neoplastic and pre-neoplastic lesions, including the clinical practice guidelines on management of gastric intestinal metaplasia, as well as AGA CPUs on atrophic gastritis, high-quality upper endoscopy, and screening and surveillance of individuals at increased risk for gastric cancer.**

METHODS: **This expert review was commissioned and approved by the AGA Institute Clinical Practice Updates Committee (CPUC) and the AGA Governing Board to provide timely guidance on a topic of high clinical importance to the AGA membership, and underwent internal peer review by the CPUC and external peer review through standard procedures of *Clinical Gastroenterology and Hepatology*. These Best Practice Advice (BPA) statements were drawn from a review of the published literature and from expert opinion. Because systematic reviews were not performed, these BPA statements do not carry formal ratings regarding the quality of evidence or strength of the presented considerations.**

BEST PRACTICE ADVICE STATEMENTS

BPA 1: **Gastric polyps are frequently identified during upper endoscopy exams and include different histologic subtypes, such as fundic gland polyps (FGPs), gastric hyperplastic polyps (GHPs), hamartomatous polyps, gastric adenomas (GAs), pyloric gland adenomas, oxyntic gland adenomas, and gastric neuroendocrine tumors (G-NETs).**

BPA 2: **Clinicians should be aware that different types of gastric polyps may coexist in the same person.**

Abbreviations used in this paper: **AG, atrophic gastritis; AGA, American Gastroenterological Association; BLI, blue laser imaging; BPA, best practice advice; CI, confidence interval; CPU, clinical practice update; EGGIM, endoscopic grading of gastric intestinal metaplasia; EMR, endoscopic mucosal resection; ESD, endoscopic submucosal dissection; FAP, familial adenomatous polyposis; FGP, fundic gland polyp; G-NET, gastric neuroendocrine tumor; GA, gastric adenoma; GAPPS, gastric adenocarcinoma and proximal polyposis syndrome of the stomach; GHP, gastric hyperplastic polyp; GIM, gastric intestinal metaplasia; *H pylori*, *Helicobacter pylori*; HD-WLE, high-definition white-light endoscopy; HP, hyperplastic polyp; IEE, image-enhanced endoscopy; LCI, linked color imaging; LR, likelihood ratio; NBI, narrow-band imaging; P-CAB, potassium-competitive acid blocker; PPI, proton pump inhibitor; RCT, randomized controlled trial.**

Most current article

1542-3565/$36.00
https://doi.org/10.1016/j.cgh.2026.01.007

BPA 3: **Clinicians should be aware that different types of gastric polyps are associated with varying spectra of histopathologic abnormalities in the surrounding gastric mucosa, which may aid in their identification and diagnosis.**

BPA 4: **Systematic endoscopic examination of the polyps and the surrounding gastric mucosa is essential in assessing the underlying gastric mucosa pathology (eg, *Helicobacter pylori* gastritis, autoimmune gastritis, gastric intestinal metaplasia [GIM]) and determining subsequent management: biopsies of the polyps, biopsies of the surrounding mucosa, and resection of the polyps.**

BPA 5: **All patients with adenomatous or hyperplastic gastric polyps should be tested and treated if positive for *H pylori* infection.**

BPA 6: **Patients who are using proton pump inhibitors (PPIs) for valid reasons do not need to discontinue these medications in the presence of documented fundic gland hyperplasia-related gastric polyps.**

BPA 7: **Clinicians should be aware that different histological types of gastric polyps have unique/characteristic topographical features, endoscopic features, and size.**

BPA 8: **Endoscopic evaluation of patients with gastric polyps should include complete inspection with high-definition white-light and enhanced imaging, such as virtual chromoendoscopy. Endoscopists should recognize and photo-document the endoscopic features of gastric polyps as well as the surrounding gastric mucosal abnormalities.**

BPA 9: **Clinicians should be aware that endoscopic resection of the polyps includes traditional techniques (snare and biopsy forceps, mucosal resection) or endoscopic submucosal dissection.**

BPA 10: **In the presence of numerous gastric polyps of varied sizes, the largest polyps should be resected when possible, and the smaller polyps sampled or resected.**

BPA 11: **Suspected abnormalities in the surrounding mucosa, such as GIM or atrophic gastritis, should undergo targeted biopsies according to the existing protocols.**

BPA 12: **Surveillance plans in patients with gastric polyps should be formulated based on the histopathological type of the polyps and the surrounding gastric mucosa.**

BPA 13: **When a dysplastic lesion in the polyp is confirmed and resected completely, a follow-up surveillance endoscopy should be completed in 1 year for patients with low-grade dysplasia polyps and 6 months for patients with high-grade dysplasia polyps. If the polyp is biopsied or resection is incomplete, follow-up endoscopy is advised within 3 months for high-grade dysplasia and 6 months for low-grade dysplasia.**

BPA 14: **Endoscopic surveillance is advised in patients with gastric polyps when the histopathology of adjacent mucosa confirms GIM and/or atrophic gastritis.**

Keywords: Endoscopic Management; Gastric Polyps; Natural History; Risk Factors; Surveillance.

Gastric polyps can occur in the context of several heterogeneous conditions, including *Heliobactor pylori*-related gastritis, atrophic gastritis, autoimmune gastritis, and reactive gastropathy, as well as in normal mucosa and in several polyposis syndromes. Management of gastric polyps includes detection, characterization, biopsies, and/or resection, assessment of the entire gastric mucosa, and post-endoscopic treatment surveillance. Adequate evaluation of gastric polyps and adjacent mucosa is crucial to improve the detection and management of pre-neoplastic and neoplastic gastric conditions. The current clinical guidance for best practices in diagnosis and management remains limited.

This clinical practice update (CPU) focusing on gastric polyps complements the 2020 American Gastroenterological Association (AGA) clinical practice guidelines on the management of gastric intestinal metaplasia, the 2021 AGA CPU on atrophic gastritis and the 2024 AGA CPU on high-quality upper endoscopy, and the 2024 AGA CPU on screening and surveillance in

individuals at increased risk for gastric cancer in the United States.[1–4] The goal of this CPU is to assist and guide clinicians in managing patients with gastric polyps and to recognize the significance of evaluating adjacent gastric mucosa.

Epidemiology, Risk Factors, and Natural History of Gastric Polyps (BPA 1–3, 5–6)

BPA 1: Gastric polyps are frequently identified during upper endoscopy exams and include different histologic subtypes, such as fundic gland polyps (FGPs), gastric hyperplastic polyps (GHPs), hamartomatous polyps, gastric adenomas (GAs), pyloric gland adenomas, oxyntic gland adenoma, and gastric neuroendocrine tumors (G-NETs).

BPA 2: Clinicians should be aware that different types of polyps may coexist in the same person.

BPA 3: Clinicians should be aware that different types of gastric polyps are associated with varying histopathologic abnormalities in the surrounding gastric mucosa, which might aid in their identification and diagnosis.

BPA 5: All patients with adenomatous or hyperplastic gastric polyps should be tested and treated if positive for *H pylori* infection.

BPA 6: Patients who are using proton pump inhibitors (PPIs) for valid reasons do not need to discontinue these medications in the presence of documented fundic gland hyperplasia-related gastric polyps.

Gastric epithelial (or mucosal) polyps identified during upper gastrointestinal endoscopy are comprised of several subtypes (Table 1), each with distinct histological characteristics. These polyps frequently occur with associated risk factors or conditions, alterations in the background mucosa, and varying levels of malignant potential.

The prevalence of GAs, GHPs, and neuroendocrine lesions increases with advancing age, whereas that of FGPs decreases after age 60.[5] GAs and GHPs show either equal distribution between sexes or a slight female predominance, whereas FGPs demonstrate a female predominance.

In Western countries, most gastric polyps are identified incidentally during endoscopy, with 70% to 94% being either FGPs or GHPs.[6,7] Multiple polyp types coexist in 2% to 3% of patients. In these cases, FGP is the most common synchronous finding, whereas gastric adenomas (GAs) are often present alongside GHPs.

