# Gastric polyps

**AUTHORS:** Varocha Mahachai, MD, David Y Graham, MD, Robert D Odze, MD, FRCPC
**SECTION EDITOR:** Mark Feldman, MD, MACP, AGAF, FACG
**DEPUTY EDITOR:** Shilpa Grover, MD, MPH, AGAF

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Literature review current through: **Sep 2023.**
This topic last updated: **Feb 20, 2023.**

## INTRODUCTION

Gastric polyps are usually found incidentally on upper gastrointestinal endoscopy performed for an unrelated indication and only in rare cases do they cause symptoms. Nevertheless, the diagnosis and appropriate management of gastric polyps are important, as some polyps have malignant potential.

This topic will review the epidemiology, clinical manifestations, histopathology, and management of gastric polyps. Our recommendations are largely consistent with the American Society for Gastrointestinal Endoscopy guidelines [1,2]. The clinical manifestations, diagnosis and management of gastric gastrointestinal stromal tumors, leiomyomas, lipomas, and other subepithelial lesions that may have a polypoid appearance on upper endoscopy are discussed in detail, separately. (See "Endoscopic ultrasound for the characterization of subepithelial lesions of the upper gastrointestinal tract" and "Clinical presentation, diagnosis, and prognosis of gastrointestinal stromal tumors" and "Local treatment for gastrointestinal stromal tumors, leiomyomas, and leiomyosarcomas of the gastrointestinal tract".)

## EPIDEMIOLOGY

Gastric polyps are found in approximately 6 percent of upper gastrointestinal endoscopic procedures in the United States [3]. However, lower rates have been reported in other countries [4,5]. Hyperplastic polyps and adenomas are relatively more prevalent as compared with fundic

gland polyps in regions where *Helicobacter pylori* infection is common [4-6]. In contrast, in Western countries, where the prevalence of *H. pylori* infection is lower and proton pump inhibitor (PPI) use is common, the most commonly encountered polyps are fundic gland polyps ( picture 1A-B) [3,6].

## INITIAL EVALUATION

The initial approach to gastric polyps should include an evaluation of both polyp histology and the surrounding mucosa.

- **Evaluation of polyp histology** – In patients with small solitary polyps, either biopsy samples should be obtained or polypectomy performed so that the polyp can be examined microscopically for histologic characterization [7-11]. Polypectomy should be performed for all known neoplastic polyps and for all polyps ≥1 cm in diameter, as biopsies alone cannot exclude foci of high-grade dysplasia or early gastric cancer [9]. In patients with multiple polyps, the largest polyp should be excised and representative biopsies obtained from the remaining polyps [1,2]. In patients with sessile polyps, endoscopic mucosal resection may be needed to provide an accurate histological assessment and achieve complete resection. Further management should be based on histology [12]. (See "Overview of endoscopic resection of gastrointestinal tumors" and 'Types of gastric polyps and specific management' below.)

- **Assessment of surrounding mucosa** – Once polyps are biopsied or resected, the normal-appearing antral and corpus mucosa should be sampled to rule out dysplasia occurring in the background of metaplastic atrophic gastritis and to diagnose *H. pylori* [2]. We obtain 7 to 12 biopsies: four quadrant biopsies from antrum (2 to 3 cm proximal to pylorus), two from the angularis, four from the mid corpus (two lesser curvature, two greater curvature), and two from the cardia. Gastritis should be classified using a validated histologic staging system such as the Operative Link on Gastritis Assessment staging system, a gastric mucosal staging system designed to provide an estimate of the risk of developing gastric cancer based on the degree of antral and corpus atrophy [13,14]. (See "Gastritis: Etiology and diagnosis" and "Metaplastic (chronic) atrophic gastritis" and "Risk factors for gastric cancer" and "Indications and diagnostic tests for Helicobacter pylori infection in adults" and 'Clinical and pathologic features' below.)

## TYPES OF GASTRIC POLYPS AND SPECIFIC MANAGEMENT

The management of gastric polyps and surveillance are specific to the underlying presentation, pathology, and malignant potential.

**Hyperplastic polyps** — Hyperplastic polyps account for approximately 75 percent of gastric polyps in geographic areas where *H. pylori* is common.

**Etiology** — Hyperplastic polyps result from hyper-regenerative epithelium in response to an underlying chronic inflammatory stimulus. They are therefore observed in the setting of chronic inflammatory conditions (eg, chronic atrophic gastritis), *H. pylori,* pernicious anemia, adjacent to ulcers and erosions, and at sites of gastroenterostomies [15].

**Clinical and pathologic features** — Males and females are equally affected. Hyperplastic polyps typically appear in mid to late adult life.

- **Clinical manifestations** – Hyperplastic polyps are usually asymptomatic and are discovered incidentally on upper endoscopy. Over time, polyps may remain stable, increase in size, or regress following *H. pylori* eradication [16,17]. Rare polyps that cause symptoms are most likely to present with gastrointestinal (GI) bleeding due to erosion of the surface epithelium. Bleeding is usually occult but occasionally overt [18,19]. In rare cases, intermittent obstruction may occur when pedunculated polyps in the antrum prolapse into or through the pylorus ( picture 2). (See "Gastric outlet obstruction in adults", section on 'Clinical manifestations'.)

- **Endoscopic features and pathology** – On upper endoscopy, hyperplastic polyps are smooth, dome-shaped, or stalked with an average size ranging from 0.5 to 1.5 cm. Hyperplastic polyps are often multiple and may develop in the antrum, body, fundus or cardia ( image 1) [4,20]. Microscopically, they are composed of elongated, dilated, and/or cystic, architecturally distorted, foveolar epithelium within an edematous, congested, and often actively and chronically inflamed lamina propria ( picture 3) [21,22]. Other features that may be seen include epithelial regenerative changes, stromal fibromuscular hyperplasia, dystrophic goblet cells, intestinal metaplasia, pyloric metaplasia (in corpus and fundic lesions), surface erosion, and ulceration.

- **Malignant potential** – Malignancy develops in hyperplastic polyps through a dysplasia/carcinoma sequence [3,23,24]. Between 1 and 20 percent of hyperplastic polyps have been reported to harbor foci of dysplasia [23-28]. The risk of malignancy in hyperplastic polyps is increased in polyps >1 cm and pedunculated in shape [23,29].

**Management** — Hyperplastic polyps measuring >0.5 cm should be resected completely [1,2]. In addition, the normal-appearing antral and corpus mucosa should be sampled to assess for

the presence of dysplasia and *H. pylori*. All patients with *H. pylori* should be eradicated [17]. (See 'Initial evaluation' above and "Treatment regimens for Helicobacter pylori in adults".)

In patients with dysplasia or carcinoma beyond the confines of the polyp, a subtotal gastrectomy or endoscopic mucosal resection should be performed. (See "Early gastric cancer: Epidemiology, clinical manifestations, diagnosis, and staging".)

- **Surveillance** – In patients with hyperplastic polyps without dysplasia or carcinoma, follow-up should be based on the cancer risk due to concurrent chronic atrophic gastritis and risk factors for gastric cancer.

