Gastric intestinal metaplasia

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INTRODUCTION

Globally, gastric cancer is the fourth leading cause of cancer mortality and the leading cause of infection-associated cancers [1]. Gastric intestinal metaplasia (GIM) is an intermediate precancerous gastric lesion in the gastric cancer cascade of chronic gastritis, atrophic gastritis, intestinal metaplasia (IM), dysplasia, and adenocarcinoma [2]. Although the risk of gastric cancer is increased in patients with GIM, the absolute risk is modest. Specific subsets of patients with GIM may be at higher risk for progression.

This topic will review the epidemiology, natural history, diagnosis, and management of distal (noncardia) GIM. Screening for gastric cancer and the management of IM at the gastric cardia (located immediately distal to the squamocolumnar junction and proximal to the oxyntic mucosa) and its distinction from Barrett's esophagus are discussed in detail separately. (See "Barrett's esophagus: Epidemiology, clinical manifestations, and diagnosis", section on 'Intestinal metaplasia at GEJ' and "Early gastric cancer: Clinical features, diagnosis, and staging" and "Early gastric cancer: Management and prognosis" and "Gastric cancer screening".)

TERMINOLOGY

GIM is defined as the replacement of the superficial foveolar and deeper glandular

epithelium in the oxyntic or antral mucosa by intestinal epithelium [3].

Classification — Several GIM classification systems are in use [4-7].

- GIM topographic extent – Based on the extent of gastric involvement on histology, GIM is classified as extensive versus limited [4,5,8,9]:
  - Extensive IM – GIM involving the corpus and either the antrum and/or incisura angularis. GIM of the corpus alone is sometimes considered a surrogate for extensive metaplasia, as antral metaplasia is usually also present, but may have been missed on biopsy given the patchy distribution.
  - Limited IM – GIM involving the antrum or incisura.
- GIM histologic subtypes
  - Based on the histology with hematoxylin and eosin staining (H&E stain):
    - Complete IM is defined by the presence of small intestinal-type mucosa with goblet cells that secrete sialomucins, a brush border, and eosinophilic enterocytes (picture 1).
    - Incomplete IM is defined by the presence of colonic-type epithelium with multiple, irregular mucin droplets of variable size in the cytoplasm and absence of a brush border (picture 2).
  - Based on mucin expression – IM has also been subtyped based on mucin expression, but its use is mainly limited to research settings. In IM, the original gastric mucins, which have a neutral pH, are replaced by acid mucins, which may be sialic or sulfated. Using a combination Alcian blue and high-iron diamine stain, GIM is classified based on the types of mucins expressed and morphology into the following types [6]:
    - Type I (complete) IM expresses only sialomucins
    - Type II (incomplete) IM expresses a mixture of gastric (neutral) mucins and intestinal sialomucins
    - Type III (incomplete) IM expresses sulfomucins
  - Combined periodic acid Schiff-AB staining may also be used to discriminate

between normal epithelium and IM [10]. It is unclear whether this approach adds to risk stratification beyond the improved identification of incomplete IM [8]. Immunohistochemical stains may also be used for GIM confirmation (eg, MUC2).

- Updated Sydney system – The operative link for gastritis assessment (OLGA) and operative link on GIM assessment (OLGIM) staging systems are used to evaluate the severity and extent of atrophy and IM. The severity of gastric atrophy and IM are graded in the antrum/incisura and corpus. Stages III and IV represent more severe extensive disease and correlate with a higher risk of progression (table 1 and table 2) [5,11,12]. (See "Metaplastic (chronic) atrophic gastritis", section on 'Diagnosis'.)

EPIDEMIOLOGY AND PATHOGENESIS

Estimates of the global prevalence of GIM vary widely, reflecting the variations in gastric cancer incidence [13-16]. GIM prevalence in high-incidence regions (Eastern Asia, Eastern Europe, South America) ranges between 19 to 24 percent [13]. In low gastric cancer incidence regions such as the United States and northern Europe, GIM prevalence is approximately 5 percent [13]. Prevalence of GIM increases with age and is higher in areas with high prevalence of *Helicobacter pylori* (*H. pylori*), in men, and in current smokers [14,17-19]. (See "Epidemiology of gastric cancer" and "Gastric cancer: Pathology and molecular pathogenesis", section on 'Intestinal metaplasia'.)

GIM is a premalignant stage in the gastric cancer cascade through a series of well-defined and recognizable precursors [20]. The gastric adenocarcinoma multistage model, the "Correa cascade," suggests that a combination of host genetic factors and responses, *H. pylori* genomics, with modulation by dietary and environmental factors, predispose to early pan-gastric mucosal inflammation, which in turn may progress sequentially to gastric atrophy, IM, dysplasia, and adenocarcinoma [21-23]. This model is most applicable to the intestinal subtype of gastric cancer. While there may be reversal of gastric atrophy with *H. pylori* eradication, GIM is considered as a "point of no return" in the multistage model, although this remains an ongoing debate.