Cross-sectional studies of national pathology registries have examined the prevalence and distribution of gastric polyps in the United States.[6,8] One study indicated that the prevalence of gastric polyps was 6.35% among patients who underwent gastric biopsy during endoscopy from 2007 to 2008. Of these, 77% were FGPs, 17% were GHPs, 0.69% were GAs, and 0.1% were inflammatory fibroids.[6] In another study of upper endoscopies performed between 2008 and 2013, a higher prevalence of gastric polyps was reported: 7.72% of all patients had FGPs, 1.79% had GHPs, 0.09% had GAs, and 0.06% had type 1 G-NETs.[8] Recent trends have shown that the prevalence of *H pylori* infection and associated diseases (eg, peptic ulcer and distal gastric cancer) has declined, and gastroesophageal reflux disease has increased.[9] During the same time frame, the most commonly diagnosed subtypes of gastric polyps have also shifted from GHPs and GAs to sporadic FGPs.[8] A study conducted from 2004 to 2013 in Beijing reported that the proportion of GHPs decreased from 65% to 15%, while the proportion of FGPs increased from 19% to 77% over the same period.[10]

The risk of dysplastic transformation is exceedingly low (~1%) in sporadic FGPs.[11] Dysplastic changes are more common in familial adenomatous polyposis-associated FGPs (25%–46%). However, progression from low-grade to high-grade dysplasia or adenocarcinoma is generally slow, with an estimated rate of 4% in familial adenomatous polyposis patients (over a mean follow-up of 6 years).[12] The risk appears to be higher in patients presenting with mucosal carpeting by polyps (ie, FGPs extending from the fundus to the antrum without intervening normal mucosa) or polyps larger than 1 cm in size.[13] Patients with gastric adenocarcinoma and proximal polyposis syndrome (GAPPS) are also at a high risk of developing gastric cancer.[14]

Dysplasia occurs in about 4% of GHPs. Some studies report a similar or higher prevalence of dysplasia in the surrounding mucosa than within the polyp itself.[15] The rate of malignant transformation in GHPs varies significantly, ranging from 0.8% to 10% of cases, with polyps >2 cm in size being more likely to harbor dysplasia or adenocarcinoma.[16] Hamartomatous polyps share considerable histological similarities with GHPs, making the accompanying clinical and endoscopic evaluation crucial for accurate patient classification.

Morphologically, GAs represent a diverse category of neoplastic lesions, including intestinal, foveolar, pyloric, and oxyntic gland variants. Among these, intestinal-type (56%) and foveolar-type GAs (41%) are the most prevalent.[15] These 2 subtypes are precursors of adenocarcinomas, and the surrounding gastric mucosa carry a higher risk of malignant transformation. The risk of adenocarcinoma increases with polyp size, particularly for lesions larger than 20 mm. The risk of transformation is also very low to nonexistent for foveolar-type GAs with a characteristic raspberry-like polyp endoscopic appearance in *H pylori*-naïve stomach.[17–20]

Recent studies of pyloric gland adenomas have reported the presence of high-grade dysplasia in up to 42% and adenocarcinoma in 12% to 47% of cases.[21–23] Larger polyp size, tubulo-villous architecture, and autoimmune gastritis are associated with an increased

Table 1. Examples of Gastric Polyps and Their Representative Characteristics

Polyp type	Prevalence	Site/number/endoscopic appearance:	Associated condition, if any/ background mucosa alterations, if any	Malignant potential
FGP	47%–77% of polyps in areas with low prevalence of GHPs	Body/can be multiple/small, sessile, usually <1 cm, glassy surface. Can be part of FAP, GAPPS	PPI-associated/sporadic, FAP GAPPS The surrounding mucosa is usually normal without any inflammatory changes.	Dysplasia in up to 48% of FAP-associated FGPs; high in GAPPS; <1% in sporadic lesions. Gastric adenocarcinoma progression in up to 15% of cases in FAP
GHP	17%–55% of polyps. Common in regions with a high prevalence of *H pylori*	Antrum > body/single or multiple/ typically sessile or pedunculated, <20 mm, surface erosions may occur in larger polyps	Chronic gastritis (*H pylori*-related/ autoimmune)	Dysplasia is detected in ∼4% of polyps. The range of malignant transformation is 0.8%–10%. Neoplastic polyps tend to be >20 mm
Adenoma[a]	1%–10% of polyps	Antrum > body/usually single/flat or sessile, <20 mm	Chronic gastritis (*H pylori*-related/ autoimmune gastritis)	Progression: 3%–4% for low-grade lesions; 5%–30% for high-grade lesions
Pyloric gland adenoma	<3% of polyps	Body > antrum/usually single/ sessile, pedunculated or masses; ranging from 1–10 cm	Autoimmune gastritis; second most common polyp in FAP. Also seen in Lynch syndrome and juvenile polyposis	High-grade dysplasia in 42% of cases. Progression in 12%–30% of cases
Oxyntic gland adenoma	Rare	Body/polypoid or raised flat/solitary, ranging from 2–20 mm, typically <10 mm	Usually normal adjacent gastric mucosa	Large polyps and cases with mucus neck cell differentiation are likely to progress
Juvenile polyp	Rare	Body > antrum/usually pedunculated or sessile/may be solitary. ranging from 2–20 mm	Polyps at other GI segments/normal adjacent gastric mucosa	Progression in up to 14% of cases and higher in cases of polyposis
Peutz-Jeghers polyp	Rare	Any site/polypoid, lobulated on a short, broad stalk/solitary or multiple, ranging from 1–3 cm	Polyps at other GI segments/associated skin changes, normal or hypertrophic gastric mucosa	Progression in 2%–3% of cases
Cowden syndrome-associated polyp	Rare	Any site/small, sessile, polypoid/ multiple	Esophageal glycogenic acanthosis and various colorectal polyps, including mucosal lipomas and ganglioneuromas	Low risk of progression
Cronkite-Canada syndrome–associated polyp	Rare	Any site/sessile or flat with diffuse mucosal thickening/solitary or multiple	Diffuse foveolar hyperplasia, glandular atrophy, and cystic changes. Intestinal polyps at other sites. Alopecia, nail atrophy, skin hyperpigmentation, and vitiligo	Progression very rare

Table 1. Continued

Polyp type	Prevalence	Site/number/endoscopic appearance:	Associated condition, if any/ background mucosa alterations, if any	Malignant potential
G-NET	Uncommon	Body > antrum/types 1 and 2 tend to be multifocal, multiple, resembling polyps or nodules and sharply outlined. Type 3 may be solitary. Size: ≤2 cm for type 1, ≤1 cm for type 2, >2 cm for type 3	Type 1: body atrophy/GIM, with ECL cell proliferation. Type 2: hypertrophic, with ECL hyperplasia Part of MEN-1 and associated with gastrinoma Type 3: Normal surrounding mucosa	Limited rate of metastasis for types 1 and 2, and a higher rate for Type 3

ECL, enterochromaffin-like; FAP, familial adenomatous polyposis; FGP, fundic gland polyp; GAPPS, gastric adenocarcinoma and proximal polyposis syndrome; GHP, gastric hyperplastic polyp; GI, gastrointestinal; GIM, gastric intestinal metaplasia; G-NET, gastric neuroendocrine tumor; MEN 1, multiple endocrine neoplasia type 1; PPI, proton pump inhibitor.
[a]Except for foveolar-type adenoma arising in *H pylori*-naïve stomach.

risk of high-grade dysplasia as well as adenocarcinoma. In patients with Lynch syndrome, a higher-grade pyloric gland adenoma has been associated with male sex.[24]

Oxyntic gland adenoma is a rare, slow-growing neoplasm with a multilineage phenotype that demonstrates differentiation toward chief cells, parietal cells, and mucous neck cells. It uniquely arises from the oxyntic mucosa. Most cases follow a benign clinical course. Even when progression occurs, endoscopic resection is typically sufficient treatment, as this polyp is considered a low-grade malignancy.[25,26]