  - In patients at high risk for gastric cancer (eg, Operative Link on Gastritis Assessment [OLGA] stage 3 or 4 with moderate diffuse or severe atrophy in the corpus or antrum usually accompanied by extensive intestinal metaplasia, migrants from areas with high gastric cancer incidence, family history of gastric cancer), we perform surveillance endoscopy at regular intervals (one to two years).

  - In patients at low risk for gastric cancer (eg, OLGA stage 1 or 2), we perform at least one follow-up endoscopy three to six months after therapy to confirm that *H. pylori* has been eradicated and that there are no new or residual polyps that warrant resection. (See "Early gastric cancer: Epidemiology, clinical manifestations, diagnosis, and staging" and "Metaplastic (chronic) atrophic gastritis" and "Risk factors for gastric cancer".)

**Fundic gland polyps** — In Western countries, where *H. pylori* infection has a low prevalence and proton pump inhibitor (PPI) use is common, fundic gland polyps are the most commonly encountered polyps.

**Etiology** — Most fundic gland polyps are sporadic. Fundic gland polyps may also occur in association with polyposis syndromes, familial adenomatous polyposis (FAP), MUTYH-associated polyposis (MAP), and gastric adenocarcinoma and proximal polyposis of the stomach (GAPPS) [30-32]. Fundic gland polyps occur in 20 to 100 percent of patients with FAP and 11 percent of patients with MAP [32,33]. (See "Clinical manifestations and diagnosis of familial adenomatous polyposis".)

Hypergastrinemia associated with a gastrinoma, Zollinger-Ellison syndrome, or long-term therapy with PPIs has been shown to be associated with fundic gland polyps [6,34-38]. In an observational study that included 599 patients undergoing endoscopy, long-term (≥5 years) PPI use was associated with a fourfold increased risk of fundic gland polyps, while short-term (<1 year) PPI therapy was not associated with increased risk [6]. Regression of fundic gland polyps following withdrawal of PPIs provides further support for this association [39].

The incidence of *H. pylori* infection is very low in patients with fundic gland polyps and infection may be protective [3]. In contrast with gastric adenomas and hyperplastic polyps which may regress with eradication of *H. pylori*, regression of fundic gland polyps has been reported following an *H. pylori* infection [35,40].

**Clinical and pathologic features** — Sporadic fundic gland polyps occur in females more often than in males and usually occur in middle age. Up to 40 percent of patients have multiple polyps. In FAP, men and women are affected equally; polyps develop at an earlier age (mean age 40 years), as compared with sporadic fundic gland polyps, and are multiple in more than 90 percent of cases.

- **Clinical manifestations** – Fundic gland polyps are usually asymptomatic and discovered incidentally at endoscopy. Only in rare cases can they reach a size large enough to cause obstruction or symptoms of abdominal pain or vomiting.

- **Endoscopic features and pathology** – Fundic gland polyps are typically small (0.1 to 0.8 cm), hyperemic, sessile, and have a smooth surface contour ( picture 1A-B). They occur exclusively in the gastric corpus. On narrow band imaging, fundic gland polyps have a honeycomb appearance with dense vasculature, a nonspecific pattern that also can be seen in hyperplastic polyps.

  On pathology, fundic gland polyps are composed of normal gastric corpus-type epithelium, arranged in a disorderly and/or microcystic configuration ( picture 4 and picture 5) [5,9,34]. Microcysts are characteristic, and may be lined by any of the normal cell types found in the gastric corpus. The glandular compartment typically reveals distorted architecture with irregular gland buds, tortuous glands, or irregular stellate glandular configurations. Inflammation is usually minimal. Mild hyperplasia of the muscularis mucosa may occur in a pericystic configuration. These lesions probably arise from proliferation and differentiation of aberrantly located proliferative cells in the stem cell compartment of the corpus epithelium.

- **Malignant potential** – Somatic *APC* gene mutations have been detected in over 70 percent of syndromic fundic gland polyps without dysplasia, but in less than 10 percent of sporadic lesions [41]. Sporadic fundic gland polyps and those associated with PPI use have virtually no malignant potential, but may rarely show dysplasia [6,36,38,42]. Between 30 to 50 percent of fundic gland polyps in patients with FAP show dysplasia, which is typically low grade [43-45]. However, fundic gland polyps, in contrast with adenomas in patients with FAP, rarely progress to cancer [46]. (See "Clinical manifestations and diagnosis of

familial adenomatous polyposis", section on 'Extracolonic manifestations' and 'Gastric adenomas' below.)

**Management** — Fundic gland polyps are frequently multiple and biopsies of one or more representative polyps is sufficient. The remaining polyps should be carefully inspected on endoscopy and any lesion that appears significantly different should be biopsied and, if possible, resected. Fundic gland polyps ≥1 cm in diameter, polyps that are ulcerated or located in the antrum should be resected to confirm the diagnosis and rule out dysplasia or neoplasia.

The possibility of a familial polyposis syndrome should be considered in patients with ≥20 polyps, fundic gland polyps in the antrum, young onset fundic polyps (prior to age 40 years), or concurrent duodenal adenomas, and a colonoscopy should be performed.

In patients with sporadic fundic gland polyps attributed to PPI use, whenever possible, we discontinue PPIs in patients with ≥20 polyps or polyps >1 cm. Hypergastrinemia in these patients suggests either the presence of a gastrinoma (Zollinger-Ellison syndrome) or more commonly PPI-induced hypochlorhydria, which if present is further reason to stop or reduce the PPI dose. If acid suppression is needed for the management of gastroesophageal reflux disease (GERD) and patients fail a trial of an H2 receptor blocker, we consider using a different PPI at the lowest effective dose [47].

- **Surveillance** – Regular surveillance by upper endoscopy is not routinely recommended for sporadic fundic gland polyps without dysplasia as progression to gastric cancer is rare. In patients with an established diagnosis of a familial polyposis syndrome (eg, FAP), surveillance with upper endoscopy is recommended for polyps in the upper GI tract [43,48-50]. (See "Familial adenomatous polyposis: Screening and management of patients and families", section on 'Gastric polyps'.)

**Gastric adenomas** — Gastric adenomas, or raised intraepithelial neoplasia, are the most common gastric neoplastic polyp [51-53]. Gastric adenomas account for 6 to 10 percent of gastric polyps [4,20].

**Etiology** — Gastric adenomas typically occur in a background of chronic and/or atrophic gastritis but can occur in stomachs without associated inflammatory changes as well. Gastric adenomas are much less common than fundic gland polyps in patients with FAP; they are typically isolated and located in the antrum and are associated with a relatively low but real risk of progression to cancer. (See "Clinical manifestations and diagnosis of familial adenomatous polyposis", section on 'Extracolonic manifestations'.)

**Clinical and pathologic features** — Sporadic gastric adenomas occur equally in men and women and are most commonly seen in the sixth or seventh decade.

- **Clinical manifestations** – Most gastric adenomas are asymptomatic. Polyps that cause symptoms are most likely to present with GI bleeding (usually occult but occasionally overt) or rarely obstruction. (See "Gastric outlet obstruction in adults", section on 'Clinical manifestations'.)

