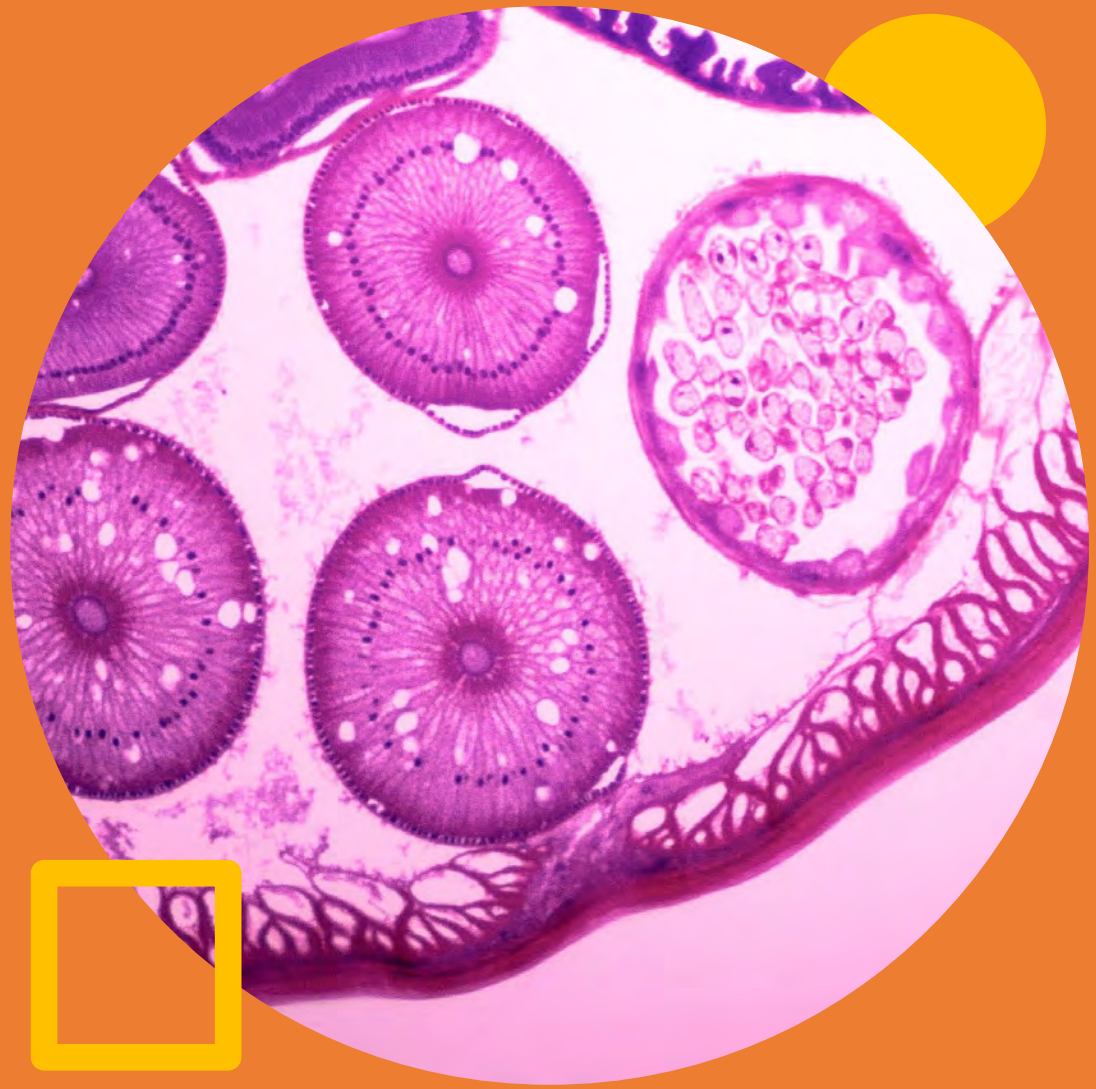
Pre-Malignant Conditions of the Stomach, Gastric Polyps and Gastric Cancer

Dr Midhun John

May 2026



OBJECTIVES FOR THIS TALK

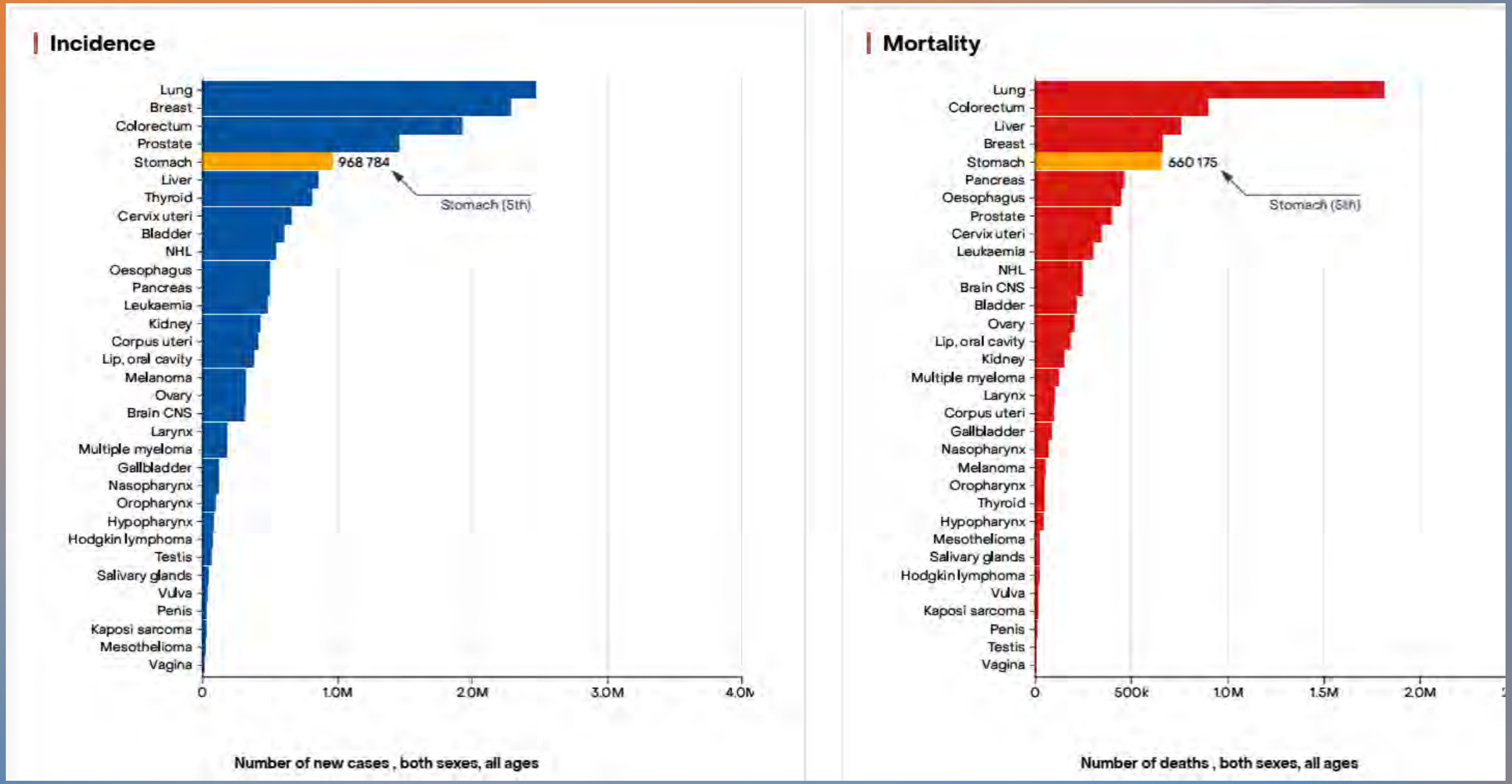
- Understand gastric carcinogenesis and the Correa Cascade
- Recognise premalignant gastric conditions
- Apply risk stratifications tools
- Differentiate polyps subtypes and their malignant potential
- Surveillance recommendations
- Modern approaches to early gastric cancer detections and management