The metaplastic foci often first appear at the antrum-corpus junction, and frequently at the incisura angularis. As the process advances, the foci enlarge and coalesce, extending to the neighboring mucosa in both the antrum and the corpus [8]. As atrophic and

metaplastic glands replace original glands, normal gastric secretions decrease, leading to hypochlorhydria and to low levels of pepsinogen produced by the chief cells of the corpus, and to high circulating levels of gastrin produced by antral G cells. The first metaplastic glands seen in the gastric mucosa phenotypically resemble those of the small intestine, with eosinophilic absorptive enterocytes with a brush border, alternating with mucus-producing goblet cells (complete or small IM). More advanced stages are characterized by phenotypic changes that are similar to colonic mucosa with the glands being lined by irregular goblet cells (incomplete or colonic metaplasia) (picture 1 and picture 2). In a subset of patients, foci of dysplasia may develop within areas of IM. (See "Gastric cancer: Pathology and molecular pathogenesis", section on 'Intestinal versus diffuse types'.)

CLINICAL FEATURES

GIM in general does not cause specific symptoms and is often found incidentally in patients undergoing upper endoscopy for dyspepsia. However, intestinal metaplasia is associated with gastric hypochlorhydria, which may predispose to small intestinal bacterial overgrowth with symptoms of bloating, abdominal discomfort, and diarrhea. (See "Small intestinal bacterial overgrowth: Clinical manifestations and diagnosis", section on 'Clinical features'.)

CANCER RISK

Incidence — Patients with GIM are thought to be at increased risk for gastric cancer but the absolute risk appears to be low in areas of low gastric cancer incidence, such as North America and northern Europe. A limited number of studies have evaluated the rate of progression to gastric cancer in patients with GIM, and estimates from small studies have varied widely and depend upon the background incidence of gastric cancer [24-27]. In European, Asian, and United States populations, the rates of progression vary between 1.1 and 2.0 per 1000 person-years, and up to 3.0 per 1000 person-years if high-grade dysplasia is included in the definition of progression [12,26,28-34].

Risk factors — Risk for gastric cancer may be higher among individuals with incomplete versus complete GIM on histology, and those with a family history of gastric cancer in a first-degree relative [12,27,29,35-38]. It is unclear if extensive metaplasia is a risk factor

[39]. In a meta-analysis of seven studies, having incomplete versus complete GIM was associated with a threefold increased risk of incident gastric cancer (over 3 to 13 years follow-up; relative risk [RR] 3.3, 95% confidence interval [CI], 1.9-5.6) [40]. Among patients with GIM who had biopsies obtained from both the gastric antrum/incisura and body, and extensive GIM versus limited involvement (ie, including involvement of at least the gastric body versus GIM of the antrum and/or incisura, respectively) the risk of incident gastric cancer was not increased (RR 2.07, 95% CI, 0.97-4.42). Among patients with GIM, having a family history of a first-degree relative with gastric cancer was associated with 4.5-fold increased risk for incident gastric cancer based on three studies (RR 4.5, 95% CI, 1.3-15.5).

Race and ethnicity may also play a role in GIM progression [18,19,32,33,41]. Immigrant populations from high-risk regions to low-incidence regions have a persistent increased risk of gastric cancer [41]. However, data on the influence of other gastric cancer risk factors (eg, smoking and alcohol use, pernicious anemia or autoimmune gastritis, *Helicobacter pylori* infection) on rates of GIM progression are lacking [13,40]. (See 'Management' below.)

DIAGNOSIS

The diagnosis of GIM may be suspected based on endoscopic findings, but is established by histology. (See 'Gastric topographic biopsy mapping' below and "Gastric cancer: Pathology and molecular pathogenesis", section on 'Intestinal metaplasia'.)

Upper endoscopy with biopsy — Endoscopically, GIM has the appearance of small grey-white, slightly elevated plaques surrounded by mixed patchy pink and pale areas of mucosa causing an irregular, uneven surface. GIM may also have the appearance of mottled patchy erythema. High-quality endoscopy examination is needed to detect GIM. We perform upper endoscopy with high-resolution scopes and use enhanced imaging techniques (eg, narrow band imaging (NBI), blue light imaging) when GIM is suspected. Several studies have demonstrated that white light endoscopy alone (without high-resolution or enhanced imaging) has low sensitivity for GIM and cannot accurately distinguish between non-atrophic gastritis, multifocal atrophic gastritis, and IM [42-44]. In addition, interobserver variability often limits the utility of endoscopic findings [45]. NBI is a high-resolution endoscopic technique that enhances the fine structure of the mucosal surface without the use of dyes. NBI should be combined with magnification endoscopy

to improve visualization of surface and vascular patterns for GIM detection. The light blue crest pattern and/or marginal turbid band are suggestive of GIM [46]. As compared with histology, NBI with magnification endoscopy has a sensitivity and specificity of 89 and 93 percent, respectively [46-51]. Further studies are needed to validate NBI patterns in GIM [52-56].