Well-differentiated G-NETs are a heterogeneous group of neoplasms with varied prognoses (intermediate to good). Most (>70%) arise in a setting of autoimmune gastritis (type 1 G-NET). Both types 1 and 2 are associated with Zollinger-Ellison syndrome with multiple endocrine neoplasia type 1 and frequently present as multiple yellowish polypoid nodules with a high risk of recurrence. Type 3 G-NETs are usually larger, mass-like, and associated with a worse prognosis.[27]

The risk of having FGPs is inversely associated with gastric atrophy, *H pylori* infection, and GIM. Conversely, patients with FGPs show positive associations with gastroesophageal reflux disease and PPI use as compared with patients with GAs.[5] The treatment and eradication of *H pylori* is associated with the elimination of GHPs. A meta-analysis of data from 6 studies, including 3 randomized controlled trials (RCTs), showed a 59% (95% confidence interval [CI], 43%–75%) reduction in prevalence of GHPs after *H pylori* treatment, and 79% (95% CI, 72%–86%) reduction following successful eradication.[28]

Over the past 2 decades, the use of PPIs has increased worldwide. Several observational studies and RCTs have reported the association between PPI use and parietal cell and enterochromaffin cell hyperplasia and FGPs.[29,30] A meta-analysis of 12 studies published through 2016 reported a 1.5- to 2.5-fold increase in the risk of FGPs with PPI use and up to a 5-fold increase with PPI use for at least 12 months.[29] Similar findings were reported in another meta-analysis with a significant association of 1.43 (95% CI, 1.24–1.64) and 2.45 (95% CI, 1.24–4.83) from fixed- and random-effects models, respectively.[31] Some of these associations can be attributed to confounders (eg, age, sex, indication for endoscopy).[32] Patients who are using PPIs for valid reasons do not need to discontinue these medications in the presence of documented fundic gland hyperplasia-related gastric polyps. However, for patients without clear indications for PPI use, discontinuing these medications is beneficial and requires a systematic approach.[33]

Similar associations are reported with the use of potassium-competitive acid blockers (P-CABs).[34] Results from the VISION trial highlighted an increase in the prevalence rate of hyperplastic polyps (HPs) from 3.7% to 14.7% over 3 years in patients initiating P-CAB treatment.[35]

Shinozaki et al reported a more than 3-fold elevation in the risk of having HPs among patients receiving P-CAB for more than 1 year compared with that of non-P-CAB users.[36] Case reports underscore the regression of HPs following P-CAB discontinuation.[37]

In patients not receiving PPIs, the detection of multiple FGPs at a younger age (20–40 years) or in pediatric age should prompt consideration of familial adenomatous polyposis (FAP). Syndromic patients have a high incidence of FGPs (over 80%) that typically develop within the first 3 decades of life.[38,39]

In this setting, the distinctive lesions are smaller than their PPI-associated or sporadic counterparts, display subtle morphologic differences, and are not associated with parietal cell hyperplasia.[40] Clinicians should remain vigilant, as FAP is now recognized to have a broader phenotypic spectrum than previously understood, and the classic presentation of massive colonic polyposis may not always be present, requiring careful clinical assessment with potential recourse to genetic evaluation.[41]

Anemia occurs more frequently in patients with GHPs, GAs, or G-NETs. G-NETs often occur in the setting of autoimmune gastritis, with associated corpus-restricted inflammation and corpus AG/GIM. Serum gastrin measurement, parietal cell antibodies, and the pepsinogen I to pepsinogen II ratio may be helpful in screening for autoimmune gastritis in patients with existing autoimmune diseases, unexplained iron-deficiency anemia, pernicious anemia, or a family history of autoimmune gastritis.[42,43] However, in most patients with gastric polyps, following gastrin levels does not provide any additional diagnostic information, and the laboratory testing of pepsinogen I to pepsinogen II ratio is not available in most clinical laboratories. There is no evidence to support the use of noninvasive biomarker testing for screening or monitoring gastric polyps and dysplasia, nor do any biomarkers effectively differentiate between low- and high-risk patients.[44]

Endoscopic Detection and Characterization of Gastric Polyps (BPA 4, 7–8)

BPA 4: Systematic endoscopic examination of the polyps and the surrounding gastric mucosa is essential in assessing the underlying gastric mucosa pathology (eg, *H pylori*-related gastritis, autoimmune gastritis, gastric intestinal metaplasia [GIM]) and determining subsequent management: biopsies of the polyps, biopsies of the surrounding mucosa, and resection of the polyps.

BPA 7: Different histological types of gastric polyps have unique/characteristic topographical features, endoscopic appearance, and size.

BPA 8: Endoscopic evaluation of patients with gastric polyps should include complete inspection with high-definition white-light and enhanced imaging, such as virtual chromoendoscopy. Endoscopists should recognize and photo-document the endoscopic features of gastric polyps as well as the surrounding gastric mucosal abnormalities.

The entire gastric mucosa should be carefully inspected using high-definition white-light endoscopy (HD-WLE) with adequate distension of the stomach, clearance of any mucus debris, and photo documentation of gastric landmarks, gastric lesions, and mucosal alterations. Visualization using HD-WLE alone is suboptimal for diagnosing mucosal alterations, such as gastric atrophy and GIM, or for accurately distinguishing dysplastic and early cancerous polyps without traditional histopathology. The rate at which neoplastic lesions are missed on upper endoscopy and result in cancer diagnosis within 3 years ranges from 4.7% to 11.3%.[45–52] Subtle mucosal lesions surrounding GIM have been associated with interval development of early cancer.[45]

Therefore, gastric polyps are either biopsied or resected for definitive histopathologic analysis to guide management decisions.[7] The systematic endoscopic examination and sampling of polyps and adjacent gastric mucosa are critical for evaluating *H pylori*-related gastritis, autoimmune gastritis, and GIM and determining subsequent management. Systematic sampling consists of biopsy/resection of polyps, targeted biopsies of suspicious mucosa, and random biopsies of different gastric regions for *H pylori*, atrophy, and GIM when polyps are suspected to be GAs or GHPs.[1] When sampling is unavailable, we advised testing *H pylori* using serology, a breath test, or stool antigen analysis. This approach is essential if adenomas or HPs are identified, as it can guide further management and treatment.

Table 1 lists examples of gastric polyps and their endoscopic characteristics. GHPs can be sessile or pedunculated, less than 20 mm in diameter, typically occurring as a single polyp in the antrum; however, they can arise as multiple polyps anywhere in the gastric mucosa.[6,53,54] FGPs are usually small, multiple, and sessile and are uniquely located in the gastric body and fundus. In the setting of FAP, they can coalesce and are at risk of becoming dysplastic.[37] Hamartomatous gastric polyps are typically sessile and solitary, predominantly seen in the gastric body, and are not easily distinguishable from GHPs.[55,56]

GAs account for 1% to 10% of all gastric polyps. They are typically single polyps, flat, and less than 20 mm, and occur in the gastric antrum. Intestinal-type adenomas of larger sizes, above 20 mm, and with villous histology have a higher risk of gastric cancer. G-NETs account for up to 2% of all gastric polyps. Small type 1 or type 2 sessile lesions are associated with autoimmune gastritis or Zollinger-Ellison syndrome with multiple endocrine neoplasia type 1, respectively. They can exist with or without a central depression, typically occur in the gastric body and fundus, and are associated with autoimmune gastritis, pernicious anemia, achlorhydria, and hypergastrinemia. Type 3 G-NETs account for

approximately 15% of NETs; can occur as solitary, larger polyps up to 5 cm; are not associated with hypergastrinemia; and have the worst prognosis.[57,58]

Virtual chromoendoscopy techniques with image-enhanced endoscopy (IEE), including narrow-band imaging (NBI), blue laser imaging (BLI), and linked color imaging (LCI) with or without magnifying endoscopy, have demonstrated improvement in the detection and characterization of dysplastic gastric lesions and abnormal gastric mucosa.[59–62] These modalities enhance color contrast between dysplastic areas and surrounding mucosa, enabling clear visualization of surface structures and blood vessels during close-up observations. In particular, LCI highlights the purple color of GIM, distinguishing it from other forms of inflammatory gastric mucosa and aiding in the identification of early gastric dysplastic or neoplastic lesions, which are often adjacent to GIM. Figure 1 shows images of representative polyps and adjacent mucosal changes.