INTRODUCTION AND EPIDEMIOLOGY

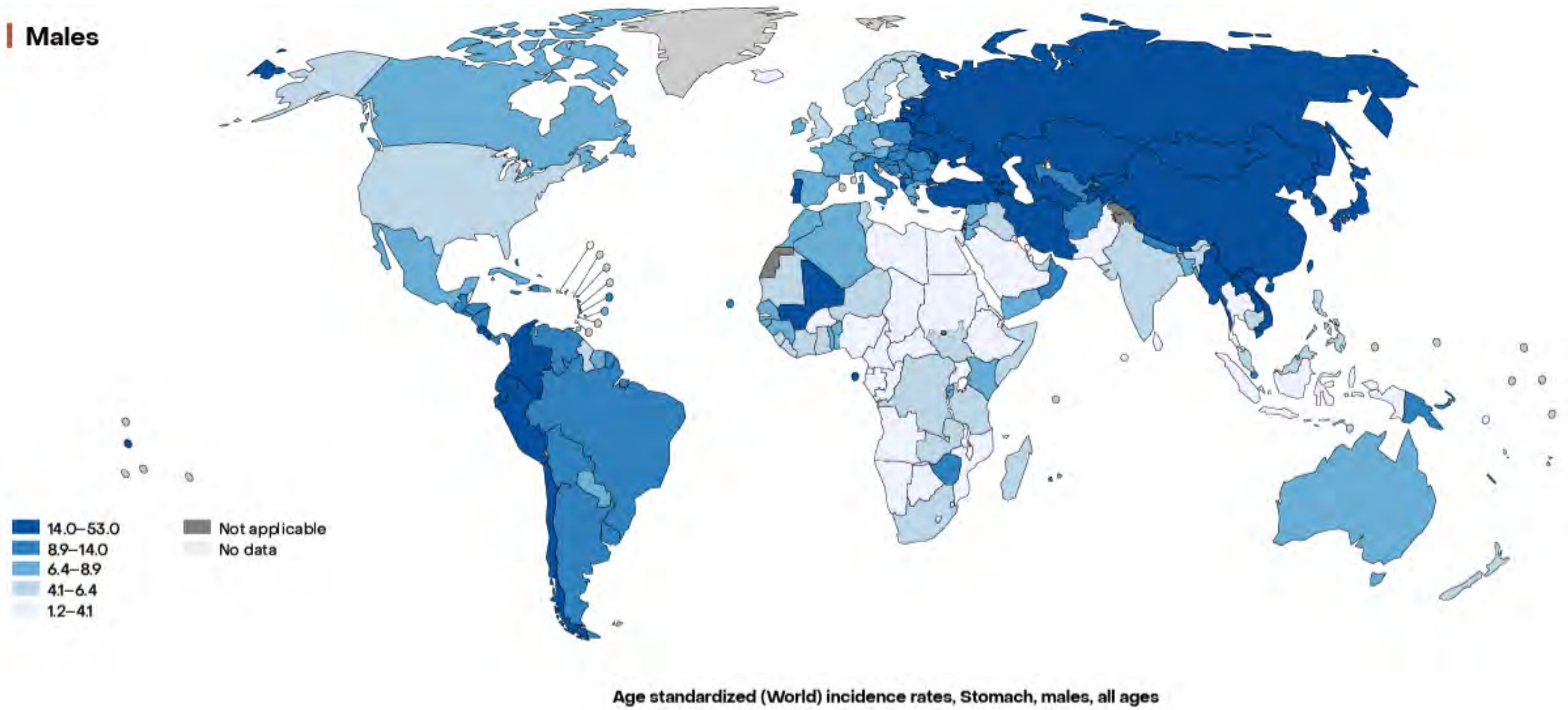
INTRODUCTION AND EPIDEMIOLOGY

- Gastric cancer (GC)
  - 5th most commonly diagnosed cancer globally
  - Nearly 1 million new cases and over 660000 deaths reported annually
  - Heaviest burden in Eastern Asia
  - Sharp rise in cases in sub-Saharan Africa
  - Men>Women (66% of all case)
  - Regions:
    - Asia 71%
    - Europe 14%
    - Africa 3.4%

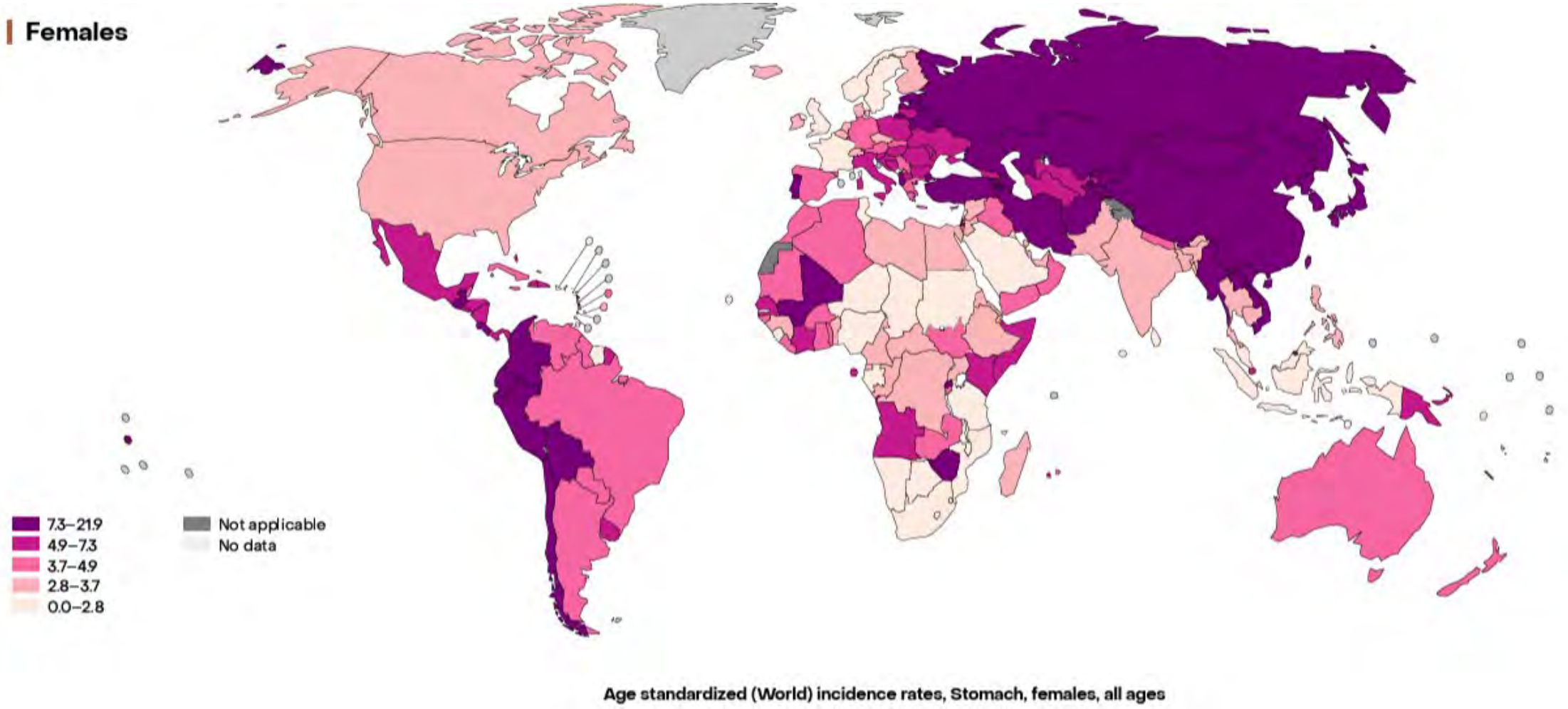
Morgan et al. The global epidemiology of gastric cancer and Helicobacter Pylori. 2025



International Agency for Research on Cancer (IARC), *GLOBOCAN 2022: Stomach Fact Sheet*, Lyon: World Health Organization, 2024. Available at: IARC GLOBOCAN 2022 Stomach Fact Sheet



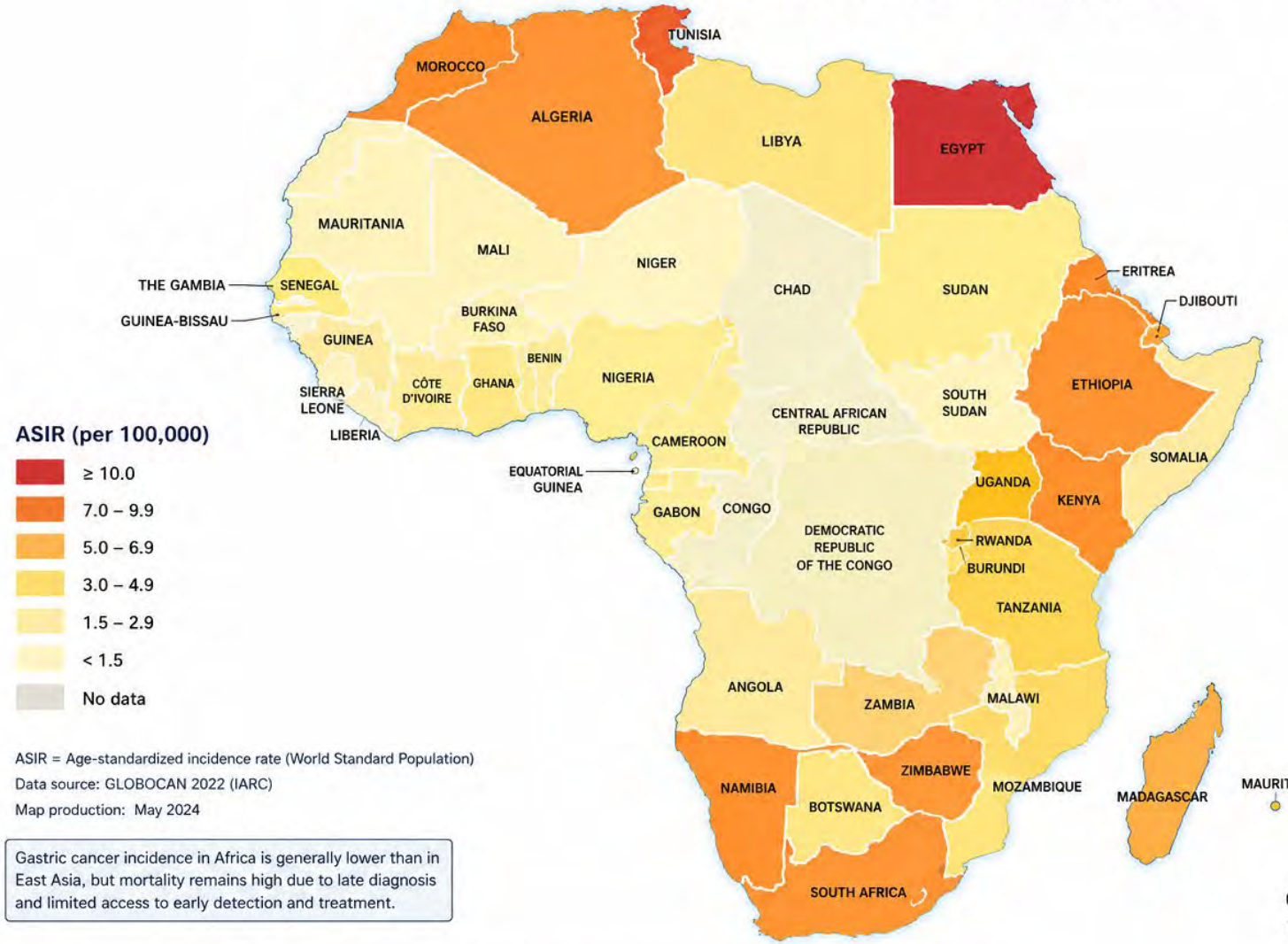
International Agency for Research on Cancer (IARC), *GLOBOCAN 2022: Stomach Fact Sheet*, Lyon: World Health Organization, 2024. Available at: IARC GLOBOCAN 2022 Stomach Fact Sheet



International Agency for Research on Cancer (IARC), *GLOBOCAN 2022: Stomach Fact Sheet*, Lyon: World Health Organization, 2024. Available at: IARC GLOBOCAN 2022 Stomach Fact Sheet

GASTRIC CANCER INCIDENCE IN AFRICA, 2022

Age-Standardized Incidence Rate (ASIR) per 100,000 population



ASIR (per 100,000)

- ≥ 10.0
- 7.0 – 9.9
- 5.0 – 6.9
- 3.0 – 4.9
- 1.5 – 2.9
- < 1.5
- No data

ASIR = Age-standardized incidence rate (World Standard Population)

Data source: GLOBOCAN 2022 (IARC)

Map production: May 2024

Gastric cancer incidence in Africa is generally lower than in East Asia, but mortality remains high due to late diagnosis and limited access to early detection and treatment.