- **Endoscopic features and pathology** – There are four general categories of gastric adenomas. These include the intestinal type, foveolar type, pyloric gland type, and the oxyntic type [54]. Adenomas may be flat or polypoid, and are usually <2 cm in size ( picture 6). Adenomas are usually solitary. Most are found in the antrum (intestinal and foveolar), but some occur in the corpus and cardia (pyloric and oxyntic). The narrow band imaging features of gastric adenomas have not been well defined.

  Microscopically, intestinal type adenomas are similar to typical colonic adenomas ( picture 7). They may be tubular, tubulovillous, or villous (papillary), are sessile or stalked, and occasionally reach large sizes (up to 15 cm). They consist of dysplastic columnar cells with striated borders, often intermixed with dysplastic goblet cells, Paneth cells, parietal cells, or endocrine cells. The dysplastic cells show elongated, pencil-shaped nuclei with hyperchromasia, clumped chromatin, and pseudostratification. These lesions are often associated with underlying chronic gastritis with intestinal metaplasia. Foveolar adenomas are composed of dysplastic foveolar-type epithelium rich in cytoplasmic mucin. Pyloric adenomas consist of neoplastic pyloric type glands and the oxyntic type if composed of columnar cells with chief cell, parietal cell, or both, differentiation, and have a high rate of submucosal invasion.

  The most common type of adenomas is the intestinal type, which is often associated with chronic gastritis and gastric cancer. In foveolar-type gastric adenomas, goblet and Paneth cells are absent, but the epithelium contains gastric foveolar cells with mucin. Although foveolar-type gastric adenomas are associated with familial adenomatous polyposis (FAP), most polyps in patients with FAP are fundic gland type. Pyloric gland-type adenomas are pyloric gland lined and often contain cystically dilated tubules with no goblet cells. Pyloric gland adenomas are associated with chronic gastritis and frequently contain areas of associated mucosal dysplasia [55,56]. Most are present in the gastric fundus, but they have been described throughout the stomach as well as in the duodenum, bile ducts, and gall bladder [57]. Oxyntic gland adenomas are typically found deep in the mucosa of the proximal stomach and are lined by mucus neck, chief, and parietal cells [58]. The different

types of gastric polyp have different patterns of staining for MUC2, MUC5AC, and MUC6 [54].

- **Malignant potential** – It is estimated that 8 to 59 percent of adenomas are associated with synchronous gastric carcinomas [59]. The presence of invasive carcinoma in an intestinal adenoma correlates with increasing size, villous contour, and the degree of dysplasia [23,60]. The risk of malignancy is higher in flat adenomas [61]. High-grade dysplasia has been identified in close proximity to a high proportion (40 to 100 percent) of early gastric cancers [62]. The risk in the various pathologic subgroups are different. Intestinal adenomas pose the highest risk, whereas foveolar polyps have a negligible risk of neoplastic progression. Pyloric and oxyntic lesions also have a high risk of synchronous and metachronous cancer, but the studies on progression are limited.

  Dysplasia is a precursor of invasive adenocarcinoma. Patients with high-grade dysplasia, in general, have a higher risk of association or progression as compared with patients with low-grade dysplasia [63,64]. A nationwide cohort study in the Netherlands evaluated the progression of dysplasia to frank gastric cancer in 165 patients with high-grade dysplasia and 270 patients with low-grade dysplasia. Gastric cancer was diagnosed within 1, 5, and 10 years from the initial diagnosis in 25, 30, and 33 percent of patients with high-grade dysplasia and 2, 3, and 4 percent with low-grade dysplasia, respectively [63].

  The risk of gastric cancer in patients with FAP is discussed in detail, separately. (See "Clinical manifestations and diagnosis of familial adenomatous polyposis", section on 'Extracolonic manifestations'.)

**Management** — Given the increased risk of gastric cancer, all gastric adenomas should be resected. This can usually be accomplished endoscopically, but on occasion surgery may be required for lesions that contain invasive carcinoma or in patients with multiple adenomas.

Because of the association of gastric dysplasia with synchronous gastric carcinomas, the remainder of the stomach must be examined carefully [13,14]. In addition, as adenomatous polyps are associated with atrophic gastritis, the normal-appearing antral and corpus mucosa should be sampled to assess the stage of gastritis and, thus, cancer risk. All patients should be tested for active *H. pylori* infection and, if present, the infection should be eradicated. (See "Treatment regimens for Helicobacter pylori in adults" and "Early gastric cancer: Epidemiology, clinical manifestations, diagnosis, and staging" and 'Initial evaluation' above.)

- **Surveillance** – We perform an upper endoscopy for surveillance one year after initial resection of adenomatous gastric polyps to assess recurrence at the prior excision site,

new or previously missed polyps, confirm eradication of *H. pylori*, and/or detect an early carcinoma [1].

In individuals at high risk for gastric cancer (eg, migrants from areas with high gastric cancer incidence, family history of gastric cancer, OLGA stage 3 or 4 with moderate diffuse or severe atrophy in the corpus or antrum usually accompanied by extensive intestinal metaplasia, patients with FAP), surveillance is continued indefinitely. (See "Risk factors for gastric cancer" and "Familial adenomatous polyposis: Screening and management of patients and families", section on 'Gastric polyps'.)

**Gastric neuroendocrine tumors** — Gastric neuroendocrine tumors are mostly derived from enterochromaffin-like (ECL) cells, however, some are somatostatin, gastrin, or serotonin producing.

**Etiology** — Gastric neuroendocrine tumors are generally subdivided into types 1 to 3 as they have different etiologies, biologic behavior, and prognoses [65-68]. Type 1 tumors represent 70 to 80 percent of all gastric neuroendocrine tumors. They are associated with prolonged hypergastrinemia typically resulting from autoimmune (corpus-restricted) atrophic gastritis. Type 2 gastric neuroendocrine tumors account for 5 to 8 percent of gastric neuroendocrine tumors and result from prolonged hypergastrinemia from a gastrin-secreting tumor. Type 3 neuroendocrine tumors are sporadic and account for 20 percent of gastric neuroendocrine tumors. (See "Clinical characteristics of well-differentiated neuroendocrine (carcinoid) tumors arising in the gastrointestinal and genitourinary tracts".)

**Clinical and pathologic features**

- **Clinical manifestations** – Type 1 gastric neuroendocrine tumors are found more commonly in older adults, particularly women, with atrophic gastritis and often are associated with pernicious anemia. Type 2 gastric neuroendocrine tumors are frequently detected as part of the work-up for MEN-1 syndrome or for Zollinger–Ellison syndrome in patients who present with peptic ulcer disease, abdominal pain, diarrhea, or bleeding. Type 3 tumors can be associated with "atypical carcinoid syndrome." (See "Multiple endocrine neoplasia type 1: Clinical manifestations and diagnosis" and "Clinical features of carcinoid syndrome", section on 'Gastric NET variant syndrome' and "Causes and pathophysiology of vitamin B12 and folate deficiencies", section on 'Pernicious anemia'.)

- **Endoscopic features and pathology** – Gastric neuroendocrine tumors associated with hypergastrinemia (types 1 and 2) are usually multiple, broad based, firm yellowish lesions in the fundus and body of the stomach and rarely measure >2 cm ( picture 8). In patients with MEN-1 syndrome, the gastric mucosa is normal or mildly inflamed, but not atrophic.