Other endoscopic measures that may increase the yield of upper endoscopy include adequate examination time (seven minutes intubation to extubation), mucosal cleaning, and air insufflation for mucosal visualization [57-60]. Visual inspection of the stomach should be accompanied by photographic documentation (antrum, pylorus, incisura, lesser curve, greater curve, fundus, and cardia; however, the optimal number is unclear, and suggested ranges vary widely (4 to 28 minutes) [58,61,62].) Other station-based screening protocols (with 22 or 26 pictures) have also been proposed in areas of high cancer incidence for detection of gastric neoplasia whereby each area of the stomach is viewed and photographed [63-65].

(See "Magnification endoscopy" and "Barrett's esophagus: Evaluation with optical chromoscopy", section on 'Principles'.)

Gastric topographic biopsy mapping — Diagnostic findings of GIM on gastric biopsy include replacement of the foveolar and glandular epithelium in the oxyntic and/or antral mucosa by metaplastic epithelium. However, gastric topographic mapping is required to determine the type and extent of metaplasia and guide management (algorithm 1) [8,10,25]. For gastric topographic mapping, biopsies are obtained from a minimum of five nontargeted biopsy sites (the lesser and greater curvatures of both the antrum and corpus, and the incisura angularis), with one or two biopsies ("bites") per site (figure 1) [4]. Specimens should be placed in a minimum of two bottles, for the corpus and antrum with incisura, respectively [66-72]. In addition, targeted biopsies should be obtained from irregular areas of the mucosa to rule out dysplasia and early gastric cancer. Pathology reports should include comments on the GIM subtypes, which are routine with H&E staining. (See 'Terminology' above.)

Tests with unclear role

- **Other endoscopic modalities** – Several other endoscopic modalities have been evaluated to detect early gastric cancer and other gastric precancerous lesions, and endoscopy imaging technologies are in rapid evolution [73,74]. However, further

studies are needed before they can be routinely recommended [75-77].

- Magnification chromoendoscopy – The use of magnification chromoendoscopy for GIM and early gastric cancer is limited to specialized centers, primarily in Asia. Magnification chromoendoscopy involves the topical application of stains or pigments to improve tissue localization, characterization, or diagnosis with magnification endoscopy. Although several agents (eg, methylene blue, acetic acid) have been studied, indigo carmine is the most commonly used stain [78,79]. Magnification chromoendoscopy requires specialized training and lengthens the procedure duration. While some studies have demonstrated a significant improvement in detection of GIM with the use of chromoendoscopy with indigo carmine, discordant data have also been published. (See "Chromoendoscopy" and "Magnification endoscopy", section on 'Stomach'.)

- Confocal endomicroscopy – Confocal endomicroscopy is based upon the principle of illuminating a tissue with a low-power laser and then detecting fluorescent light reflected from the tissue. The laser is focused at a specific depth and only light reflected back from that plane is refocused and able to pass through the pinhole confocal aperture. As a result, scattered light from above and below the plane of interest is not detected, increasing spatial resolution. The area being examined is scanned in the horizontal and vertical planes and an image is reconstructed. The applications and efficacy of confocal microscopy are discussed in detail separately. (See "Confocal laser endomicroscopy and endocytoscopy".)

- **Biomarkers** – Serum pepsinogen I and the pepsinogen I/II ratio have been studied extensively as a screening modality for gastric atrophy, GIM, dysplasia, and adenocarcinoma in Asian and European populations [80-82]. The testing has been combined with the *Helicobacter pylori* serology and/or gastrin 17 in some protocols [83-85]. The limited sensitivity and specificity of pepsinogen and related assays testing preclude its use. (See "Gastric cancer screening", section on 'Serum pepsinogen'.)

POST-DIAGNOSTIC EVALUATION

Assessment of cancer risk — Patients with GIM are considered at high-risk for gastric cancer if they have any one of the following risk factors for gastric cancer [41,57,61,86,87]:

- Family history of gastric cancer in a first-degree relative
- Incomplete GIM
- Extensive or corpus GIM
- Immigrants from high gastric cancer-incidence areas

Determining the type and extent of GIM requires gastric topographic mapping (algorithm 1) [8,10,25]. If GIM is suspected on the index upper endoscopy, biopsy mapping may be performed. However, if GIM is discovered incidentally on a gastric biopsy but without complete biopsy mapping, the decision to perform a repeat upper endoscopy and the timing of the procedure is based on risk factors for gastric adenocarcinoma (eg, family history of gastric cancer, racial/ethnic minorities, and/or immigrants from high-incidence areas). (See 'Endoscopic surveillance in selected patients' below and 'Gastric topographic biopsy mapping' above.)