EXAMPLE COUNTRY INCIDENCE (ASIR)

Country	ASIR (per 100,000)
Egypt	11.1
Morocco	8.3
Ethiopia	6.2
Kenya	5.6
Algeria	5.1
Tunisia	4.8
South Africa	4.5
Rwanda	4.4
Uganda	4.2
Ghana	2.3
Nigeria	1.8
DR Congo	1.3
Niger	0.9

N

0 750 1,500 km

Reference: Sung H, Ferlay J, Siegel RL, et al. Global Cancer Statistics 2022: GLOBOCAN Estimates of Incidence and Mortality Worldwide for 36 Cancers in 185 Countries. *CA Cancer J Clin.* 2024;74(3):229–263. doi:10.3322/caac.21834.

WHY SO LOW IN AFRICA?

1. Diagnostic Gap and Data Collection
    - Inadequate data collection
    - Limited access to endoscopy
    - Under-reporting
    - Late diagnosis

2. Biologic and Host Factors
    - Bacterial strains differ to the world
    - Host immune response
    - Co-infection

3. Environmental; and Dietary Factors
    - Dietary protective factors
    - Life expectancy

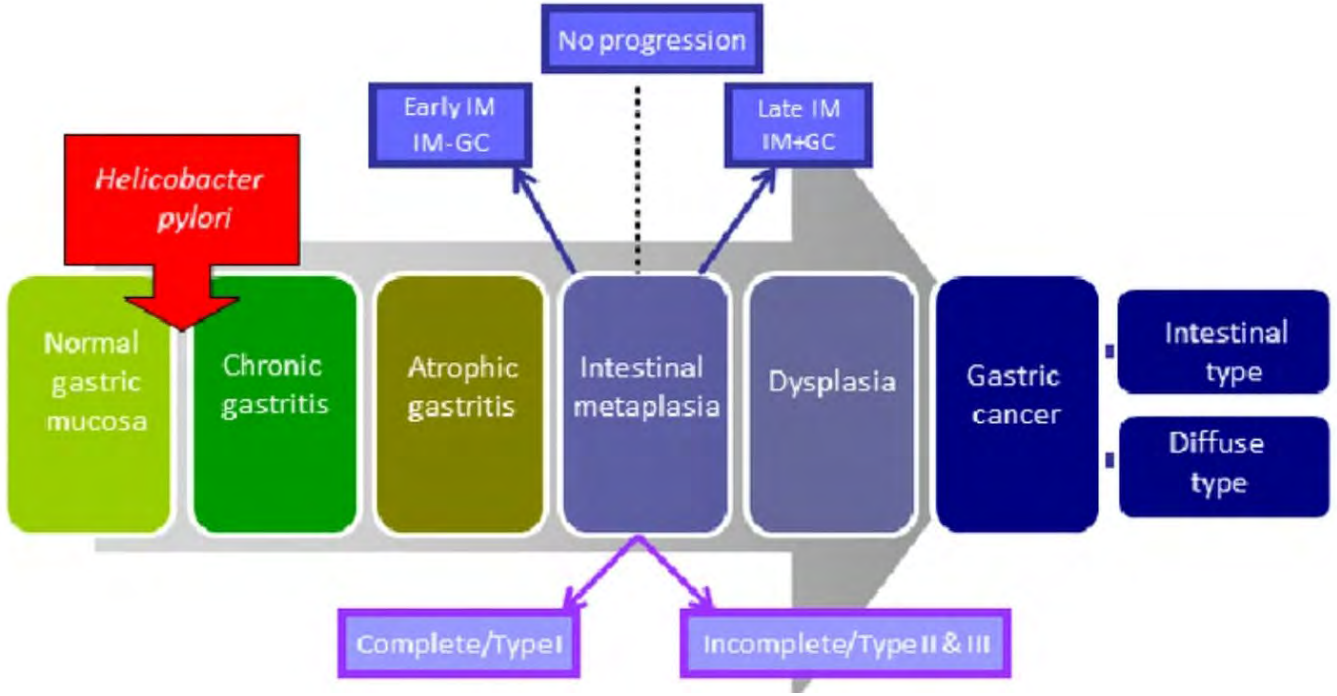
Graham et al. African, Asian or Indian enigma, the East Asian Helicobacter pylori: facts or medical myths. 2009
Asombang et al. Gastric Cancer in Africa. 2014

GASTRIC CARCINOGENESIS

GASTRIC CARCINOGENESIS

Correa Cascade

- Chronic gastritis→ Atrophy→ Intestinal Metaplasia→ Dysplasia→ Adenocarcinoma



Correa et al. Human Gastric carcinogenesis. 1992

GASTRIC CARCINOGENESIS- RISK FACTORS

1. Infectious Agents
   - Helicobacter pylori (H. pylori)-90% of all non-cardia GC
   - Epstein-Barr Virus (EBV)-5-10% of GC typically in the proximal stomach

2. Dietary and Lifestyle Factors
   - High salt intake food
   - Processed and refined meats
   - Smoking and alcohol
   - Obesity
   - Low Fruit/Vegetable Intake

Correa et al. Human Gastric carcinogenesis. 1992
MAPS III Guidelines 2025

GASTRIC CARCINOGENESIS- RISK FACTORS

3. Medical and Host Factors

- Chronic Gastric Conditions-Pernicious anaemia, CAG, adenomas
- Previous Gastric Surgery
- Blood Type A

4. Genetic and Hereditary Factors

- Family History
- Inherited Syndromes

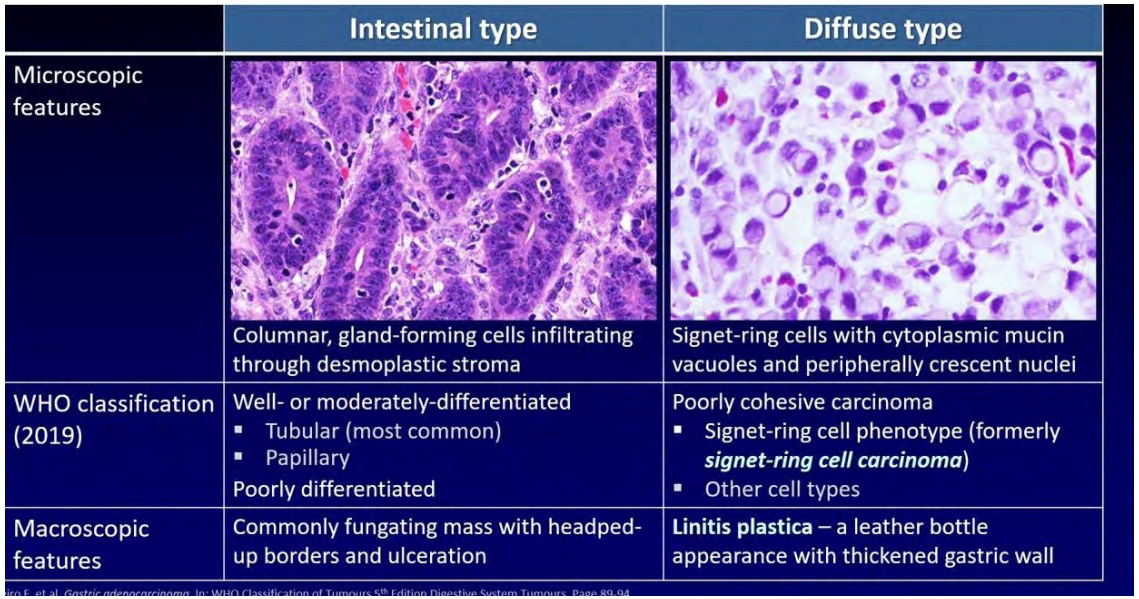
Correa et al. Human Gastric carcinogenesis. 1992
MAPS III Guidelines 2025

HISTOLOGICAL SUBTYPES

Feature	Intestinal Type	Diffuse Type	Mixed Type
Classification	Lauren Classification	Lauren Classification	Lauren Classification
Histology	Gland-forming adenocarcinoma	Poorly cohesive cells; signet-ring cells common	Combination of intestinal and diffuse patterns
Cell cohesion	Cohesive	Non-cohesive	Variable
Typical WHO subtype correlation	Tubular, papillary, mucinous	Poorly cohesive / signet-ring cell carcinoma	Mixed adenocarcinoma
Pathogenesis	Chronic inflammation → atrophy → intestinal metaplasia → dysplasia	Loss of cell adhesion (often E-cadherin/CDH1 related)	Combined mechanisms
Association with H. pylori	Strong	Less strong	Variable
Age group	Older patients	Younger patients	Intermediate
Sex predominance	Male predominance	More common in females	Variable
Gross appearance	Exophytic or ulcerating mass	Diffuse infiltrative thickening (“linitis plastica”)	Mixed appearance
Common location	Distal stomach/antrum	Entire stomach; often body	Variable
Precursor lesion	Intestinal metaplasia and dysplasia	Usually absent	Variable
Metastatic pattern	Liver metastases more common	Peritoneal spread more common	Mixed
Prognosis	Generally better	Worse prognosis	Intermediate
Molecular associations	Chromosomal instability, HER2 amplification	CDH1 mutation, genomically stable subtype	Heterogeneous
Response to chemotherapy	Often better response	Less responsive	Variable

Important points:

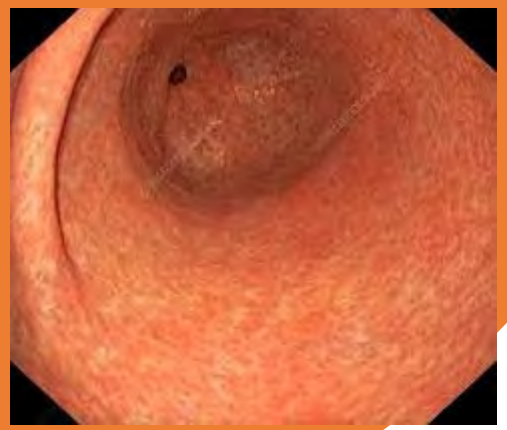
- Lauren Classification
- Diffuse, Intestinal and Mixed
- Intestinal follows Correa Cascade
- Diffuse associated with E-Cadherin dysfunction



