AGA Clinical Practice Update on the Diagnosis and Management of Atrophic Gastritis

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The Emoroid Digest
@EmoryGastroHep

Background

- **Atrophic gastritis (AG):** Preneoplastic condition defined by loss of gastric glands +/- metaplasia in the setting of chronic inflammation
  - First of a multistep precancerous cascade, more advanced stages are gastric intestinal metaplasia (GIM), dysplasia, and gastric adenocarcinoma
  - Patients with chronic AG are at increased risk of type 1 NETs
- **Etiology:** Two most common are *Helicobacter pylori* (HpAG) > autoimmunity (AIG)
- **Prevalence:** Up to 15% in US populations, may be higher in specific populations with more *H pylori* or gastric adenocarcinoma
- **Risk factors:** For nonautoimmune AG- *H pylori*, age, tobacco use, high salt diet, chronic bile acid reflux. For AIG- age, gender (F>M), presence of other autoimmune diseases
- **Diagnosis:** Made by histopathology

Histopathology & Anatomic Distribution

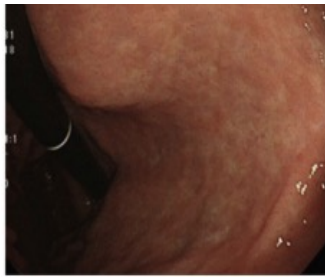
- Gastric glands are replaced with either connective tissue (non-metaplastic atrophy) or different epithelium (metaplastic atrophy)
- Intestinal metaplasia is most frequently diagnosed and almost invariably implies AG
- **HpAG:** Initially atrophic changes in incisura and antrocorporal transitional mucosa and eventually spread to corpus/fundus
- **AIG:** Corpus predominant atrophy with antral sparing

Optimizing Evaluation

1) Excellent mucosal visualization w/ air insufflation, mucosal cleaning, & mucolytic agents
2) Examine lumen including color and texture, appearance of submucosal blood vessels, and architecture of gastric rugae, followed by targeted examinations of focal abnormalities using high-definition white-light endoscopy or narrow-band imaging
3) Photographic documentation to cover the cardia and fundus, lesser and greater curvature of corpus and antrum, incisura angularis, and pylorus

Endoscopic Appearance

- Atrophic mucosa typically has a pale appearance, w/ increased visibility of submucosal blood vessels due to thinning of gastric mucosa and loss of gastric folds



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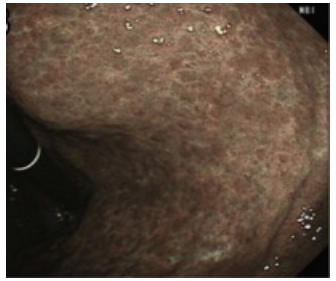
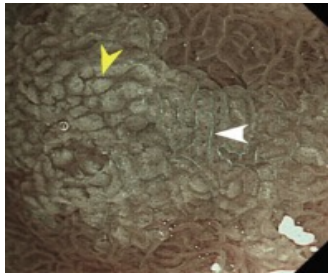


NBI

- On NBI, characteristic signs of IM include the **light blue crest** sign (LBC, fine, blue-white lines on the crests of the epithelial surface) and **white opaque field** (WOF, light scattering at microscopic lipid drops that accumulate in IM mucosa)

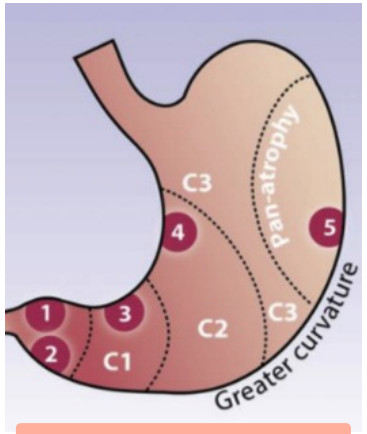


LBC= white arrowhead

WOF= yellow arrowhead

Biopsy Protocol

- **5 gastric biopsies** in separately labeled jars:
  - 2 from the antrum along lesser + greater curvature (within 2-3 cm of the pylorus)
  - 2 from the gastric corpus (including 1 from the lesser curvature at 4 cm proximal to the incisura and 1 from the middle portion of the greater curvature of the body at 8 cm from the cardia)
  - 1 from the incisura angularis
- Targeted biopsies from any visible mucosal abnormalities go in a separate jar