A simplified NBI classification of abnormal gastric mucosa was proposed in a validation study by Pimentel-Nunes et al.[60] In this classification, pattern A is characterized by regular vessels with circular mucosa and was associated with normal histology (accuracy 83%; 95% CI, 75%–90%). Pattern B has tubulo-villous mucosa and was linked to GIM (accuracy 84%; positive likelihood ratio [LR+], 4.75). Pattern C has irregular vessels and mucosa and was associated with dysplasia (accuracy 95%; 95% CI, 90%–99%; LR+, 44.33). The "light-blue crest" sign showed moderate reliability ($k = 0.49$) but was specific (87%) for GIM. Variable vascular density was the best feature for identifying *H pylori*-related gastritis (accuracy: 70%).

The endoscopic diagnostic criteria for early gastric cancer lesions have been specified in the vessel-plus-surface classification and include features such as an irregular microvascular pattern and/or an irregular microsurface pattern along with a demarcation line that separates the early cancerous lesion from normal mucosa.[63] Magnifying BLI and NBI have shown similar sensitivities for identifying the demarcation line between normal and early cancerous lesions, with sensitivities of 96.1% and 98.1%, respectively, as well as for detecting irregular microvessel patterns, with sensitivities of 95.1% and 96.2%, respectively.[64] Additionally, irregular microsurface patterns were observed in 97.1% of lesions using BLI compared with 78.8% with NBI.[64]

These enhanced imaging tools have also been employed to evaluate abnormal gastric mucosa with GIM as well as AG and non-atrophic gastritis. Additional validated endoscopic classifications for gastric atrophy (Kimura-Takemoto classification) and GIM (endoscopic grading of GIM [EGGIM]) have been introduced but have not been routinely applied in daily practice by most endoscopists in the United States.[65–67] The interobserver reliability of endoscopic findings using these classifications remains moderate overall and depends on years of endoscopic experience and training.[37,68,69] In our opinion, the future implementation of these classification systems, following adequate training, could help standardize endoscopic findings, develop targeted biopsy guidance, and optimize endoscopic procedures.

Recent guidelines and statements from the AGA, American College of Gastroenterology, and European organizations recommend the routine use of IEE as a guiding tool for assessing and staging atrophic and metaplastic changes while targeting dysplastic lesions.[1,2,44,62] Further, IEE techniques that incorporate computer-assisted artificial intelligence systems have been introduced and are expected to facilitate the early detection of dysplastic lesions. Although IEE is commonly used in Eastern countries, it remains underutilized by endoscopists in the United States.[70] Until United States endoscopists acquire the necessary skills to distinguish mucosal abnormalities using IEE and accurately adopt classification systems accurately, meticulous endoscopic evaluations and the collection of random and targeted biopsies of polyps and surrounding gastric mucosa according to established gastric biopsy protocols will remain essential.

Endoscopic Management of Gastric Polyps and Surrounding Gastric Mucosa (BPA 9–11)

BPA 9: Endoscopic resection of the polyps includes traditional techniques (snare and biopsy forceps, mucosal resection) or endoscopic submucosal dissection.

BPA 10: In the presence of numerous gastric polyps of varied sizes, the largest polyps should be resected when possible, and the smaller polyps sampled or resected.

BPA 11: Suspected abnormalities in the surrounding mucosa, such as GIM or AG, should undergo targeted biopsies according to the existing protocols.

Endoscopic polyp resection aims to completely remove the lesion while providing tissue to accurately assess the polyp's most advanced pathological features. Methods for endoscopic gastric polypectomy include biopsy polypectomy, snare polypectomy, endoscopic mucosal resection (EMR), and endoscopic submucosal dissection (ESD). The choice of polypectomy technique incorporates the presumed polyp pathology, size, location, and the endoscopist's expertise. Little direct data exists on the use of cold forceps for small gastric polyps $\leq$3 mm in size, but their use is supported by literature on polypectomy of the colon. One large, prospective study found that, for colonic polyps $\leq$3 mm in size, cold forceps polypectomy (with 2.4-mm-diameter large forceps) was shown to be noninferior to cold snare polypectomy in achieving complete resection, and the procedure can be performed more quickly.[71] Polyps $\geq$4 mm in size should not be resected using the forceps technique. Several prospective studies have evaluated forceps compared with EMR for polypectomy in larger polyps ($\geq$5 mm).[72–75] All 4 studies demonstrated that forceps cannot reliably establish the presence of underlying neoplasia in larger polyps. Thus, for polyps between 4 and 10 mm, either cold snare or EMR are

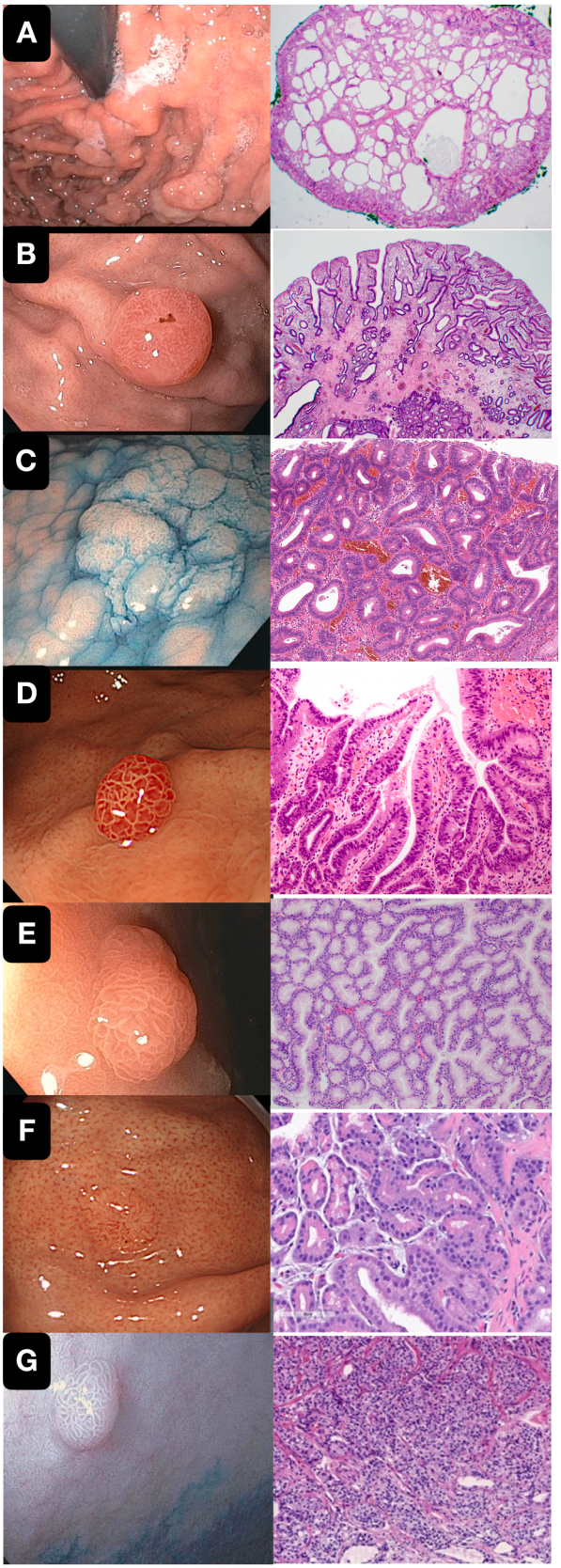

Figure 1. Representative images of gastric polyps and representative mucosal abnormalities with corresponding HD-WLE/IEE imaging and corresponding histologic images. (*A*) Fundic gland polyp. Dilated fundic glands are a key characteristic. They are predominantly lined with parietal cells and occasionally chief cells or foveolar neck cells. (*B*) GHP. The lesion is composed of elongated and tortuous pits with cystic dilations and outpouchings. The lamina propria is edematous and variably inflamed. (*C*) Intestinal-type adenoma (low grade) showing dense glandular proliferation with simple glandular architecture, enlarged penicillate nuclei, and limited pseudo-stratification. High-grade dysplasia is characterized by a more complex architecture and pronounced nuclear atypia. (*D*) Raspberry-like polyp in *H pylori*-naïve patient. The irregularly shaped tubulo-papillary structures are lined by low-grade foveolar dysplastic epithelium. (Courtesy Professor T. Ushiku, Tokyo, Japan). (*E*) Pyloric gland adenoma. The packed glands are lined by a monolayer of pale eosinophilic low columnar cells with round open-chromatin nuclei. (*F*) Oxyntic gland adenomas are composed of tightly packed tubules made of an admixture of chief and parietal cells in most cases. Presented is a subtype predominantly composed of mucous neck cell differentiation. (*G*) Well-differentiated G-NET. The lesion is well-circumscribed and composed of anastomosing clusters and trabeculae of uniform cuboidal cells immunoreactive with synaptophysin antibody.