Varon et al. Stomach Cancer. 2020

PREMALIGNANT CONDITIONS OF THE STOMACH

CHRONIC ATROPHIC GASTRITIS

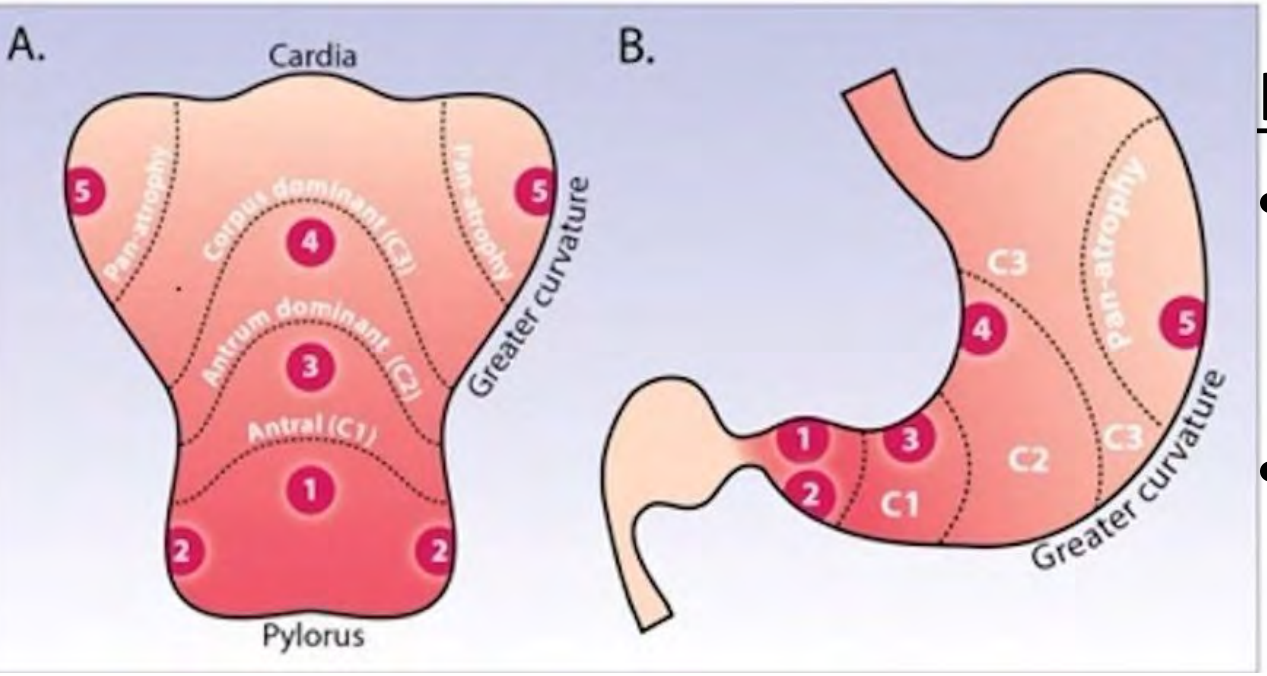


- 2 main types: Environmental Metaplastic Atrophic Gastritis (EMAG) and Autoimmune Metaplastic Atrophic Gastritis (AMAG)

- EMAG:
  - Usually H. Pylori related and initially affects the antrum →multi-focal

- AMAG
  - Corpus predominant disease associated with pernicious anaemia, Type 1 gastric neuroendocrine tumours and GC

CHRONIC ATROPHIC GASTRITIS



Kimura-Takemoto Classification

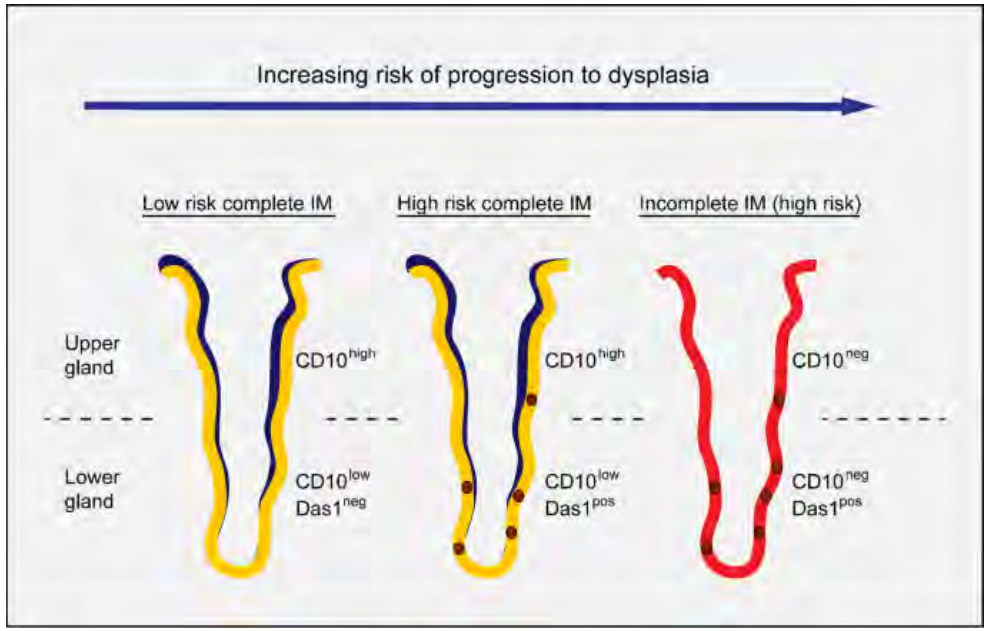
- Endoscopic grading system that evaluates the severity and progression of CAG
- 2 Primary categories-Closed and Open-type

Kotelevets SM, et al. Updated Kimura Takemoto.2021

GASTRIC INTESTINAL METAPLASIA

Complete Intestinal Metaplasia (Type 1)

- Histology resembles small intestine, goblet cells, absorptive enterocytes with brush border, Paneth cells may be present
- Mucin: Sialomucin predominant
- Lower malignant potential

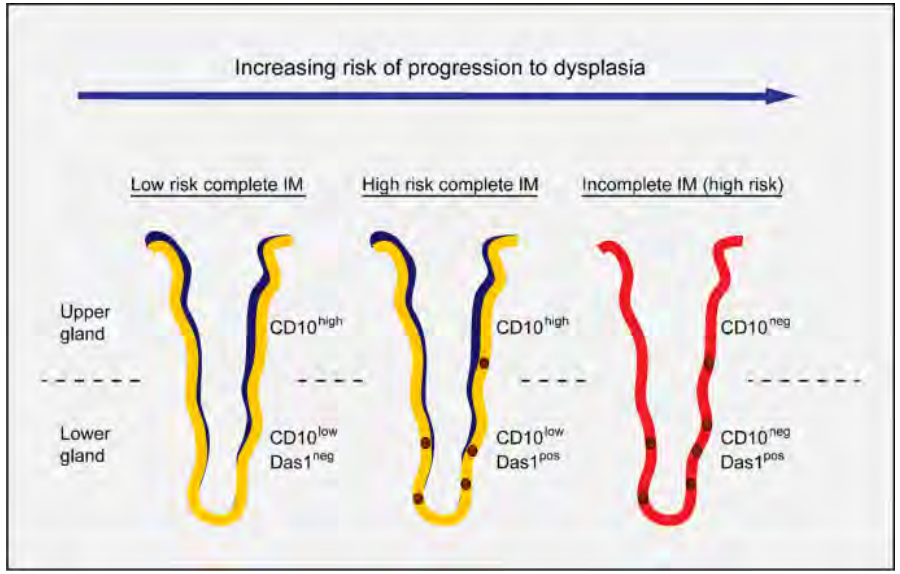


Morgan et al. ACG Clinical Guidelines. Diagnosis and Management of Gastric Premalignant Conditions. ACG. 2025

GASTRIC INTESTINAL METAPLASIA

Incomplete Intestinal Metaplasia(Types II and III)

- Resembles colonic epithelium, irregular mucin droplets, absence of mature absorptive cells and disorganised architecture
- Mucin: Sulfomucin common
- Higher GC risk