Updated Sydney Protocol

Shah SC, Piazuelo MB, Kuipers EJ, Li D. AGA Clinical Practice Update on the Diagnosis and Management of Atrophic Gastritis: Expert Review . Gastroenterology. 2021; 161:1325-332.

Serologic Diagnosis

- In patients with histology compatible with AIG, providers should check **parietal cell antibodies (PCAs)** and **intrinsic factor antibodies (IFAs)** to assist with diagnosis
- **PCA** is the most sensitive serum biomarker for AIG, but can have false positives with *H pylori* infection and other autoimmune diseases
- **IFA** has low sensitivity but high specificity, and is positive later in the disease course

Management of AG

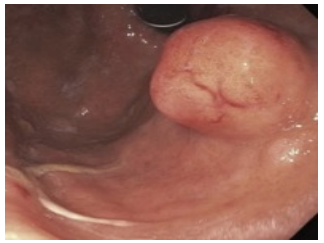
- Test all patients with AG for *H pylori* and eradicate if positive
- H pylori eradication can return gastric mucosa to normal In some patients, but most have passed a 'point of no return' and mucosal damage can't be reversed
- Despite persistent signs of AG, H pylori eradication does still reduce the risk of gastric cancer

Endoscopic Surveillance

- Consider surveillance **every 3 years** in patients with advanced AG (based on histology and anatomic extent). However optimal intervals remain to be determined; shorter/longer intervals may be appropriate depending on risk assessment
- **Factors to consider:** Quality of baseline endoscopy, family history of gastric cancer, immigration history from geographic regions with high incidence of gastric cancer, persistent H pylori infection, smoking history and dietary factors

- Surveillance strategy for AIG remains unclear, current ESGE guidelines suggest performing surveillance at **3–5 years**
- In patients with pernicious anemia (PA), risk of gastric adenocarcinoma might be highest within the **first year** of diagnosis; ASGE advocates for an upper endoscopy **within 6 months of the diagnosis of PA** for risk stratification and to evaluate for prevalent gastric neoplasia and NETs
- The development of upper gastrointestinal symptoms in patients w/ PA should also prompt diagnostic endoscopy

Management of Type 1 Gastric NETs



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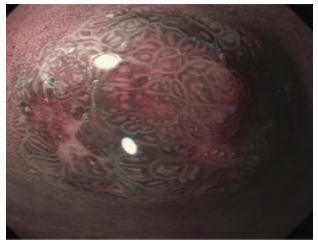

NBI

Gastric NETs associated with AG represent 80%–90% of all gastric NETs (mainly type 1)

<1 cm	1-2 cm	> 2 cm
Resect endoscopically (EMR), consider surveillance every 1-2 years depending on the burden of the NETs	Consider EUS to evaluate depth of tumor invasion & presence of local metastasis	Surgery *Also consider surgery if invasion past submucosa or lymph node metastasis*

Management of Co-existing Conditions

- Evaluate for both **iron and vitamin B12 deficiency**
  - Patients with corpus-predominant AG are at risk of deficiency due to reduced gastric acid secretion and intrinsic factor
  - Iron deficiency presents much earlier than B-12 deficiency

- Screening for **autoimmune thyroid disease** should be considered in patients diagnosed with AIG
- Maintain low threshold to evaluate for other associated autoimmune diseases, including type 1 diabetes mellitus and Addison's disease, if the clinical picture is consistent

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