The fundic mucosa of patients with Zollinger–Ellison syndrome often is hypertrophic, with long densely packed oxyntic glands and no significant inflammation. Type 3 tumors occur in the antrum and are usually single. (See "Clinical characteristics of well-differentiated neuroendocrine (carcinoid) tumors arising in the gastrointestinal and genitourinary tracts".)

The pathologic diagnosis is usually established by recognizing the characteristic cytologic and architectural pattern of the cells, combined with immunohistochemical reactivity for chromogranin and/or synaptophysin [69]. Electron microscopy may be useful to confirm the presence of neurosecretory granules in the cytoplasm of tumor cells. There are four histologic architectural patterns in gastric neuroendocrine tumors, but it is common to find a mixture of these patterns in any particular lesion: solid or insular; trabecular; glandular; and undifferentiated, or diffuse ( picture 9) [70-72]. Most gastric neuroendocrine tumors have a very prominent, solid nest or insular pattern, particularly those that arise from enterochromaffin-like cells. Cytologically, tumors show a homogeneous, regular population of round to oval-shaped cells with round to oval nuclei containing a characteristic stippled or speckled chromatin pattern. Gastric neuroendocrine tumors that develop in association with the Zollinger-Ellison syndrome or pernicious anemia may be associated with hyperplastic or dysplastic endocrine growths in the adjacent gastric mucosa [73].

- **Malignant potential** – Type 1 and 2 gastric neuroendocrine tumors usually have an indolent course. Type 3 is the most aggressive; local or hepatic metastases are present in up to 65 percent of patients who undergo resection [66,68] (see "Clinical characteristics of well-differentiated neuroendocrine (carcinoid) tumors arising in the gastrointestinal and genitourinary tracts"). Of course, frankly malignant neuroendocrine tumors (carcinoma) of the small cell and large cell type, although much less common, do occur in the stomach as well.

**Management** — For type 1 and 2 gastric carcinoids smaller than 1 to 2 cm, endoscopic resection is the treatment of choice. In patients with multiple progressive tumors, antrectomy should be considered to remove the gastrin stimulus [74]. Sporadic (type 3) gastric carcinoids are treated by partial or total gastrectomy with local lymph node resection. The management of gastric neuroendocrine tumors is discussed in detail, separately. (See "Staging, treatment, and post-treatment surveillance of non-metastatic, well-differentiated gastrointestinal tract neuroendocrine (carcinoid) tumors", section on 'Stomach'.)

- **Surveillance** – Consistent with the National Comprehensive Cancer Network [75] recommendations following treatment of type 1 and 2 gastric neuroendocrine tumors ≤2

cm, we recommend history and physical examination with upper endoscopy every 6 to 12 months for three years and annually thereafter; imaging studies only as clinically indicated. The post-treatment surveillance for gastric neuroendocrine tumors is discussed in detail, separately. (See "Staging, treatment, and post-treatment surveillance of non-metastatic, well-differentiated gastrointestinal tract neuroendocrine (carcinoid) tumors", section on 'Post-treatment follow-up'.)

**Inflammatory fibroid polyps** — Inflammatory fibroid polyps are rare benign lesions that represent less than 0.1 percent of all gastric polyps. Inflammatory fibroid polyps are mesenchymal tumors that typically, but not exclusively, arise in the submucosa of the GI tract, most often the stomach and small intestine. Immunohistochemical staining suggests that these polyps may have a dendritic cell origin [76].

**Etiology** — Although familial clustering of inflammatory fibroid polyps has been reported, the etiology of inflammatory fibroid polyps is largely unknown [77]. Most cases are sporadic, but some are familial, associated with germline mutations in platelet-derived growth factor receptor alpha (*PDGFRA)*. Activating mutations in this gene are also common in sporadic cases and most often in ones from the small intestine.

**Clinical and pathologic features**

- **Clinical manifestations** – Most inflammatory fibroid polyps are asymptomatic, but larger polyps have been reported to cause abdominal pain, early satiety, anemia, and gastric outlet obstruction ( picture 10). (See "Gastric outlet obstruction in adults", section on 'Clinical manifestations'.)

- **Endoscopic features and pathology** – On endoscopy, inflammatory fibroid polyps are usually firm, solitary, sessile or pedunculated, and often ulcerated. On endoscopic ultrasound, they have the appearance of a hypoechoic homogeneous lesion with an indistinct margin, located within the second or third layer with an intact fourth layer.

  On histology, these polyps are characterized by submucosal proliferations of spindle cells with vessels surrounded by a characteristic circumferential deposition of fibroblasts giving it an onion skin appearance, and an inflammatory infiltrate with a predominance of eosinophils. By immunohistochemistry, these lesions are normally positive for CD34 and PDGFRA.

- **Malignant potential** – Inflammatory fibroid polyps are considered benign. However, in one study of 23 inflammatory fibroid polyps, 70 percent were found to have gain-of-

function mutations in the platelet-derived growth factor receptor alpha polypeptide gene, similar to those found in CD117-negative GI stromal tumors [78].

**Management** — Following resection, inflammatory fibroid polyps typically do not recur, and surveillance is not recommended.

## INFORMATION FOR PATIENTS

UpToDate offers two types of patient education materials, "The Basics" and "Beyond the Basics." The Basics patient education pieces are written in plain language, at the 5th to 6th grade reading level, and they answer the four or five key questions a patient might have about a given condition. These articles are best for patients who want a general overview and who prefer short, easy-to-read materials. Beyond the Basics patient education pieces are longer, more sophisticated, and more detailed. These articles are written at the 10th to 12th grade reading level and are best for patients who want in-depth information and are comfortable with some medical jargon.

Here are the patient education articles that are relevant to this topic. We encourage you to print or e-mail these topics to your patients. (You can also locate patient education articles on a variety of subjects by searching on "patient info" and the keyword(s) of interest.)

- Basics topics (see "Patient education: Stomach polyps (The Basics)")

## SUMMARY AND RECOMMENDATIONS

- Most gastric polyps are asymptomatic and are discovered incidentally. Hyperplastic polyps are the most prevalent polyps in regions where *H. pylori* infection is common. In contrast, in Western countries, where *H. pylori* infection has a lower prevalence and proton pump inhibitor (PPI) use is common, fundic gland polyps are more prevalent. (See 'Epidemiology' above.)

- In patients with small solitary polyps, we recommend that polyps be biopsied and resected, if possible, for histologic characterization (**Grade 1A**). Polypectomy should be performed for all known neoplastic polyps and for all polyps ≥1 cm in diameter, as biopsies alone cannot exclude foci of high-grade dysplasia or early gastric cancer. In patients with multiple polyps, the largest polyp should be excised and representative biopsies obtained from the remaining polyps.

The normal-appearing antral and corpus mucosa should be sampled to rule out dysplasia occurring in the background of metaplastic atrophic gastritis and infection with *H. pylori.* We suggest *H. pylori* eradication therapy in those who test positive. (**Grade 2C**). (See 'Initial evaluation' above.)