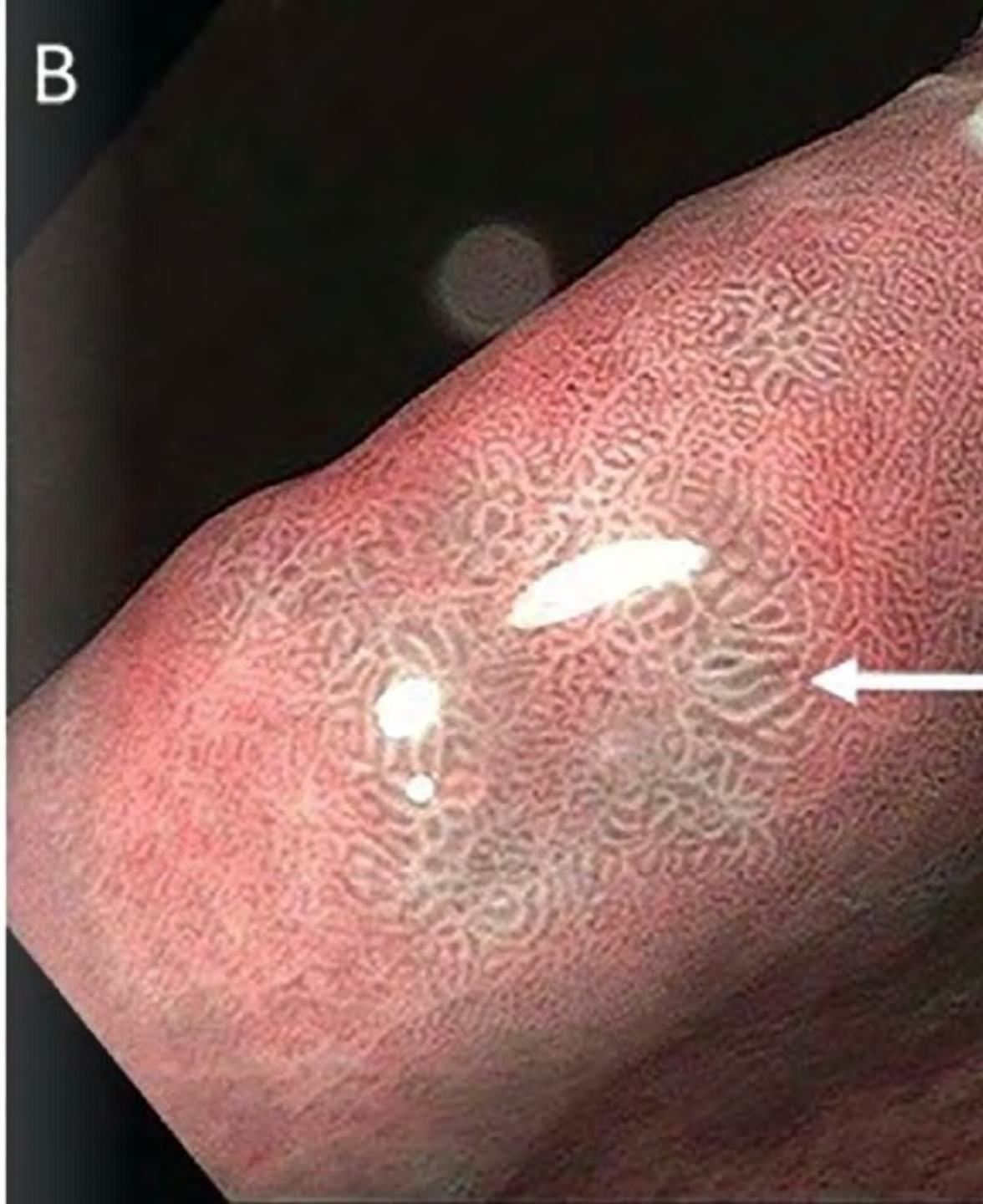
Morgan et al. ACG Clinical Guidelines. Diagnosis and Management of Gastric Premalignant Conditions. ACG. 2025

GASTRIC INTESTINAL METAPLASIA

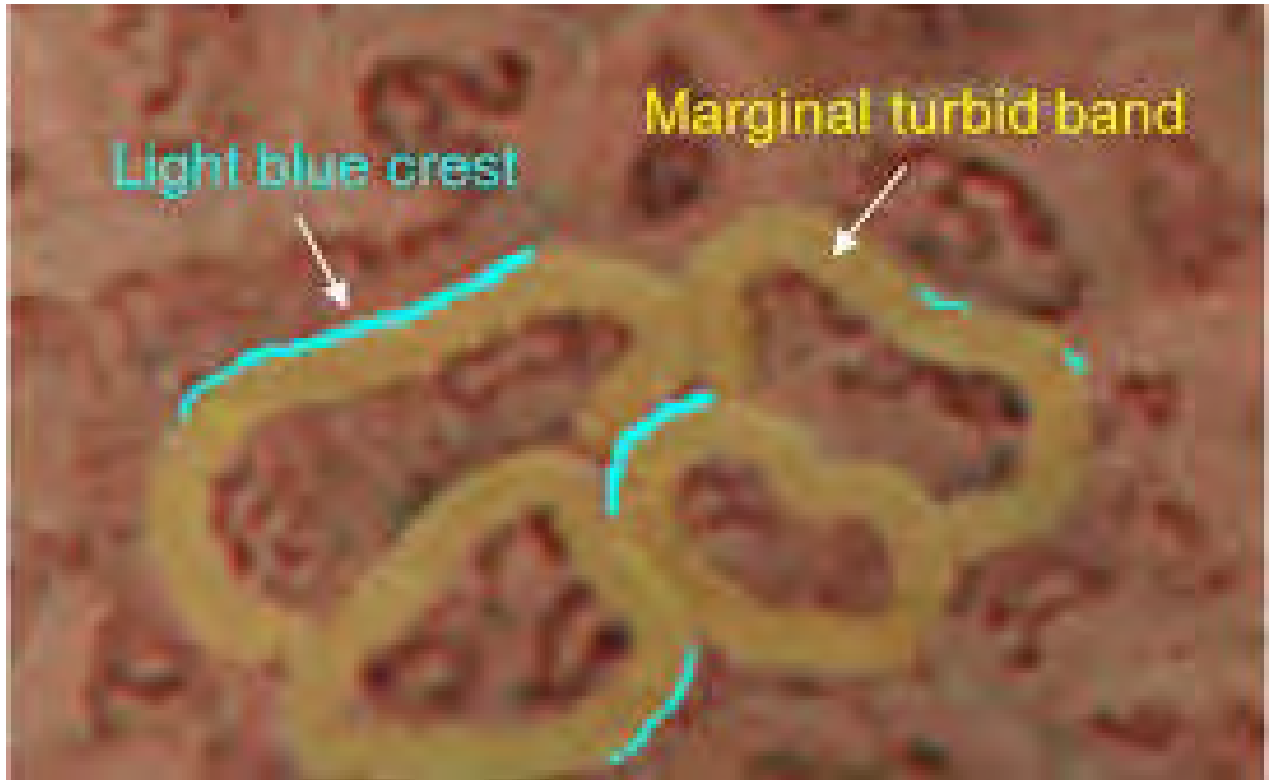
Endoscopic Features

- White light: Pale plaques, patchy discoloration, loss of rugal pattern, mucosal atrophy

- Narrow Band Imaging: Light blue crest sign, Marginal turbid bands and tubulovillous mucosal pattern



GASTRIC INTESTINAL METAPLASIA



Sydney Biopsy Protocol

- 5 biopsies
- 2 antrum, incisura and 2 corpus
- MAPS III- 2 and 2 with targeted

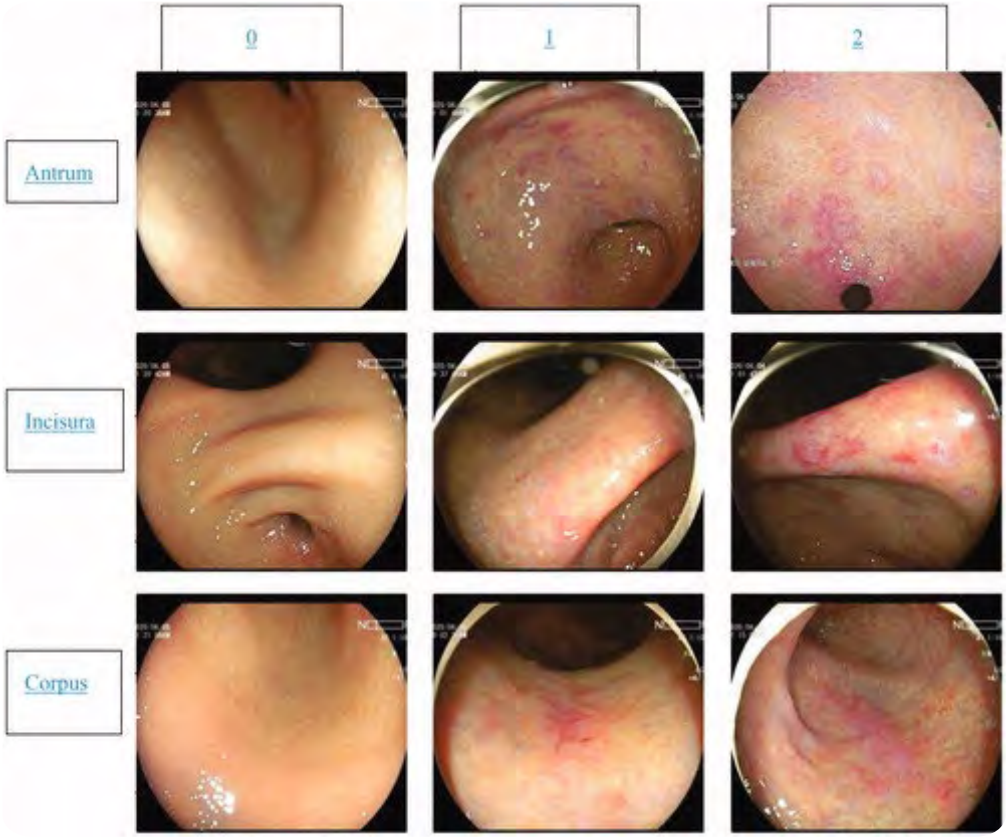
GASTRIC INTESTINAL METAPLASIA

Operative Link on Gastritis Assessment (OLGA)

- Histological staging used to assess severity of gastric atrophy and map its location
- Stages 0-IV

Operative Link on Gastric Intestinal Metaplasia (OLGIM)