appropriate. One study from Taiwan demonstrated that cold snare was noninferior to EMR with regards to adverse events (most commonly bleeding) in gastric polypectomy.[76] Another Taiwanese study found cold snare to be safe and effective in removing FGPs between 3 and 8 mm in size.[77] Regarding adverse events from EMR, the largest study to date suggests a bleeding rate of approximately 7%, the vast majority of which could be managed endoscopically. Perforation from EMR appears to be rare.[72] ESD should be restricted to polyps in which neoplasia is diagnosed or strongly suspected. A single-center Korean study of 282 gastric adenomatous polyps removed by ESD reported a high complete resection rate (96%), a low bleeding rate (1.4%), and no perforations.[78] In that study, lesion size >20 mm was predictive of early gastric cancer.[78] Two reports suggest that gastric ESD can also be performed safely and efficiently in the West.[79,80] Both EMR and ESD could allow for en bloc resection depending on polyp size and morphology, as even a >20 mm pedunculated polyp can still be resected en bloc by EMR. Referral to an endoscopist with experience in advanced resection techniques is advised if the polyp size exceeds 20 mm.

Figure 2 depicts our suggested management strategy for newly diagnosed sporadic gastric polyps. Although it has been suggested that an initial biopsy followed by resection based on histology could be appropriate,[81–83] we advise that all newly detected solitary polyps be resected at the time of index endoscopy. This course of action achieves both diagnostic and therapeutic intentions. If multiple gastric polyps are newly detected, the endoscopist needs to use knowledge of regional polyp epidemiology, clinical history, and polyp characteristics to inform the endoscopic approach. If multiple polyps are seen and all are suspected to be FGPs, we advise resecting the largest suspected FGP. Any other suspected FGPs can be resected if large (>10 mm) or visually atypical.[84] If multiple polyps are detected but

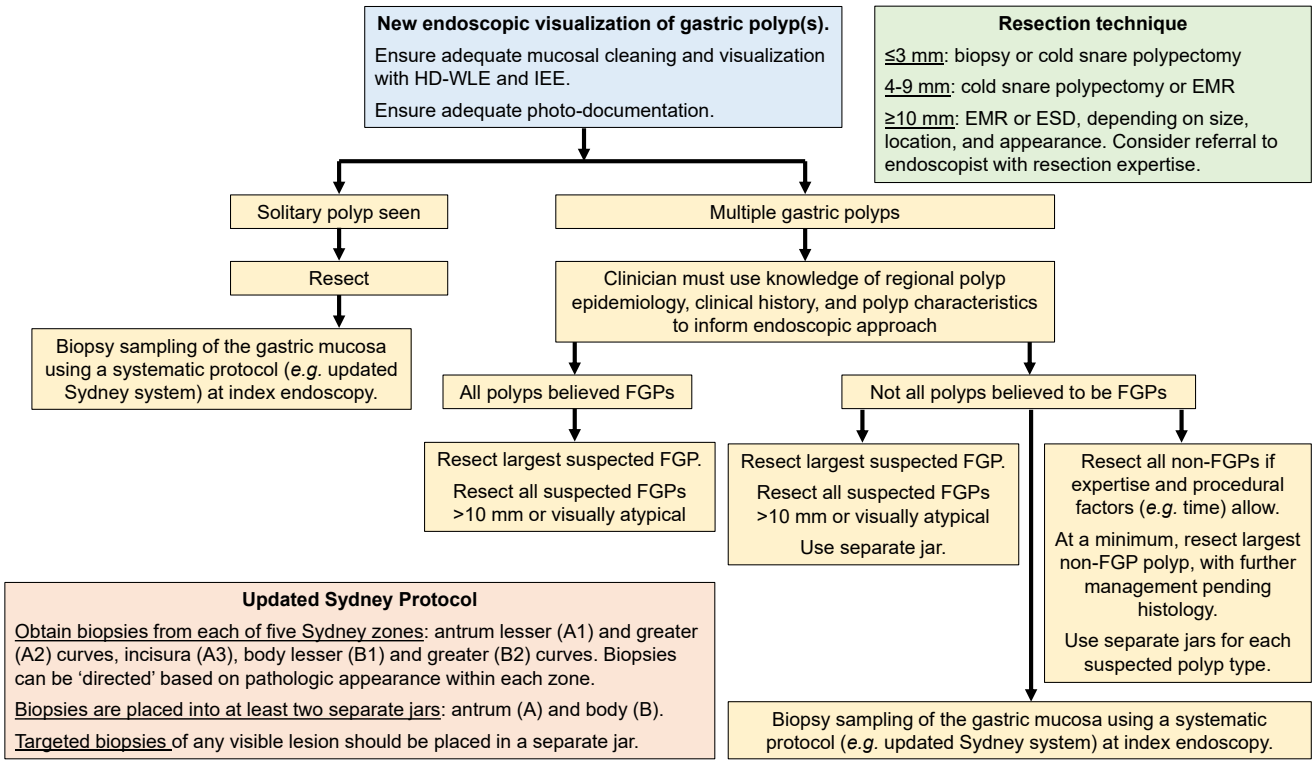

Figure 2. Proposed management strategy for newly visualized gastric polyps. This advice is appropriate for countries where *H pylori* prevalence is low, and PPI use is common, such as the United States. This advice pertains to patients without a known genetic cancer syndrome (eg, FAP). For surveillance based on index endoscopy results, see Table 2.

not all are thought to be FGPs, the approach would depend on provider expertise and procedural factors (eg, procedure length, availability of anesthesia support, staff training, equipment availability). At a minimum, resecting the largest suspected non-FGP polyp to establish a histologic diagnosis and assess for the presence of deeper neoplasia should be considered. If procedural factors allow the complete removal of all suspected non-FGP polyps, this may obviate the need for a second procedure. If multiple polyp types are suspected to be present (eg, FGPs and HPs), separate pathologic specimen jars are obtained for each suspected polyp type.

If a suspected non-FGP is detected on index endoscopy, we advise mucosal sampling using an updated Sydney System biopsy or the MAPPS II protocol.[85] Similarly, if a solitary gastric polyp is observed, we advise systematic mucosa sampling. The topographic pattern of inflammation, atrophy, and GIM obtained through these protocols can help distinguish between *H pylori*-related and autoimmune gastritis.

Post-endoscopic Treatment Management of Gastric Polyps and Surrounding Gastric Mucosa with Determinations of Intervals for Endoscopic Surveillance (BPA 12–14)

BPA 12: Surveillance plans in patients with gastric polyps should be formulated based on the histopathological type of the polyps and the surrounding gastric mucosa.

BPA 13: When a dysplastic lesion in the polyp is confirmed and resected completely, a follow-up surveillance endoscopy should be completed in 1 year for patients with low-grade dysplasia polyps and 6 months for patients with high-grade dysplasia polyps. If the polyp is biopsied or resection is incomplete, follow-up endoscopy is advised within 3 months for high-grade dysplasia and 6 months for low-grade dysplasia.