Assessment of family cancer history is important in determining cancer risk and should include a cancer history in first- and second-degree relatives. Familial clustering of gastric cancer occurs in approximately 10 percent of cases. However, hereditary gastric cancer syndromes account for approximately 1 to 3 percent of cases and include three main subgroups: hereditary diffuse gastric cancer, familial intestinal gastric cancer, and other single-gene syndromes associated with an increased risk of gastric adenocarcinoma (eg, Lynch syndrome, Cowden syndrome, Peutz Jegher syndrome, familial adenomatous polyposis).

Individuals with any one of the following should be referred for genetic evaluation for a hereditary gastric cancer syndrome:

- Gastric cancer in a first-degree relative before age 40
- Gastric cancer in two first-degree/second-degree relatives with one diagnosis before age 50
- Gastric cancer in three first-degree/second-degree relatives independent of age

Screening for Helicobacter pylori (H. pylori) — Infection with *H. pylori* is initially assessed by the histologic evaluation of gastric biopsy samples [88]. However, *H. pylori* organism loads may decrease with the development of gastric atrophy and GIM, thereby reducing biopsy sensitivity. In the event of negative biopsies for *H. pylori*, infection status should be determined with serology. Any patient with positive serology who does not have a definitive history of having been treated for *H. pylori* should receive treatment,

even if biopsies are negative. (See "Indications and diagnostic tests for Helicobacter pylori infection in adults", section on 'Noninvasive testing' and "Treatment regimens for Helicobacter pylori in adults".)

MANAGEMENT

General measures in all patients — General measures to decrease the risk of gastric cancer include smoking cessation, moderation of alcohol intake, and eradication of *Helicobacter pylori* (*H. pylori*). *H. pylori* is a known risk factor for gastric cancer [25]. Eradication of *H. pylori* appears to reverse the histologic changes in the majority of patients with chronic nonatrophic gastritis and in many patients with multifocal atrophic gastritis [89-94]. Although eradication therapy does not reverse GIM [90,95]. Persistent infection and/or reinfection appears to affect the risk of progression [96]. In patients with GIM who have undergone endoscopic treatment for early gastric cancer or surgical resection for a gastric adenocarcinoma, eradication has been shown to decrease the risk of future adenocarcinoma [97,98]. (See "Early gastric cancer: Management and prognosis", section on 'Endoscopic resection'.)

Endoscopic surveillance in selected patients — In individuals with high-risk GIM, we suggest surveillance upper endoscopy which includes a detailed visual inspection, enhanced image enhancement (eg, narrow band imaging, blue light imaging) if there is local expertise, and gastric biopsy mapping (algorithm 1). High-risk GIM groups include patients with any one of the following [41,57,61,86,87]:

- Family history of gastric cancer in a first-degree relative
- Incomplete GIM
- Extensive or corpus GIM
- Racial/ethnic minorities in the United States (including African Americans, Hispanics, and Asians)
- First-generation immigrants from high-incidence areas (eg, Eastern Asia, mountainous Latin America)

Evidence suggests that surveillance may lead to early detection of gastric cancer and improved survival [12,99-101]. However, prospective studies are needed to determine the optimal surveillance intervals. Our recommendations for surveillance, depending on the type and extent of IM, are based on expert opinion and are largely consistent with other

guidelines [57,61,86]. The role of surveillance and management of gastric cancer risk in patients with hereditary gastric cancer syndromes is discussed in detail separately. (See "Hereditary diffuse gastric cancer" and "Li-Fraumeni syndrome" and "Lynch syndrome (hereditary nonpolyposis colorectal cancer): Clinical manifestations and diagnosis" and "Clinical manifestations and diagnosis of familial adenomatous polyposis" and "Overview of hereditary breast and ovarian cancer syndromes".)

Extensive or incomplete IM — In patients with extensive IM or incomplete IM, we perform endoscopic surveillance for gastric cancer at the three-year interval [59,102]. Although other guidelines have suggested that those with a family history and extensive or incomplete IM undergo more intense (one to two year) surveillance endoscopy, evidence to support this recommendation are lacking [61]. (See 'Upper endoscopy with biopsy' above.)

Limited or complete GIM and additional gastric cancer risk factor — In patients with limited or complete GIM and a risk factor for gastric cancer (eg, family history of gastric cancer in a first-degree relative [without a hereditary gastric cancer syndrome], persistent *H. pylori* gastritis, pernicious anemia), we suggest endoscopic surveillance for gastric cancer every three years. (See "Metaplastic (chronic) atrophic gastritis", section on 'Endoscopic surveillance in selected patients'.)