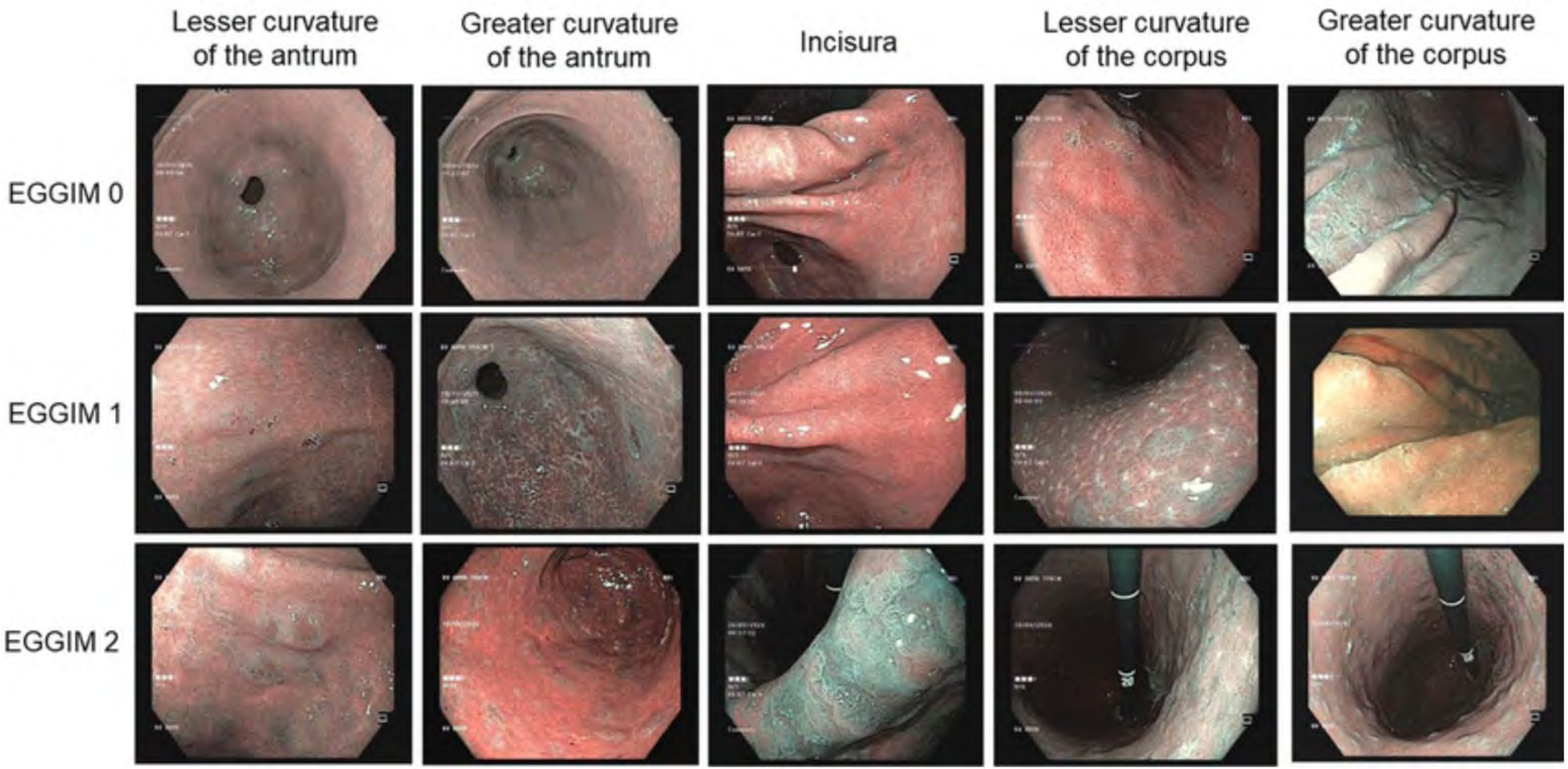
- Scores to measure extent of intestinal metaplasia to assist in prediction of risk of GC
- Stages 0-IV



GASTRIC INTESTINAL METAPLASIA

Endoscopic Grading of Gastric Intestinal Metaplasia (EGGIM)

- Scoring during endoscopy that assist in evaluating extent of stomach lining changes that could precede GC
- Use of advanced imaging techniques that examine five specific zones



Representative endoscopic images illustrating the EGGIM scoring system across five gastric sites. Images demonstrate NBI findings scored from 0 to 2 in the antrum (lesser and greater curvature), incisura angularis, and corpus (lesser and greater curvature). EGGIM 0: no visible IM; EGGIM 1: focal IM (≤ 30% of the area); and EGGIM 2: diffuse IM (> 30% of the area). These visual references were used in internal consensus training sessions to standardize EGGIM scoring among endoscopists

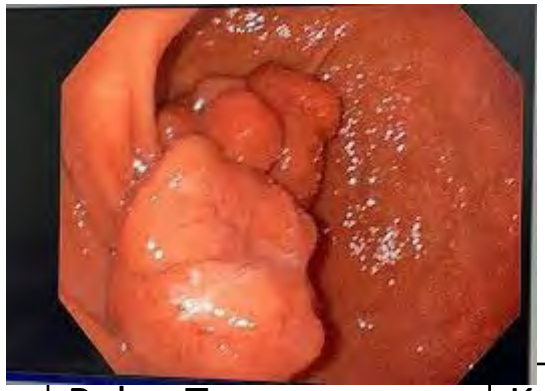
Trinh NA, et al. The predictive value of EGGIM score. 2026

GASTRIC POLYPS

GASTRIC POLYPS

Category	Malignant Potential	Typical Management	Examples
Good	Very low	Observation/limited biopsy	Fundic gland polyps, inflammatory fibroid polyps, ectopic pancreas
Bad	Intermediate/premalignant	Surveillance or resection	Hyperplastic polyps, adenomas, Type 1/2 NETs, hamartomatous polyps
Ugly	High/overt malignancy	Definitive oncologic management	Type 3 NETs, early gastric cancer

Costa et al. Novel Classfiication of gastric polyps. 2024



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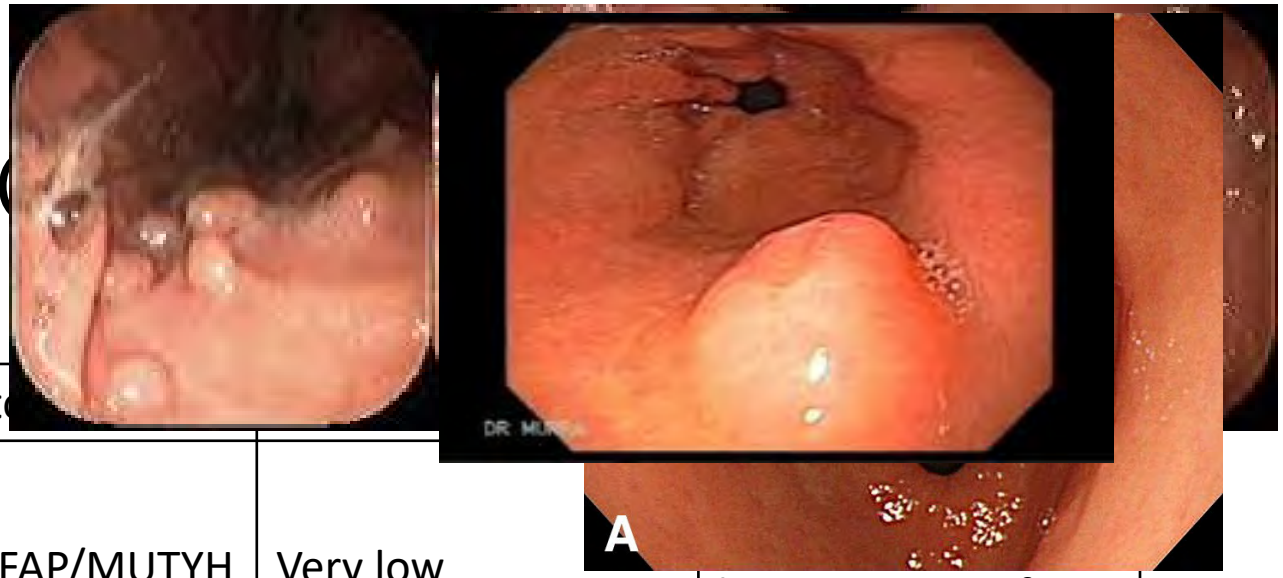


Polyp Type	Key Features	Risk Fact		
Fundic gland polyp (FGP)	Small, smooth sessile body/fundus polyps	PPI use, FAP/MUTYH	Very low	lesions; resect if >1 cm or atypical
Inflammatory fibroid polyp	Rare submucosal lesion, often antral	PDGFRA mutation	Very low	Endoscopic/surgical resection if symptomatic
Ectopic pancreas	Subepithelial lesion with central umbilication	Congenital	Benign	Usually no follow-up
Sporadic hamartomatous polyp	Rare benign lesion	Variable	Low	Individualized

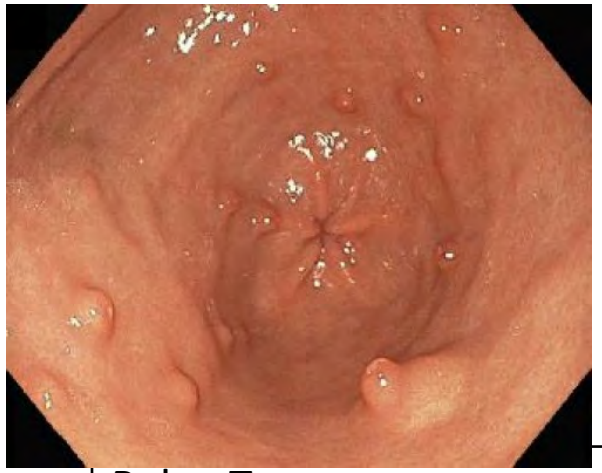
Costa et al. Novel Classfiication of gastric polyps. 2024

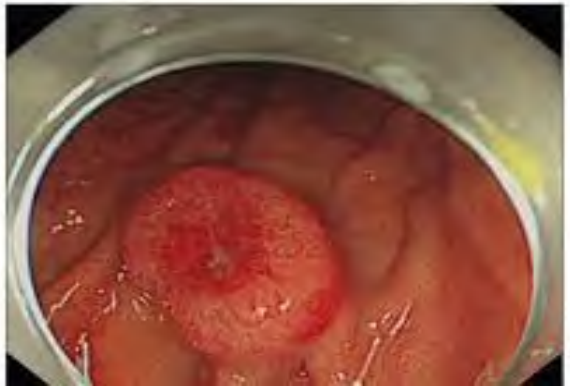
THE GOOD

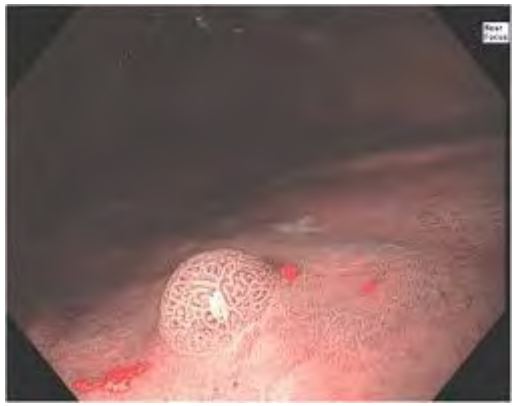
Red Flags requiring resection:

- More than 1 cm
- Antral location
- Irregular surface
- Ulceration
- Erythema
- Abnormal NBI vessels