- Fundic gland polyps may occur sporadically, in association with polyposis syndromes, or in response to chronic PPI therapy. They have very low to no malignant potential when associated with PPI use or occurring sporadically. Fundic gland polyps associated with polyposis syndromes have been associated with dysplasia but progression to malignancy is rare. Fundic gland polyps ≥1 cm in diameter, polyps with ulceration, or located in the antrum should be resected. In patients with multiple (≥20) or large (≥1 cm) sporadic fundic gland polyps, withdrawal of the PPI should be considered. (See 'Fundic gland polyps' above.)

- Hyperplastic polyps occur in association with *H. pylori*-related atrophic gastritis, pernicious anemia, and adjacent to ulcers and erosions. They are generally benign, but have some malignant potential. Hyperplastic polyps >0.5 cm should be resected completely. Surveillance with upper endoscopy should be performed based on the cancer risk due to concurrent chronic atrophic gastritis and other risk factors for gastric cancer. (See 'Hyperplastic polyps' above.)

- Most gastric adenomas typically form in atrophic gastric mucosa and may also be seen in association with familial polyposis syndromes. Most gastric adenomas are asymptomatic. Polyps that cause symptoms are most likely to present with gastrointestinal bleeding or rarely obstruction. Since most types of gastric adenomas are a precursor lesion of gastric adenocarcinoma, we recommend resection of all gastric adenomas endoscopically or surgically if necessary (**Grade 1A**). We perform an upper endoscopy for surveillance one year after initial resection of adenomatous gastric polyps. In individuals at high risk for gastric cancer, surveillance is continued indefinitely. (See 'Gastric adenomas' above.)

- Gastric neuroendocrine tumors may be associated with hypergastrinemia (type 1 and 2) or may occur sporadically (type 3). In contrast with patients with type 3 gastric neuroendocrine tumors who may have metastases at the time of resection, patients with type 1 and 2 gastric neuroendocrine tumors usually have an indolent course. For type 1 and 2 gastric neuroendocrine tumors smaller than 1 to 2 cm, endoscopic resection is the treatment of choice. Type 3 tumors are treated by partial or total gastrectomy with local lymph node resection. (See 'Gastric neuroendocrine tumors' above and "Staging, treatment, and post-treatment surveillance of non-metastatic, well-differentiated gastrointestinal tract neuroendocrine (carcinoid) tumors".)

- Inflammatory fibroid polyps are uncommon lesions that represent less than 0.1 percent of all gastric polyps. Following resection, inflammatory fibroid polyps typically do not recur, and surveillance is not recommended. (See 'Inflammatory fibroid polyps' above.)



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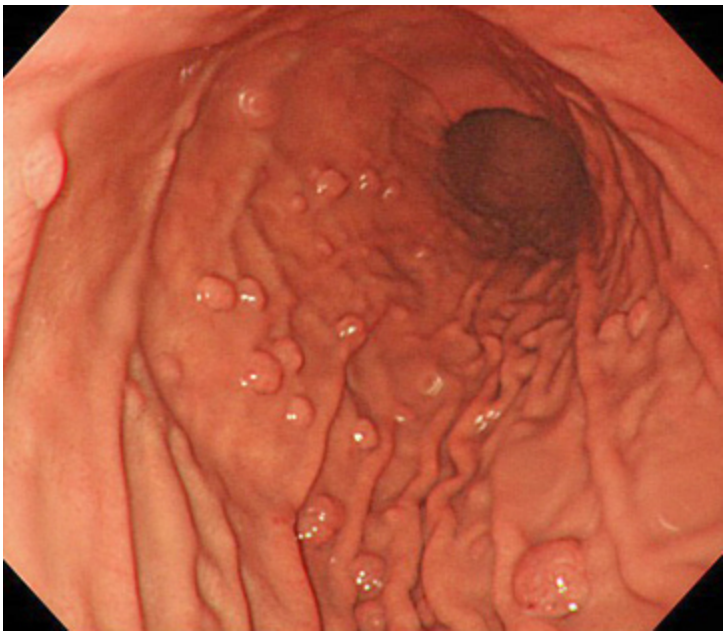
Topic 2518 Version 22.0

## GRAPHICS

### Gastric fundic gland polyp



Endoscopic view of the body of the stomach looking toward the antrum and pylorus showing numerous polypoid lesions, which on biopsy were proven to be fundic gland polyps.

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*Courtesy of Akira Horiuchi, MD.*

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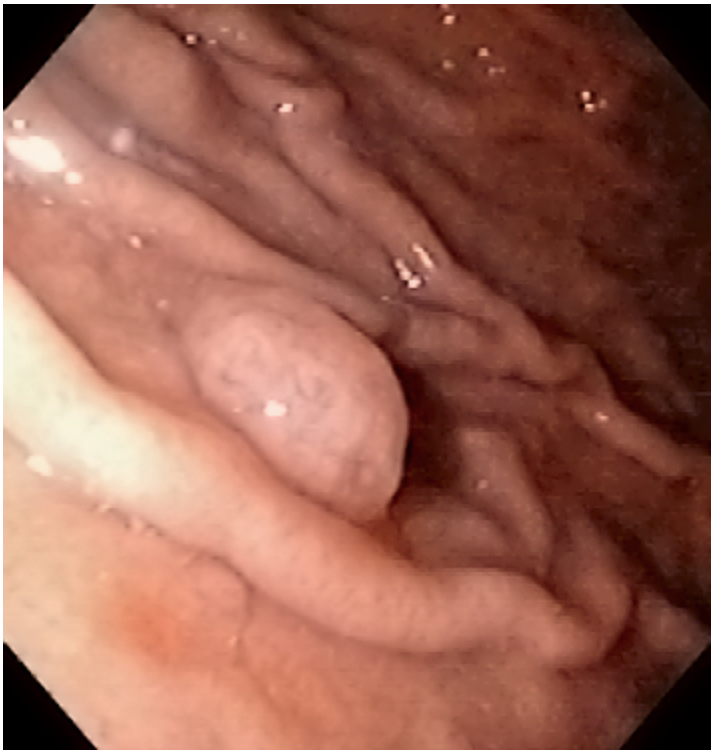
Graphic 78092 Version 1.0

## Fundic gland polyp



Upper endoscopy showing a small polyp in the gastric fundus proven to be a fundic gland polyp after it was removed.

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*Courtesy of David Y Graham, MD.*

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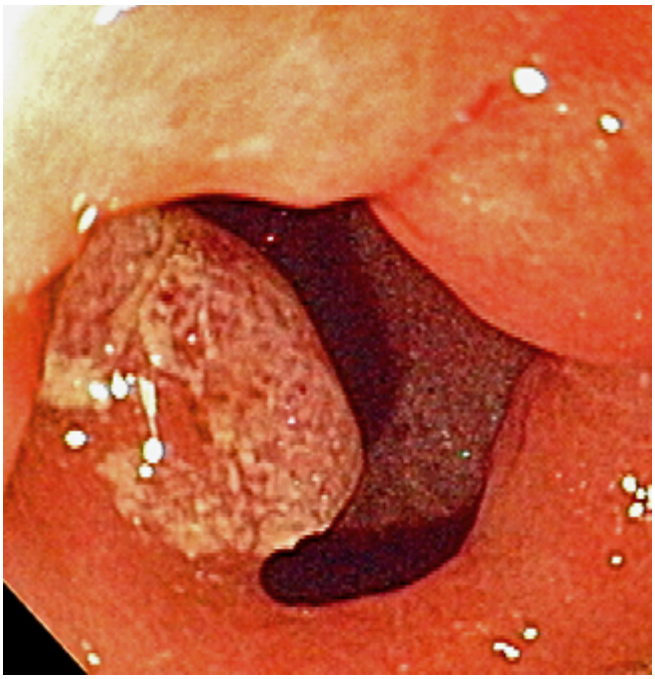
Graphic 58144 Version 1.0

## Gastric hyperplastic polyp prolapsing through the pylorus



Endoscopic view of a hyperplastic gastric polyp on a short stalk near the pylorus. The polyp prolapsed into the pylorus and duodenal bulb with peristalsis.