In patients with limited or complete GIM who do not have additional risk factors for gastric cancer, evidence to support routine endoscopic surveillance is lacking [57,61,86]. However, GIM surveillance should be driven by shared patient-physician decision making.

Unclear role of chemoprevention — Definitive evidence to support the use of chemoprevention agents in patients with GIM is lacking. Although observational studies have suggested a benefit of aspirin andnonsteroidal anti-inflammatory drugs in decreasing the risk of gastric cancer as well as the regression of IM with celecoxib use, there are no randomized controlled trials [103-107]. In addition, studies that have evaluated the effect of antioxidant vitamin supplementation (eg, ascorbic acid, beta-carotene) on the risk of progression of gastric precancerous lesions have yielded inconsistent results [108-112].

SOCIETY GUIDELINE LINKS

Links to society and government-sponsored guidelines from selected countries and regions around the world are provided separately. (See "Society guideline links: Gastric intestinal metaplasia".)

SUMMARY AND RECOMMENDATIONS

- GIM is characterized by the replacement of the foveolar and glandular epithelium in the oxyntic or antral mucosa by intestinal epithelium. (See 'Terminology' above and 'Classification' above.)

- GIM is an important premalignant stage in the gastric cancer cascade through a series of well-defined and recognizable precursors. The gastric adenocarcinoma multistage model, the "Correa cascade," suggests that a combination of host genetic factors and responses, *Helicobacter pylori* (*H. pylori*) genomics, with modulation by dietary and environmental factors, predispose to early pan-gastric mucosal inflammation, which in turn may progress sequentially to gastric atrophy, IM, dysplasia, and adenocarcinoma. (See 'Epidemiology and pathogenesis' above.)

- Patients with GIM are thought to be at increased risk for gastric cancer but the absolute risk appears to be modest in areas of low gastric cancer incidence (2.5 per 1000 person-years). However, among patients with GIM, the risk of gastric cancer is higher in individuals with extensive and incomplete IM. (See 'Cancer risk' above and 'Classification' above.)

- GIM in general does not cause specific symptoms and is often diagnosed incidentally in patients undergoing upper endoscopy for dyspepsia. However, it is associated with gastric hypochlorhydria, which may predispose to small intestinal bacterial overgrowth with symptoms of bloating, abdominal discomfort, and diarrhea. On upper endoscopy, GIM has a nonspecific appearance and involved mucosa may have rough or villous appearance, or may be seen as thin, white mucosal deposits. (See 'Clinical features' above.)

- The diagnosis of GIM may be suspected based on endoscopic findings, but is established by histology. High-quality endoscopy examination is needed to improve detection. We perform upper endoscopy with image enhancement (eg, narrow band imaging)) and biopsy mapping (figure 1). (See 'Diagnosis' above.)

- Patients with GIM are considered at increased risk for gastric cancer if they have any one of the following (see 'Classification' above and 'Assessment of cancer risk' above):

  - Family history of gastric cancer in a first-degree relative
  - Incomplete GIM
  - Extensive or corpus GIM
  - Racial/ethnic minorities in the United States (including African Americans, Hispanics, and Asians)
  - First-generation immigrants from high-incidence areas (eg, Eastern Asia, mountainous Latin America)

  Determining the type and extent of GIM requires gastric topographic mapping (figure 1).

- General measures to decrease the risk of GIM progression to gastric cancer include smoking cessation, moderation of alcohol intake, and eradication of *H. pylori*. Although eradication of *H. pylori* does not reverse GIM, it may slow progression to gastric cancer. (See 'General measures in all patients' above.)

- In individuals with high-risk GIM, we perform surveillance upper endoscopy at the three-year interval (algorithm 1). Surveillance endoscopic evaluation includes a detailed visual inspection with high-resolution endoscopes and image enhancement techniques (eg, NBI) if there is local expertise, and gastric biopsy mapping. (See 'Management' above.)