Costa et al. Novel Classfiication of gastric polyps. 2024





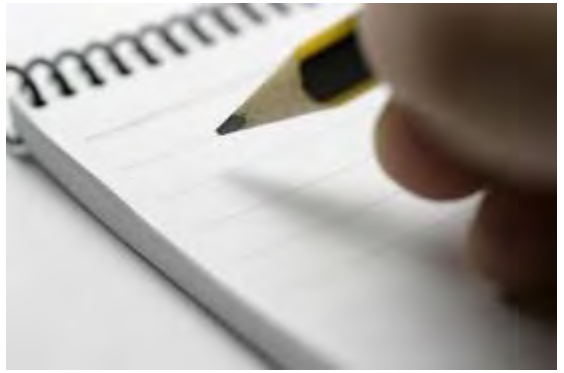


Polyp Type	Background Condition	Dysplasia/Cancer Risk	Key Management
Hyperplastic polyp	Helicobacter pylori infection, chronic gastritis	Dysplasia ~5–15%; cancer <1%	Remove if >1 cm or symptomatic
Gastric adenoma	Atrophy, intestinal metaplasia	High malignant risk	Complete endoscopic resection
Type 1 gastric NET	Autoimmune gastritis	Low metastatic risk	Endoscopic removal + surveillance
Type 2 gastric NET	MEN1/Zollinger-Ellison	Moderate metastatic risk	Treat gastrinoma + resect
Syndromic hamartomatous polyps	Peutz-Jeghers, juvenile polyposis	Significant cancer risk	Genetic evaluation and surveillance

Costa et al. Novel Classfiication of gastric polyps. 2024

THE BAD

- Hyperplastic
  - Associated with chronic gastritis, atrophic gastritis and Hp Infection
  - However malignancy risk increases with size more than 1 cm, pedunculated morphology and post gastrectomy



- Gastric adenomas
  - All need resection and surveillance

- GNETs

Type	Association	Gastrin Level	Metastatic Risk	Prognosis
Type 1	Autoimmune gastritis	High	Low	Excellent
Type 2	MEN1/ZES	High	Intermediate	Good
Type 3	Sporadic	Normal	High	Poorer

THE UGLY

Lesion	Key Features	Prognosis	Management
Type 3 gastric NET	Solitary, aggressive	Poorer	Surgery ± oncology therapy
Early gastric cancer	Dysplasia/invasive cancer	Variable	ESD/EMR or surgery

Type 3 GNETs (antrum and fundus)

- are usually solitary,
- larger,
- invasive,
- highly metastatic,
- and require aggressive oncologic therapy.

Costa et al. Novel Classfiication of gastric polyps. 2024

KEY RED FLAGS

Feature	Concern
Ulceration	Dysplasia/cancer
Irregular vascularity	Neoplasia
Surface depression	Invasive potential
Large size (>1 cm)	Increased malignant risk
Rapid growth	Neoplasia
Solitary large lesion	Adenoma/NET/cancer

Costa et al. Novel Classfiication of gastric polyps. 2024

MANAGEMENT PRINCIPLES

Lesion Type	Suggested Action
Small classic FGP	Biopsy ± observe
Hyperplastic polyp >1 cm	Resect
Any adenoma	Complete resection
NET suspected	EUS + staging
Suspicious lesion	En bloc resection/staging
Background gastritis present	Sydney mapping biopsies

Costa et al. Novel Classfiication of gastric polyps. 2024

AGA 2026 GASTRIC POLYPS

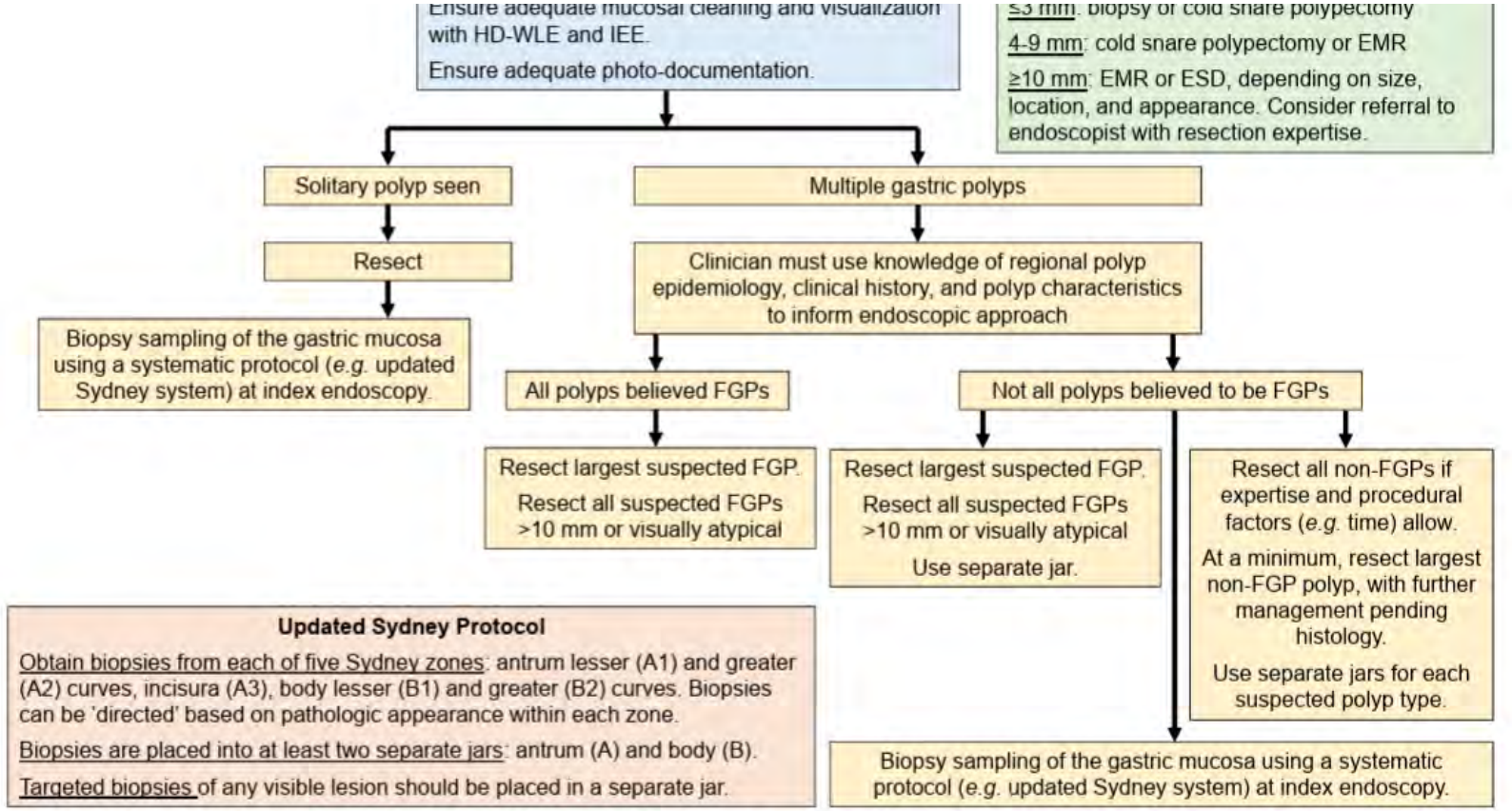


Figure 2. Proposed management strategy for newly visualized gastric polyps. This advice is appropriate for countries where *H pylori* prevalence is low, and PPI use is common, such as the United States. This advice pertains to patients without a

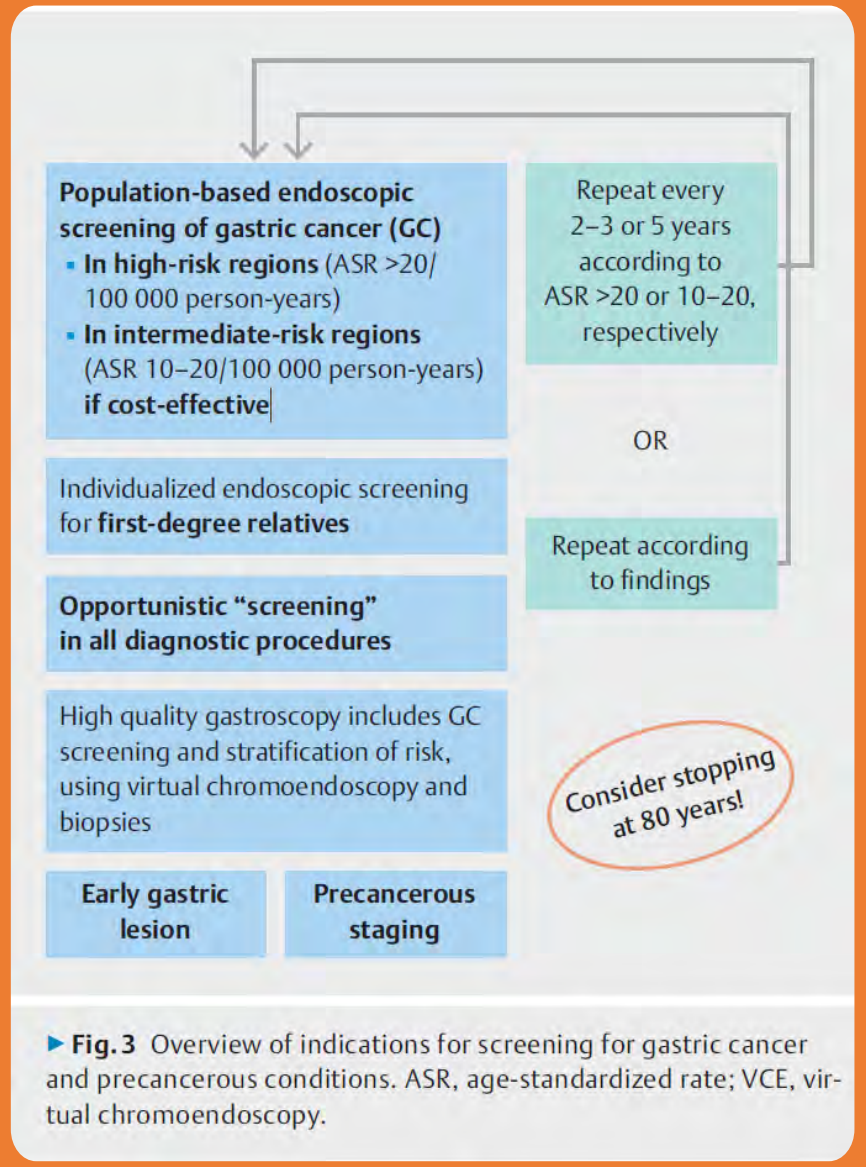
AGA 2026 GASTRIC POLYPS

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	

SCREENING, MANAGEMENT AND SURVEILLANCE

SCREENING



▶ **Fig. 3** Overview of indications for screening for gastric cancer and precancerous conditions. ASR, age-standardized rate; VCE, virtual chromoendoscopy.