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*Courtesy of David Y Graham, MD.*

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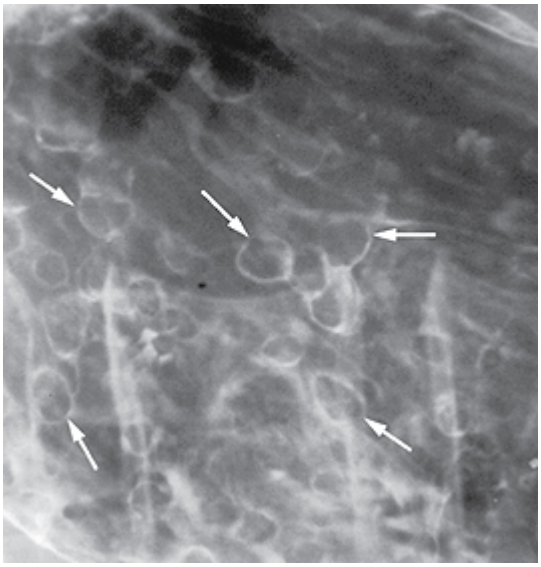
Graphic 50167 Version 1.0

## Hyperplastic gastric polyps



Double contrast upper gastrointestinal study shows multiple well-circumscribed uniformly sized hyperplastic polyps arising from the gastric mucosa (arrows).

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*Courtesy of Jonathan Kruskal, MD, PhD.*

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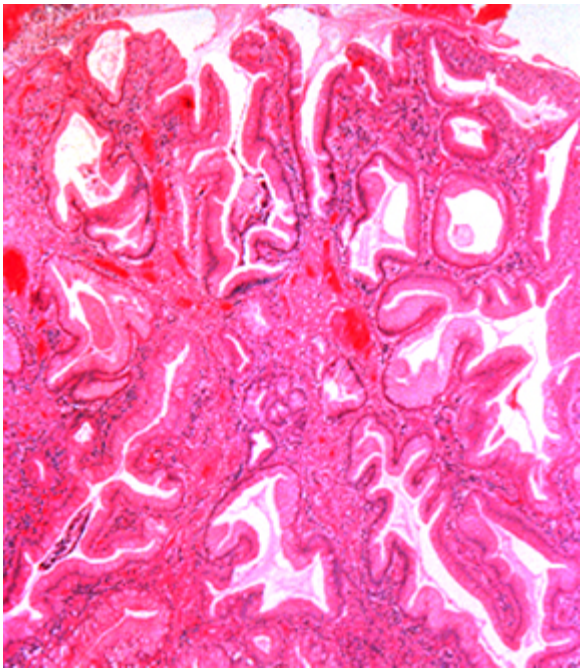
Graphic 79352 Version 2.0

## Hyperplastic gastric polyp

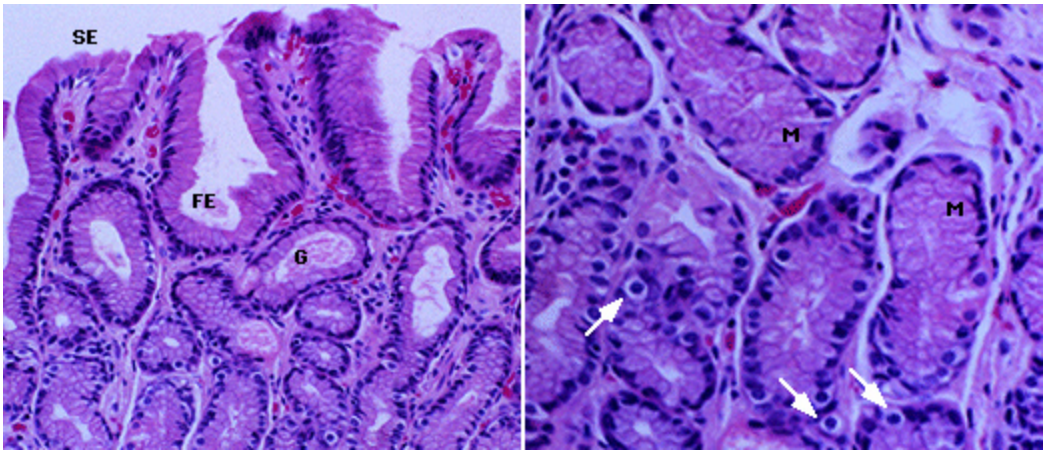


Low power view of a hyperplastic gastric polyp which is composed of elongated, dilated, and architecturally distorted foveolar epithelium within an edematous, congested, and inflamed lamina propria.

*Courtesy of Robert Odze, MD.*

Graphic 72171 Version 1.0

## Normal gastric antrum




Left panel: Normal surface (SE) and foveolar epithelium (FE) and glands (G). Right panel: Higher power view of the glands shows mucous cells (M) and gastrin-secreting endocrine cells (arrows).

*Courtesy of Robert Odze, MD*

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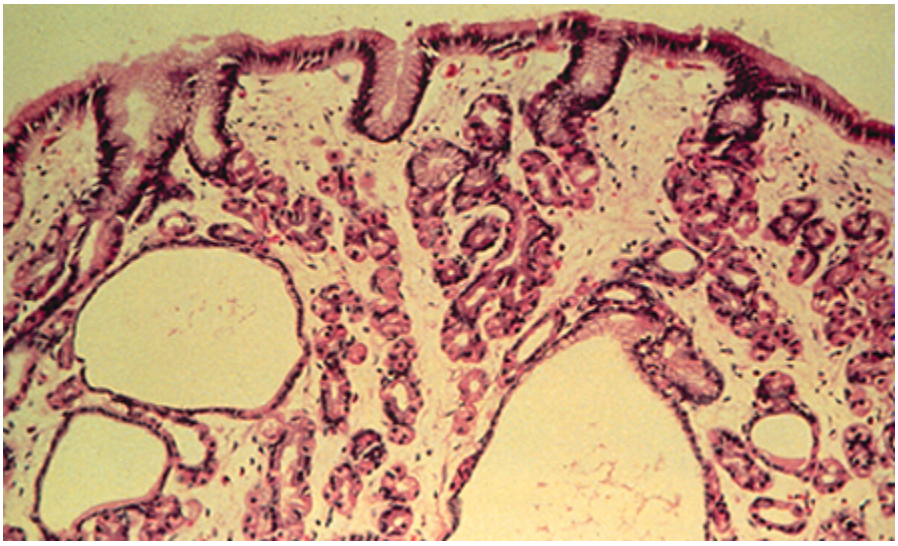
Graphic 79895 Version 1.0

## Fundic gland polyp



Low power view of a fundic gland polyp shows normal gastric corpus-type epithelium, arranged in a disorderly and microcystic configuration.