BPA 14: Endoscopic surveillance is advised in patients with gastric polyps when the histopathology of adjacent mucosa confirms GIM and/or atrophic gastritis (AG).

The malignant potential of gastric polyps varies based on type. Accurate histological classification of gastric polyps is crucial for determining the appropriate therapeutic and surveillance strategies (Table 2, Figure 2). Thus, a comprehensive histological assessment of the polyp and surrounding gastric mucosa is needed to evaluate the patient's risk for malignant progression. Although ethnic origin and family history of gastric cancer is associated with the risk of gastric cancer, the frequency of surveillance should not be altered based on these factors. There is little evidence that these associations are mediated by factors other than the gastric mucosal status (eg, polyps, atrophy).

In general, polyp size (irrespective of type) and the presence of dysplasia guide management. Surveillance endoscopy is not usually needed for non-syndromic FGPs devoid of dysplasia. However, watchful follow-up is advised for syndromic patients with a diagnosis of dysplastic FGP, large polyps (>10 mm), or mucosal

Table 2. Advice on Surveillance of Gastric Polyps

Polyp histology and characteristics	Endoscopic surveillance interval	Other management considerations
FGP[a]		
<10 mm, no dysplasia	No surveillance	-
>10 mm, no dysplasia	1 y	Discontinue PPI therapy.
Mucosal carpeting (>20 polyps)	1 y	Discontinue PPI therapy and consider evaluation for familial polyposis syndrome.
With dysplasia	If high-grade, 6 mo; if low-grade, 1 y	Evaluate for familial polyposis syndrome.
HP		
<10 mm, no dysplasia	Not clearly indicated if all polyps resected	Ensure *H pylori* negativity. If gastric premalignant conditions present, follow AGA advice.[b]
>10 mm, no dysplasia	Endoscopic surveillance in 1 year	
With dysplasia	If high-grade, 6 mo; if low-grade, 1 y	
Adenoma (including intestinal-type, pyloric gland, and oxyntic gland)	Ensure complete removal of index adenoma. Consider referral to endoscopic with experience in advanced resection techniques. Survey every 6 mo for high-grade dysplasia and every year for low-grade dysplasia, and then annually thereafter.	Ensure *H pylori* negativity. If gastric premalignant conditions are present, follow AGA advice.[b]
G-NET		
Type I or type II	If ≥2 cm, consider referral to oncologic center with expertise in management If <2 cm, every 1–2 y	If autoimmune gastritis is present, follow existing AGA advice.[b] If type II, evaluation for source of inappropriate gastrin production.
Type III	Referral to oncologic center with expertise in management	

NOTE. This advice is appropriate for countries where *Helicobacter pylori* (*H pylori*) prevalence is low, and PPI use is common, such as the United States. If multiple polyp types are present, advice for the highest-grade polyp should take precedence.
AG, atrophic gastritis; AGA, American Gastroenterological Association; FGP, fundic gland polyp; G-NET, gastric neuroendocrine tumor; GIM, gastric intestinal metaplasia; HP, hyperplastic polyp; PPI, proton pump inhibitor.
[a]This advice pertains to patients without a known genetic cancer syndrome.
[b]If gastric premalignant conditions (eg, chronic AG, GIM) are detected on index endoscopy, see existing AGA advice on chronic AG.[2,4]

carpeting by polyps because they are associated with the development of gastric cancer.[13] For GHPs, endoscopic resection is advised for large polyps (>10 mm), and if dysplasia is noted on biopsy, periodic (eg, yearly) surveillance is advised.[86] Given the potential for progression of GAs, we advise a surveillance strategy with an initial follow-up at 6 months for high-grade dysplasia and 12 months for low-grade after excision and subsequent follow-up annually to monitor for malignant changes.[83] If the polyp is biopsied or resection is incomplete, follow-up endoscopy is suggested within 3 months for high-grade dysplasia and 6 months for low-grade dysplasia polyps. We suggest the same approaches for pyloric gland and oxyntic gland adenomas. Decisions regarding the endoscopic management of G-NETs depend on the size, number, and grade. For example, larger lesions (>2 cm) are associated with higher metastatic risk. Routine tattooing is not advised for most diagnostic or therapeutic gastric polypectomies; however, it can be considered (optional) for the site of incompletely resected large polyps, suspected malignant polyps, or when surgery or EMR/ESD follow-up is planned.[87–89]

Assessment of the gastric mucosa is essential because patients with longstanding *H pylori*-related or autoimmune gastritis who have developed mucosal atrophy, pseudo-pyloric metaplasia, or GIM have an increased risk of gastric polyps. We advise obtaining biopsies from the gastric antrum and corpus at a minimum. Successful eradication of *H pylori* infection can lead to regression of hyperplastic polyps,[90] improve AG, and decrease the risk of gastric cancer.[62,91] Targeted biopsies every 3 years are considered for patients with persistent *H pylori* infection, advanced AG when incomplete GIM is detected, or patients with mild/focal GIM and a family history of gastric cancer.[3] The duration of the surveillance has not yet been determined. Clinicians should assess multiple high-risk features in their patients and engage in shared decision-making to ensure a comprehensive approach to care.

Conclusion

This CPU summarizes the BPA statements for diagnosing and managing gastric polyps. Gastric polyps are

among the most encountered lesions detected during upper endoscopy. The proposed management includes endoscopic characterization of polyps and adjacent mucosa using image-enhancement tools, followed by biopsy, resection, and appropriate follow-up surveillance exams.