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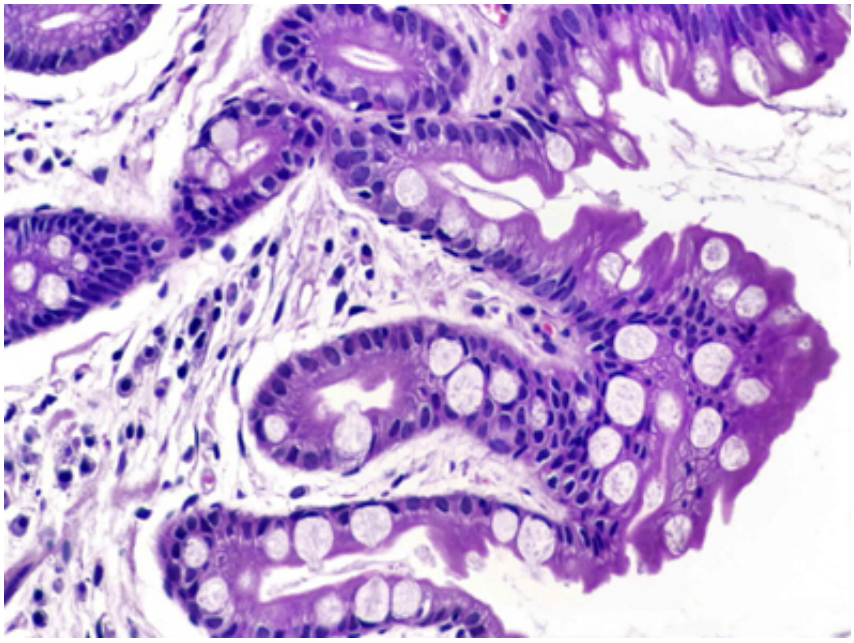
Topic 16636 Version 19.0

GRAPHICS

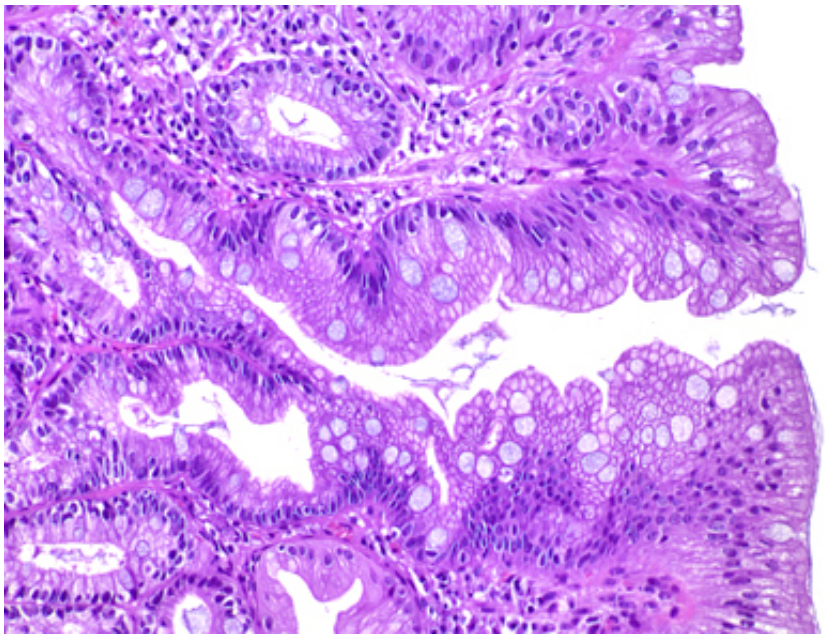
Complete gastric intestinal metaplasia



In complete intestinal metaplasia, the glands and foveolar epithelium are replaced by small intestine type mucosa with goblet cells, eosinophilic enterocytes and a "brush border."

Graphic 81064 Version 2.0

Incomplete gastric intestinal metaplasia



Incomplete intestinal metaplasia with colonic epithelium, multiple, irregular goblet cells without a brush border.

Graphic 77310 Version 2.0

OLGA staging system for identifying patients with metaplastic (chronic) atrophic gastritis who are at high risk for evolution to gastric cancer

Stage 0	Scores of 0 (no atrophy) in corpus and antrum
Stage I	Score of 1 (mild atrophy) in corpus with score of 0 or 1 in antrum, **or** score of 0 in corpus and score of 1 in antrum
Stage II	Score of 2 (moderate atrophy) or 3 (severe atrophy) in corpus with score of 0 in antrum, **o** score of 2 in corpus with score of 1 in antrum, **or** score of 0 or 1 in corpus with score of 2 in antrum
Stage III	Score of 3 in corpus with score of 1 in antrum, **or** score of 2 in corpus and 2 in antrum, **or** score of 0 or 1 in corpus with score of 3 in antrum
Stage IV	Scores of 3 in corpus and antrum, **or** score of 3 in corpus and 2 in antrum or score of 2 in corpus and 3 in antrum

OLGA: Operative Link for Gastritis Assessment.

Original table modified for this publication. From: Rugge M, Correa P, DiMario F, et al. OLGA staging for gastritis: A tutorial. Dig Liver Dis 2008; 40:650. Table used with the permission of Elsevier Inc. All rights reserved.