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*Courtesy of Robert Odze, MD.*

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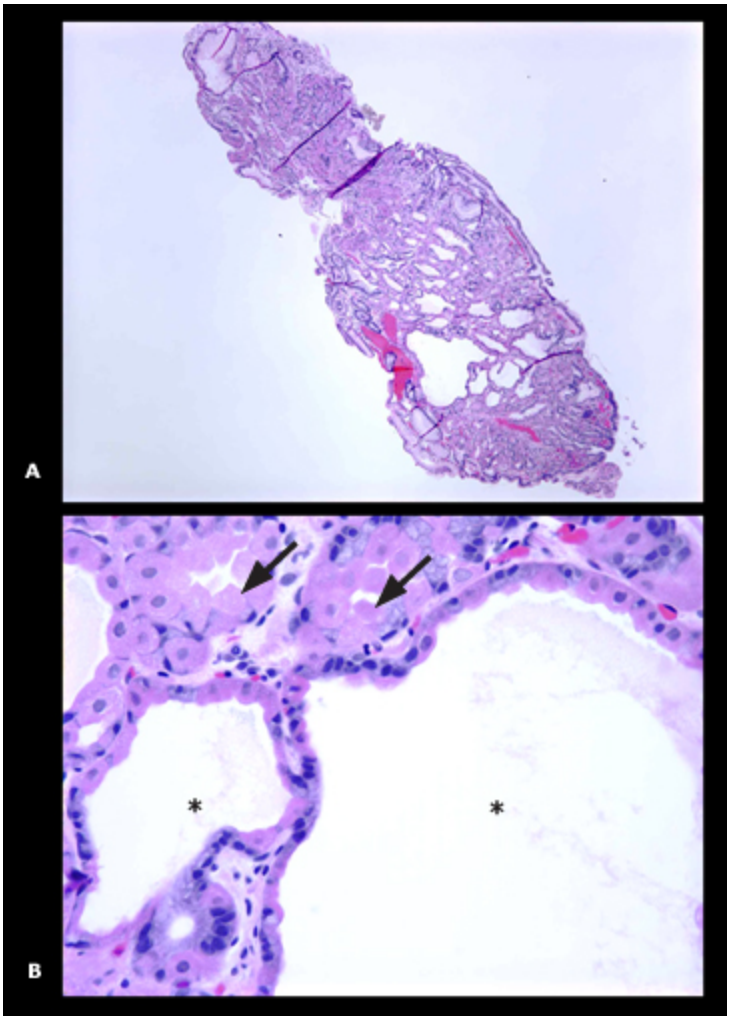
Graphic 66607 Version 1.0

## Fundic gland polyp




Biopsy of a gastric nodule in the fundus showing the classic appearance of a fundic gland polyp. At low power (upper panel), there are variably dilated glands with an unremarkable surface/foveolar zone. At high power (lower panel), the dilated glands (asterisks) are lined by the cells indigenous to corpus mucosa. Normal corpus glands are shown (arrows).

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*Courtesy of Donald Antonioli, MD.*

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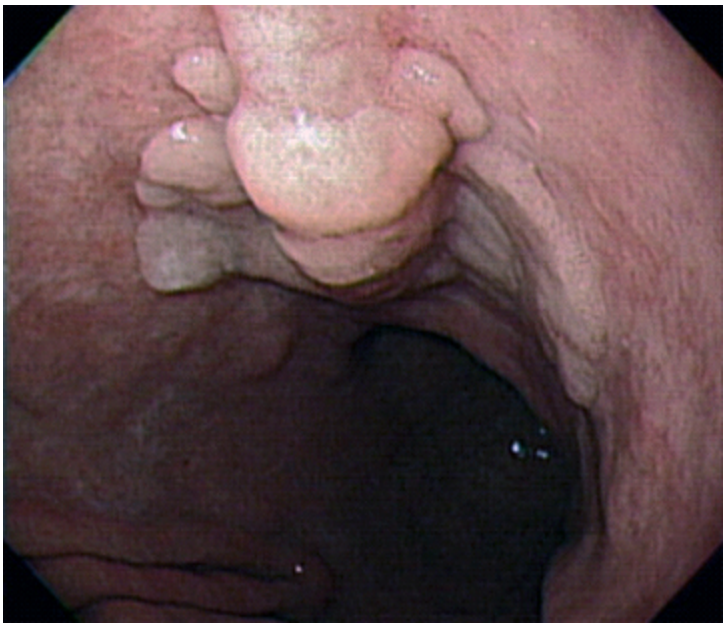
Graphic 77794 Version 1.0

## Gastric adenoma (noninvasive intraepithelial neoplasia)



Large gastric adenoma on the lesser curve of the distal corpus.

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*Courtesy of Jin-Seok Jang, MD.*

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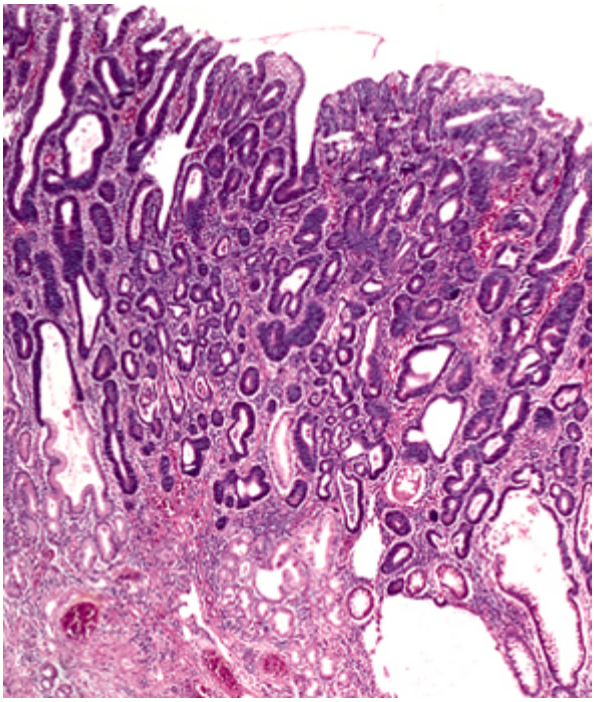
Graphic 64653 Version 1.0

## Gastric adenoma



Medium power light micrograph of a small sessile adenoma located in the gastric antrum. The darkly-staining dysplastic epithelium is seen on the superficial layer while residual antral epithelium with a few cystic tubules is seen in the basal half.