References

1. Nagula S, Parasa S, Laine L, Shah SC. AGA clinical practice update on high-quality upper endoscopy: expert review. Clin Gastroenterol Hepatol 2024;22:933–943.
2. Shah SC, Wang AY, Wallace MB, Hwang JH. AGA clinical practice update on screening and surveillance in individuals at increased risk for gastric cancer in the United States: expert review. Gastroenterology 2025;168:405–416.e1.
3. Gupta S, Li D, El Serag HB, et al. AGA clinical practice guidelines on management of gastric intestinal metaplasia. Gastroenterology 2020;158:693–702.
4. Shah SC, Piazuelo MB, Kuipers EJ, Li D. AGA clinical practice update on the diagnosis and management of atrophic gastritis: expert review. Gastroenterology 2021;161:1325–1332.e7.
5. Sonnenberg A, Genta RM. Prevalence of benign gastric polyps in a large pathology database. Dig Liver Dis 2015;47:164–169.
6. 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; 104:1524–1532.
7. ASGE Standards of Practice Committee, Muthusamy VR, Lightdale JR, et al. The role of endoscopy in the management of GERD. Gastrointest Endosc 2015;81:1305–1310.
8. O'Connor HJ. Forty years of Helicobacter pylori infection and changes in findings at esophagogastroduodenoscopy. Helicobacter 2023;28:e13026.
9. el-Serag HB, Sonnenberg A. Opposing time trends of peptic ulcer and reflux disease. Gut 1998;43:327–333.
10. Fan NN, Yang J, Sun G, et al. Changes in the spectrum of gastric polyps in the Chinese population. World J Gastroenterol 2015;21:9758–9764.
11. Wu TT, Kornacki S, Rashid A, et al. Dysplasia and dysregulation of proliferation in foveolar and surface epithelia of fundic gland polyps from patients with familial adenomatous polyposis. Am J Surg Pathol 1998;22:293–298.
12. Arnason T, Liang WY, Alfaro E, et al. Morphology and natural history of familial adenomatous polyposis-associated dysplastic fundic gland polyps. Histopathology 2014; 65:353–362.
13. Wang KK, Kim K, Bancila L, et al. Novel endoscopic polypectomy surveillance technique for fundic gland polyps in familial adenomatous polyposis can improve early detection of dysplasia and gastric cancer. Am J Gastroenterol 2022; 117:1246–1254.
14. de Boer WB, Ee H, Kumarasinghe MP. Neoplastic lesions of gastric adenocarcinoma and proximal polyposis syndrome (GAPPS) are gastric phenotype. Am J Surg Pathol 2018;42:1–8.
15. Kovari B, Kim BH, Lauwers GY. The pathology of gastric and duodenal polyps: current concepts. Histopathology 2021; 78:106–124.
16. Forte E, Petit B, Walter T, et al. Risk of neoplastic change in large gastric hyperplastic polyps and recurrence after endoscopic resection. Endoscopy 2020;52:444–453.
17. Naka T, Hashimoto T, Cho H, et al. Sporadic and familial adenomatous polyposis-associated foveolar-type adenoma of the stomach. Am J Surg Pathol 2023;47:91–101.
18. Shibagaki K, Mishiro T, Fukuyama C, et al. Sporadic foveolar-type gastric adenoma with a raspberry-like appearance in Helicobacter pylori-naive patients. Virchows Arch 2021; 479:687–695.
19. Kim M, Kim TS, Min BH, et al. Clinicopathologic features and outcomes of endoscopic submucosal dissection for foveolar-type adenocarcinoma of the stomach. J Gastric Cancer 2024; 24:367–377.
20. Sakamoto U, Adachi K, Kishi K, et al. Prevalence of raspberry-type gastric foveolar-type tumor in individuals undergoing medical checkups. Intern Med 2023;62:2775–2781.
21. Chen ZM, Scudiere JR, Abraham SC, Montgomery E. Pyloric gland adenoma: an entity distinct from gastric foveolar type adenoma. Am J Surg Pathol 2009;33:186–193.
22. Vieth M, Montgomery EA. Some observations on pyloric gland adenoma: an uncommon and long ignored entity! J Clin Pathol 2014;67:883–890.
23. Choi WT, Brown I, Ushiku T, et al. Gastric pyloric gland adenoma: a multicentre clinicopathological study of 67 cases. Histopathology 2018;72:1007–1014.
24. Lee SE, Kang SY, Cho J, et al. Pyloric gland adenoma in Lynch syndrome. Am J Surg Pathol 2014;38:784–792.
25. Ushiku T, Kunita A, Kuroda R, et al. Oxyntic gland neoplasm of the stomach: expanding the spectrum and proposal of terminology. Mod Pathol 2020;33:206–216.
26. Benedict MA, Lauwers GY, Jain D. Gastric adenocarcinoma of the fundic gland type: update and literature review. Am J Clin Pathol 2018;149:461–473.
27. Vanoli A, La Rosa S, Miceli E, et al. Prognostic evaluations tailored to specific gastric neuroendocrine neoplasms: analysis of 200 cases with extended follow-up. Neuroendocrinology 2018;107:114–126.
28. 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: e12838.
29. Shanika LGT, Reynolds A, Pattison S, Braund R. Proton pump inhibitor use: systematic review of global trends and practices. Eur J Clin Pharmacol 2023;79:1159–1172.
30. Martin FC, Chenevix-Trench G, Yeomans ND. Systematic review with meta-analysis: fundic gland polyps and proton pump inhibitors. Aliment Pharmacol Ther 2016;44:915–925.
31. Tran-Duy A, Spaetgens B, Hoes AW, et al. Use of proton pump inhibitors and risks of fundic gland polyps and gastric cancer: systematic review and meta-analysis. Clin Gastroenterol Hepatol 2016;14:1706–1719.e5.
32. Lam SK, Lau GKK. Proton pump inhibitors are not associated with fundic gland polyps - a systematic review that takes into consideration all known confounders. Eur J Gastroenterol Hepatol 2024;36:831–844.
33. Targownik LE, Fisher DA, Saini SD. AGA clinical practice update on de-prescribing of proton pump inhibitors: expert review. Gastroenterology 2022;162:1334–1342.
34. Kubo K, Kimura N, Kato M. Potassium-competitive acid blocker-associated gastric mucosal lesions. Clin Endosc 2024; 57:417–423.
35. Haruma K, Kinoshita Y, Yao T, et al. Randomised clinical trial: 3-year interim analysis results of the VISION trial to evaluate the

long-term safety of vonoprazan as maintenance treatment in patients with erosive oesophagitis. BMC Gastroenterol 2023; 23:139.
36. Shinozaki S, Osawa H, Hayashi Y, et al. Changes in gastric morphology during long-term use of vonoprazan compared to proton pump inhibitors. Singapore Med J 2022;63:283–287.
37. Jin EH, Chung SJ, Lim JH, et al. Training effect on the inter-observer agreement in endoscopic diagnosis and grading of atrophic gastritis according to level of endoscopic experience. J Korean Med Sci 2018;33:e117.
38. Iida M, Yao T, Itoh H, et al. Natural history of fundic gland polyposis in patients with familial adenomatosis coli/Gardner's syndrome. Gastroenterology 1985;89:1021–1025.
39. Bianchi LK, Burke CA, Bennett AE, et al. Fundic gland polyp dysplasia is common in familial adenomatous polyposis. Clin Gastroenterol Hepatol 2008;6:180–185.
40. Kovari B, El Naili R, Pereira DV, et al. Fundic gland polyps related to diverse aetiologies show subtle morphological differences: a multicentre retrospective study. Histopathology 2022;80:827–835.
41. Zare B, Monahan KJ. Guidelines for Familial Adenomatous Polyposis (FAP): challenges in defining clinical management for a rare disease. Fam Cancer 2025;24:35.
42. Taylor L, McCaddon A, Wolffenbuttel BHR. Creating a framework for treating autoimmune gastritis-the case for replacing lost acid. Nutrients 2024;16:662.
43. Lenti MV, Rugge M, Lahner E, et al. Autoimmune gastritis. Nat Rev Dis Primers 2020;6:56.
44. Morgan DR, Corral JE, Li D, et al. ACG clinical guideline: diagnosis and management of gastric premalignant conditions. Am J Gastroenterol 2025;120:709–737.
45. Cho YS, Chung IK, Kim JH, et al. Risk factors of developing interval early gastric cancer after negative endoscopy. Dig Dis Sci 2015;60:936–943.
46. Alexandre L, Tsilegeridis-Legeris T, Lam S. Clinical and endoscopic characteristics associated with post-endoscopy upper gastrointestinal cancers: a systematic review and meta-analysis. Gastroenterology 2022;162:1123–1135.
47. Hernanz N, Rodriguez de Santiago E, Marcos Prieto HM, et al. Characteristics and consequences of missed gastric cancer: a multicentric cohort study. Dig Liver Dis 2019;51:894–900.
48. Kitagawa Y, Suzuki T, Nankinzan R, et al. Comparison of endoscopic visibility and miss rate for early gastric cancers after Helicobacter pylori eradication with white-light imaging versus linked color imaging. Dig Endosc 2020;32:769–777.
49. Ang TL, Pittayanon R, Lau JY, et al. A multicenter randomized comparison between high-definition white light endoscopy and narrow band imaging for detection of gastric lesions. Eur J Gastroenterol Hepatol 2015;27:1473–1478.
50. Ono S, Kawada K, Dohi O, et al. Linked color imaging focused on neoplasm detection in the upper gastrointestinal tract : a randomized trial. Ann Intern Med 2021;174:18–24.
51. Honing J, Keith Tan W, Dieninyte E, et al. Adequacy of endoscopic recognition and surveillance of gastric intestinal metaplasia and atrophic gastritis: a multicentre retrospective study in low incidence countries. PLoS One 2023;18:e0287587.
52. Matsumoto K, Ueyama H, Yao T, et al. Diagnostic limitations of magnifying endoscopy with narrow-band imaging in early gastric cancer. Endosc Int Open 2020;8:E1233–E1242.
53. Deppisch LM, Rona VT. Gastric epithelial polyps. A 10-year study. J Clin Gastroenterol 1989;11:110–115.
54. Castro R, Pimentel-Nunes P, Dinis-Ribeiro M. Evaluation and management of gastric epithelial polyps. Best Pract Res Clin Gastroenterol 2017;31:381–387.
55. Park DY, Lauwers GY. Gastric polyps: classification and management. Arch Pathol Lab Med 2008;132:633–640.
56. Jung I, Gurzu S, Turdean GS. Current status of familial gastrointestinal polyposis syndromes. World J Gastrointest Oncol 2015;7:347–355.
57. Costa D, Ramai D, Tringali A. Novel classification of gastric polyps: the good, the bad and the ugly. World J Gastroenterol 2024;30:3640–3653.
58. Gluckman CR, Metz DC. Gastric neuroendocrine tumors (Carcinoids). Curr Gastroenterol Rep 2019;21:13.
59. Miura Y, Osawa H, Sugano K. Recent progress of image-enhanced endoscopy for upper gastrointestinal neoplasia and associated lesions. Dig Dis 2024;42:186–198.
60. Pimentel-Nunes P, Dinis-Ribeiro M, Soares JB, et al. A multicenter validation of an endoscopic classification with narrow band imaging for gastric precancerous and cancerous lesions. Endoscopy 2012;44:236–246.
61. Li HY, Dai J, Xue HB, et al. Application of magnifying endoscopy with narrow-band imaging in diagnosing gastric lesions: a prospective study. Gastrointest Endosc 2012; 76:1124–1132.
62. Pimentel-Nunes P, Libanio D, Marcos-Pinto R, et al. Management of epithelial precancerous conditions and lesions in the stomach (MAPS II): European Society of Gastrointestinal Endoscopy (ESGE), European Helicobacter and Microbiota Study Group (EHMSG), European Society of Pathology (ESP), and Sociedade Portuguesa de Endoscopia Digestiva (SPED) guideline update 2019. Endoscopy 2019;51:365–388.
63. Yao K, Anagnostopoulos GK, Ragunath K. Magnifying endoscopy for diagnosing and delineating early gastric cancer. Endoscopy 2009;41:462–467.
64. Dohi O, Yagi N, Majima A, et al. Diagnostic ability of magnifying endoscopy with blue laser imaging for early gastric cancer: a prospective study. Gastric Cancer 2017;20:297–303.
65. Quach DT, Hiyama T. Assessment of endoscopic gastric atrophy according to the Kimura-Takemoto classification and its potential application in daily practice. Clin Endosc 2019; 52:321–327.
66. Maric L, Castaneda D, Singh H, et al. Kimura-Takemoto classification: a tool to predict gastric intestinal metaplasia progression to advanced gastric neoplasia. Dig Dis Sci 2022; 67:4092–4099.
67. Rugge M, Genta RM, Malfertheiner P, et al. RE.GA.IN.: the Real-world Gastritis Initiative-updating the updates. Gut 2024; 73:407–441.
68. Tiankanon K, Pittayanon R, Faknak N, et al. Diagnostic validity and learning curve of non-NBI expert endoscopists in gastric intestinal metaplasia diagnosis. Surg Endosc 2023; 37:6771–6778.
69. Miwata T, Quach DT, Hiyama T, et al. Interobserver and intraobserver agreement for gastric mucosa atrophy. BMC Gastroenterol 2015;15:95.
70. Marcos P, Brito-Goncalves G, Libanio D, et al. Endoscopic grading of gastric intestinal metaplasia on risk assessment for early gastric neoplasia: can we replace histology assessment also in the West? Gut 2020;69:1762–1768.
71. Wei MT, Louie CY, Chen Y, et al. Randomized controlled trial investigating cold snare and forceps polypectomy among small