Graphic 121370 Version 2.0

OLGIM staging system for identifying patients with metaplastic (chronic) atrophic gastritis who are at high risk for evolution to gastric cancer

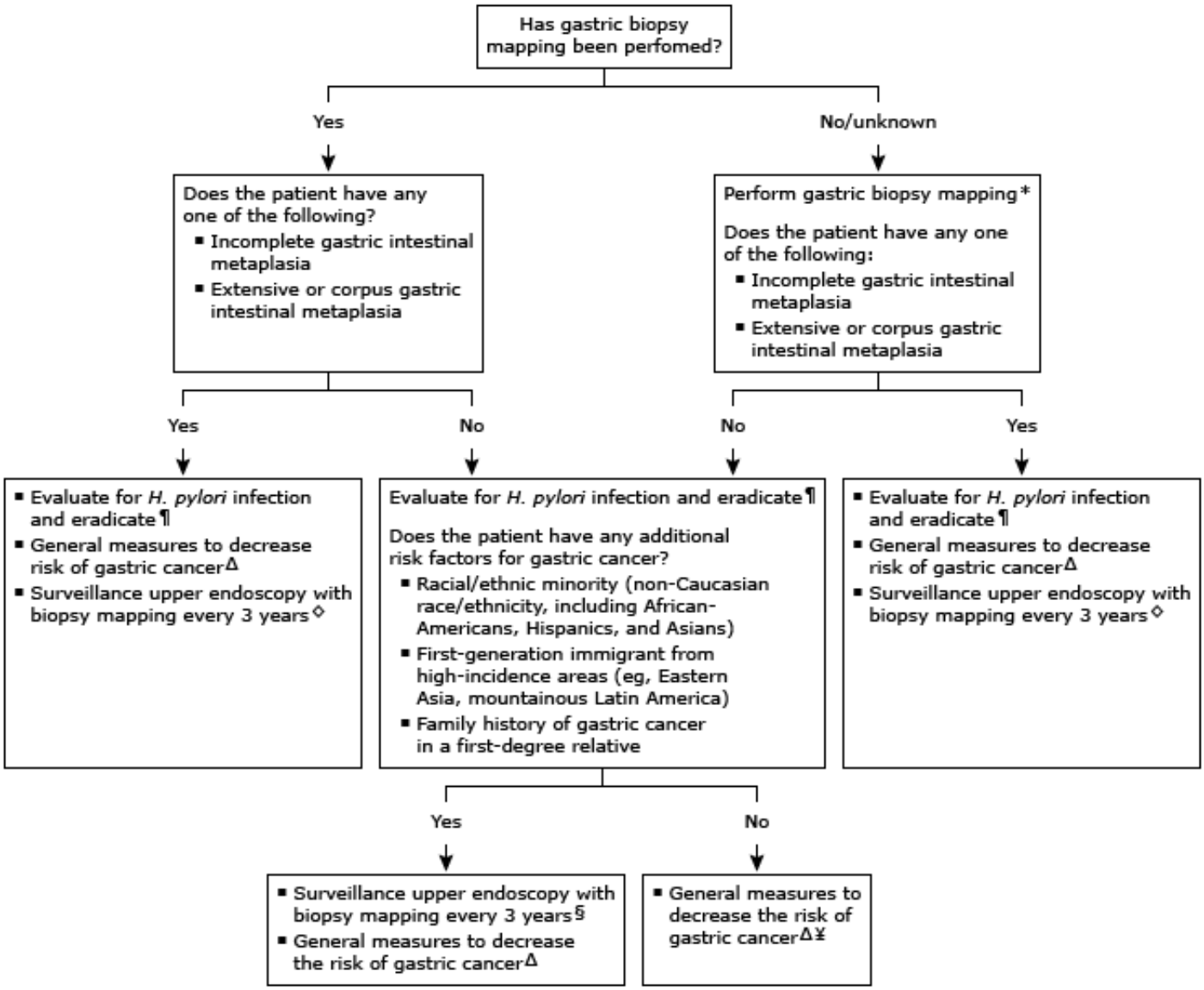
Stage 0	Scores of 0 (no IM) in corpus and antrum
Stage I	Score of 1 (mild IM) in corpus with score of 0 or 1 in antrum, **or** score of 0 in corpus and score of 1 in antrum
Stage II	Score of 2 (moderate IM) or 3 (severe IM) in corpus with score of 0 in antrum; score of 2 in corpus with score of 1 in antrum; **or** score of 0 or 1 in corpus with score of 2 in antrum
Stage III	Score of 3 (severe IM) in corpus with score of 1 or 2 in antrum, **or** score of 0 or 1 in corpus with score of 3 in antrum
Stage IV	Scores of 3 in corpus and antrum, **or** score of 3 in corpus and 2 in antrum or score of 2 in corpus and 3 in antrum

IM: intestinal metaplasia; OLGIM: Operative Link on Gastric Intestinal Metaplasia Assessment.