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*From Lewin KJ, Appelman HD. Tumors of the esophagus and stomach. Atlas of tumor pathology (electronic fascicle), Third series, fascicle 18, 1996, Washington, DC. Armed Forces Institute of Pathology.*

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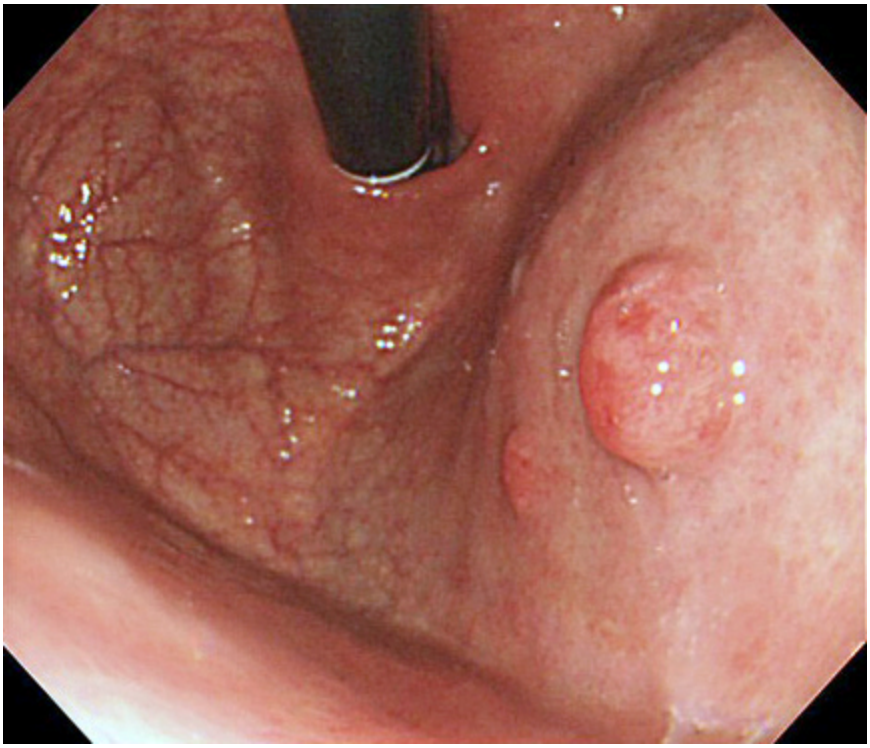
Graphic 74367 Version 2.0

## Gastric carcinoids



Gastric carcinoids in the proximal gastric body.

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*Courtesy of Akira Horiuchi, MD.*

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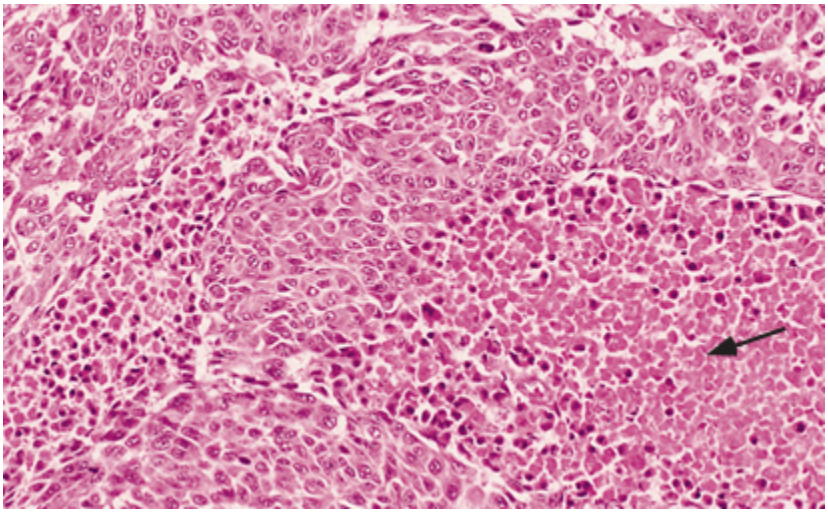
Graphic 77477 Version 1.0

## Gastric neuroendocrine carcinoma (Grade 2)*



Low power view of a gastric carcinoid reveals sheets of poorly differentiated tumors cells with areas of necrosis (arrow).

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* All neuroendocrine tumors of the GI tract, including gastric carcinoids, are graded as grade 1, 2, or 3 (large cell or small cell type neuroendocrine carcinoma) based on the mitotic count and/or the Ki67 proliferation index.

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*From Lewin KJ, Appelman HD. Tumors of the esophagus and stomach. Atlas of tumor pathology (electronic fascicle), Third series, fascicle 18, 1996, Washington, DC. Armed Forces Institute of Pathology.*

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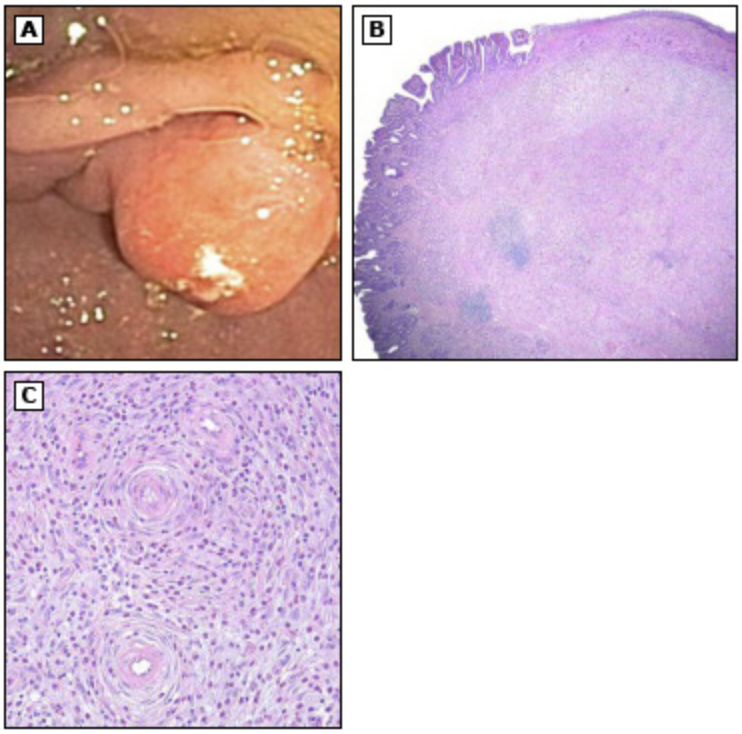
Graphic 63323 Version 3.0

## Inflammatory fibroid polyp




(A) Endoscopic view of an inflammatory fibroid polyp in the antrum showing a firm, well-circumscribed submucosal lesion.

(B) Histologically, a flattened, often eroded, gastric epithelium lines a compact aggregate of fibrous tissue mixed with inflammatory cells.

(C) Vessels usually are surrounded by a characteristic circumferential deposition of fibroblasts (onion skin), and the stroma contains myriad eosinophils.

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Graphic 91612 Version 1.0

## Contributor Disclosures

**Varocha Mahachai, MD** No relevant financial relationship(s) with ineligible companies to disclose. **David Y Graham, MD** Grant/Research/Clinical Trial Support: RedHill Biopharma [Helicobacter pylori, mycobacteria as a possible cause of Crohn disease]. All of the relevant financial relationships listed have been mitigated. **Robert D Odze, MD, FRCPC** Consultant/Advisory Boards: CDx Diagnostics [Barretts esophagus]. All of the relevant financial relationships listed have been mitigated. **Mark Feldman, MD, MACP, AGAF, FACG** No relevant financial relationship(s) with ineligible companies to disclose. **Shilpa Grover, MD, MPH, AGAF** No relevant financial relationship(s) with ineligible companies to disclose.

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