POLYPs in rates of complete resection: the TINYPOLYP trial. Am J Gastroenterol 2022;117:1305–1310.
72. Muehldorfer SM, Stolte M, Martus P, et al. , Multicenter Study Group "Gastric Polyps". Diagnostic accuracy of forceps biopsy versus polypectomy for gastric polyps: a prospective multicentre study. Gut 2002;50:465–470.
73. Szaloki T, Toth V, Nemeth I, et al. Endoscopic mucosal resection: not only therapeutic, but a diagnostic procedure for sessile gastric polyps. J Gastroenterol Hepatol 2008; 23:551–555.
74. Szaloki T, Toth V, Tiszlavicz L, Czako L. Flat gastric polyps: results of forceps biopsy, endoscopic mucosal resection, and long-term follow-up. Scand J Gastroenterol 2006;41:1105–1109.
75. Sung HY, Cheung DY, Cho SH, et al. Polyps in the gastrointestinal tract: discrepancy between endoscopic forceps biopsies and resected specimens. Eur J Gastroenterol Hepatol 2009;21:190–195.
76. Ma TL, Yang SC, Wu CK, et al. Safety of cold snare polypectomy vs conventional hot polypectomy for 4 to 10 mm gastric polyps: a single-center retrospective study. Adv Dig Med 2024;11:24–32.
77. Chen CH, Huang JC, Shin JS. Cold snare polypectomy for fundic gland polyps. Adv Dig Med 2022;9:17–22.
78. Choi CW, Kang DH, Kim HW, et al. Endoscopic submucosal dissection as a treatment for gastric adenomatous polyps: predictive factors for early gastric cancer. Scand J Gastroenterol 2012;47:1218–1225.
79. Tate DJ, Klein A, Sidhu M, et al. Endoscopic submucosal dissection for suspected early gastric cancer: absolute versus expanded criteria in a large Western cohort (with video). Gastrointest Endosc 2019;90:467–479.e4.
80. Ngamruengphong S, Ferri L, Aihara H, et al. Efficacy of endoscopic submucosal dissection for superficial gastric neoplasia in a large cohort in North America. Clin Gastroenterol Hepatol 2021;19:1611–1619.e1.
81. ASGE Standards of Practice Committee, Sharaf RN, Shergill AK, et al. Endoscopic mucosal tissue sampling. Gastrointest Endosc 2013;78:216–224.
82. ASGE Standards of Practice Committee, Evans JA, Chandrasekhara V, et al. The role of endoscopy in the management of premalignant and malignant conditions of the stomach. Gastrointest Endosc 2015;82:1–8.
83. Goddard AF, Badreldin R, Pritchard DM, et al. , British Society of Gastroenterology. The management of gastric polyps. Gut 2010;59:1270–1276.
84. Shaib YH, Rugge M, Graham DY, Genta RM. Management of gastric polyps: an endoscopy-based approach. Clin Gastroenterol Hepatol 2013;11:1374–1384.
85. 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;20:1161–1181.
86. Banks M, Graham D, Jansen M, et al. British Society of Gastroenterology guidelines on the diagnosis and management of patients at risk of gastric adenocarcinoma. Gut 2019;68:1545–1575.
87. Committee AT, Kethu SR, Banerjee S, et al. Endoscopic tattooing. Gastrointest Endosc 2010;72:681–685.
88. Kato MUN, Hokimoto N, Iishi H. Current status of EMR/ESD in the stomach: indications, outcomes and limitations. Dig Endosc 2020;32(Suppl 1):5–12.
89. Manta RZA, Parodi A, D'Alba L, et al. Clinical indications for endoscopic tattooing and impact on clinical management: a multicenter European survey. Endosc Int Open 2021;9:E39–E44.
90. Nam SY, Lee SW, Jeon SW, et al. Helicobacter pylori eradication regressed gastric hyperplastic polyp: a randomized controlled trial. Dig Dis Sci 2020;65:3652–3659.
91. Sugano K. Effect of Helicobacter pylori eradication on the incidence of gastric cancer: a systematic review and meta-analysis. Gastric Cancer 2019;22:435–445.

Correspondence
Address correspondence to: Anna M. Buchner, MD, PhD, Division of Gastroenterology and Hepatology, University of Pennsylvania, 3400 Civic Center Boulevard, 741 S, Philadelphia, Pennsylvania 19104. e-mail: anna.buchner@pennmedicine.upenn.edu.

CRediT Authorship Contributions
Anna M. Buchner (Writing – review & editing; Writing – original draft; Conceptualization)
Robert J. Huang (Writing – review & editing; Writing – original draft; Conceptualization)
Gregory Y. Lauwers (Writing – review & editing; Writing – original draft; Conceptualization)
Hashem B. El-Serag (Conceptualization)

Conflicts of interest
The authors disclose no conflicts.