Original table modified for this publication. From: Capelle LG, de Vries AC, Haringsma J, et al. The staging of gastritis with the OLGA system by using intestinal metaplasia as an accurate alternative for atrophic gastritis. Gastrointestinal Endoscopy 2010; 1150. Table used with the permission of Elsevier Inc. All rights reserved.

Graphic 116254 Version 2.0

Approach to the management of patients with gastric intestinal metaplasia



H. pylori: Helicobacter pylori.

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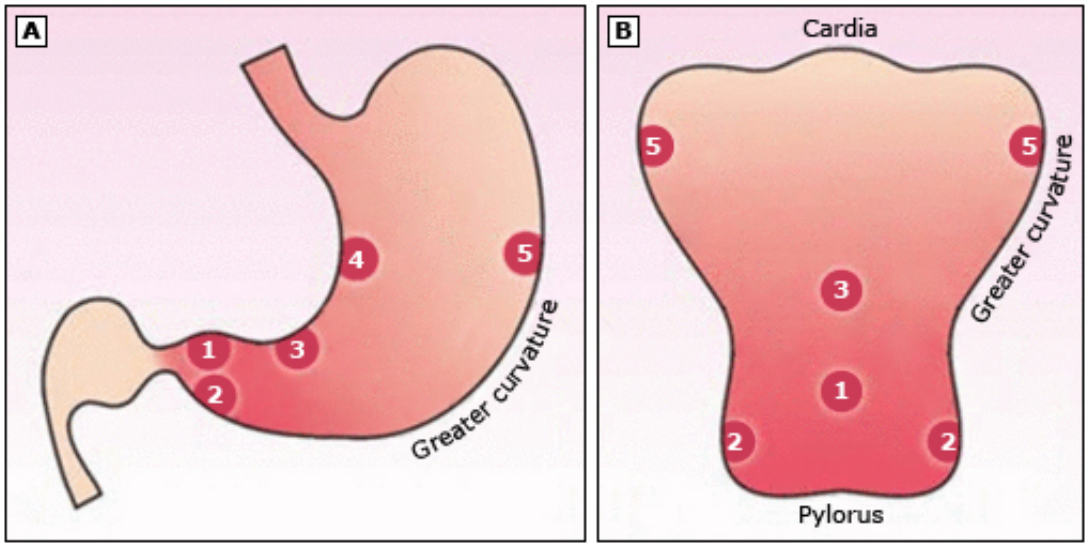
* In patients with known risk factors for gastric cancer, who are amenable to endoscopic surveillance, we generally perform a repeat endoscopy with gastric biopsy mapping within one year. In patients without known risk factors for gastric cancer, but who are amenable to endoscopic surveillance if indicated, we perform a repeat upper endoscopy within three years.
¶ Infection with *H. pylori* is initially assessed by the histologic evaluation of gastric biopsy samples. In the event of negative biopsies for *H. pylori*, infection status should be determined with serology. Any patient with positive serology who does not have a definitive history of having been treated for *H. pylori* should receive treatment, even if biopsies are negative.
Δ General measures to decrease the risk of gastric cancer include smoking cessation and moderation of alcohol intake.

◇ Surveillance endoscopic evaluation includes detailed visual inspection with high-resolution endoscopes, narrow band imaging if there is local expertise, and gastric biopsy mapping.
§ These recommendations do not apply to individuals with hereditary gastric cancer syndromes. Refer to UpToDate topics on gastric cancer for additional details.
¥ In patients with limited or complete GIM who do not have additional risk factors for gastric cancer, evidence to support routine endoscopic surveillance is lacking. GIM surveillance should be driven by shared patient-physician decision making.

Graphic 127117 Version 1.0

Gastric biopsy sampling protocol



- **(Panel A)** Sydney protocol biopsy sites in the anatomic view.
- **(Panel B)** Sydney protocol biopsy sites in the opened stomach along the greater curvature.
- **Gastric biopsies should be obtained from the following sites:**
    1. Distal antrum, lesser curvature, within 3 to 5 cm of pylorus
    2. Distal antrum, greater curvature, within 3 to 5 cm of pylorus
    3. Lesser curvature of the incisura angularis
    4. Proximal corpus, lesser curvature
    5. Proximal corpus, greater curvature

Adapted from: Nieuwenburg SA, Waddingham WW, Graham D. Accuracy of endoscopic staging and targeted biopsies for routine gastric intestinal metaplasia and gastric atrophy evaluation study protocol of a prospective, cohort study: the estimate study. BMJ Open 2019; 9:e032013. Copyright © 2019 The Authors. Available at: https://bmjopen.bmj.com/content/9/9/e032013 (Accessed on February 18, 2020). Reproduced under the terms of the Creative Commons Attribution License.

Graphic 127130 Version 2.0

Contributor Disclosures

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Conflict of interest policy

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