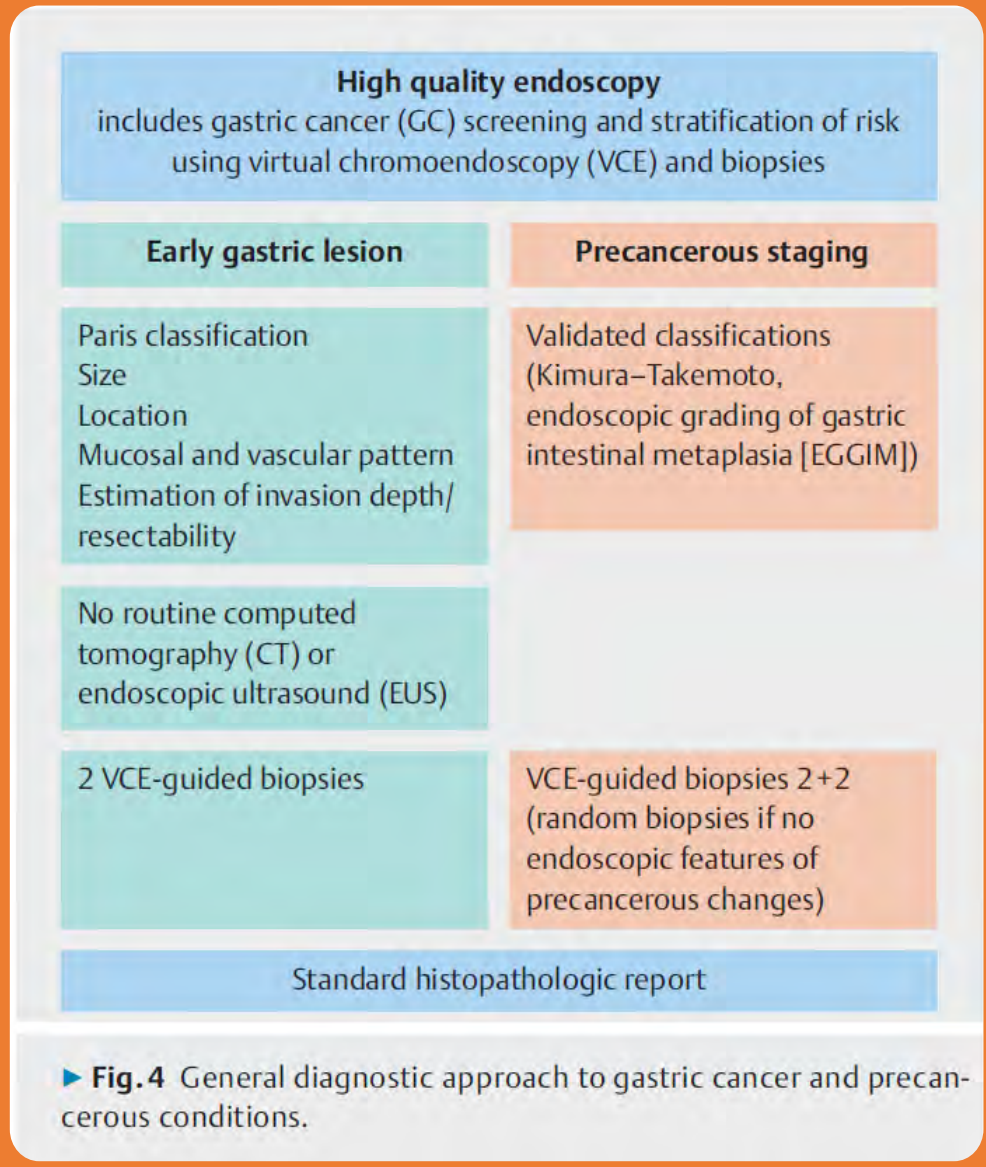
SCREENING

- First degree relative with GC
  - H Pylori non invasive screening and eradication between ages 20 and 30
  - Endoscopic screening at age 45 years or at 10 years prior to age of GC diagnosis

- Suspicious lesions need to be properly described (size, morphology according to Paris, ulcerative, location, vascular and mucosal patterns. Photo document and 2 targeted biopsies

- Use of Kimura-Takemoto or EGGIM for atrophy and IM
- Biopsy of 2 fragments from antrum/incisura and 2 from corpus, placed in two separate pots

SCREENING



▶ **Fig. 4** General diagnostic approach to gastric cancer and precancerous conditions.

MANAGEMENT

- Non visible Dysplasia
  - Refer for high quality white-light endoscopy with virtual chromoendoscopy, staging of precancerous conditions and Hp testing
  - If still lesions not seen again→ repeat high quality endoscopy in 6 months if HGD and 12 months if LGD/indefinite for dysplasia

- Visible Dysplasia
  - Needs to undergo staging and treatment

MANAGEMENT

Staging of the disease

Table 2. Diagnostic and staging investigations in gastric cancer.

Procedure	Purpose
FBC	Assess for iron deficiency anaemia
Renal and liver function	Assess renal and liver function to determine appropriate therapeutic options
Endoscopy and biopsy	Obtain tissue for diagnosis, histological classification and molecular biomarkers, e.g. HER2 status
CT of thorax + abdomen ± pelvis	Staging of tumour – to detect local/distant lymphadenopathy and metastatic disease or ascites
EUS	Accurate assessment of T and N stage in potentially operable tumours Determine the proximal and distal extent of tumour
Laparoscopy + washings	Exclude occult metastatic disease involving peritoneum/diaphragm
PET, if available	May improve detection of occult metastatic disease in some cases. Often negative in diffuse-type gastric cancer
Assessment of nutritional status	May detect relevant dietary and nutritional deficiencies in both localised and advanced disease settings

CT, computed tomography; EUS, endoscopic ultrasound; FBC, full blood count; HER2, human epidermal growth factor receptor 2; N, node; PET, positron emission tomography; T, tumour.

Lordick F et al. Ann Oncol 2022

MANAGEMENT

- In the TNM staging system,
- **T (Tumor)** indicates the depth of penetration
  - T1a denotes a tumor that invades the lamina propria or mucosa,
  - T1b denotes invasion of the submucosa,
  - T2 denotes invasion of the muscularis propria,
  - T3 denotes invasion of the subserosal connective tissue,
  - T4a denotes invasion of the serosa (visceral peritoneum), and
  - T4b denotes invasion into adjacent organs or structures.
- **N (Nodes**) indicates the amount of lymph node invasion:
  - N0 denotes no lymph node involvement,
  - N1 denotes involvement of 1 to 2 lymph nodes,
  - N2 denotes involvement of 3 to 6 lymph nodes, and
  - N3 denotes involvement of 7 or more lymph nodes.
- **M (Metastasis)** indicates the presence of metastases, with
  - M0 denoting no metastases and
  - M1 denoting distant metastases, including positive peritoneal cytology

MANAGEMENT

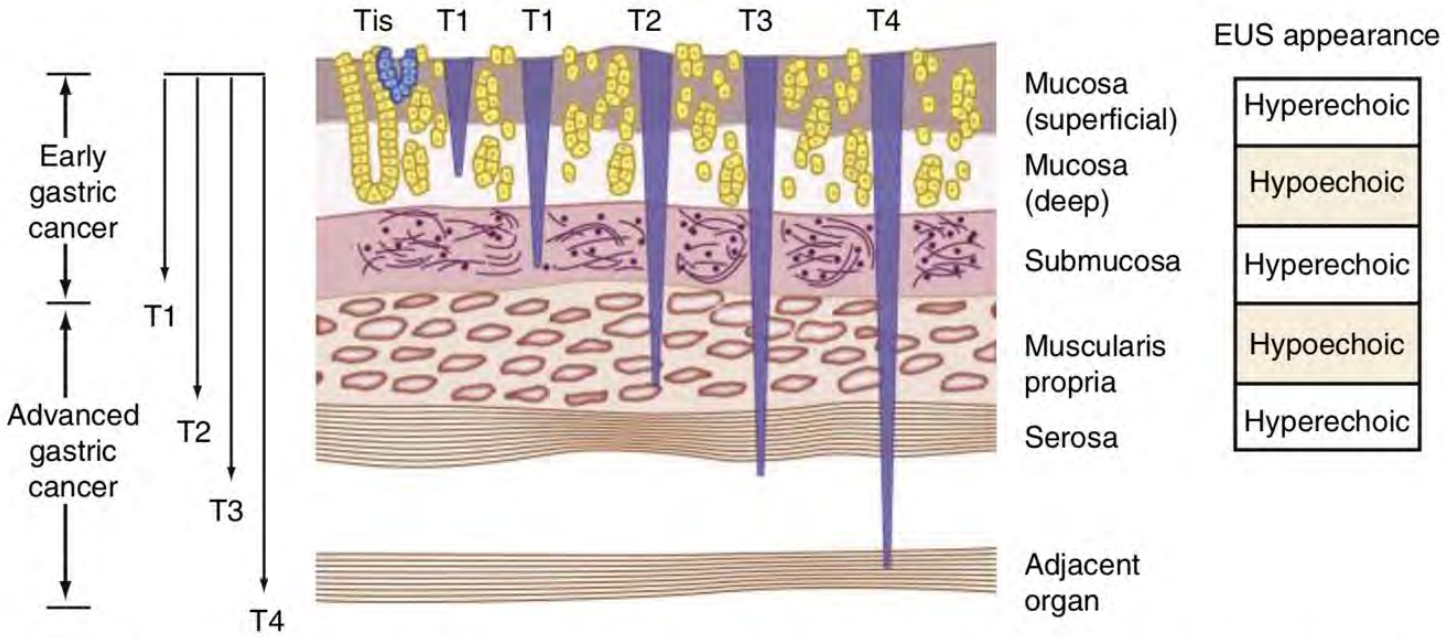
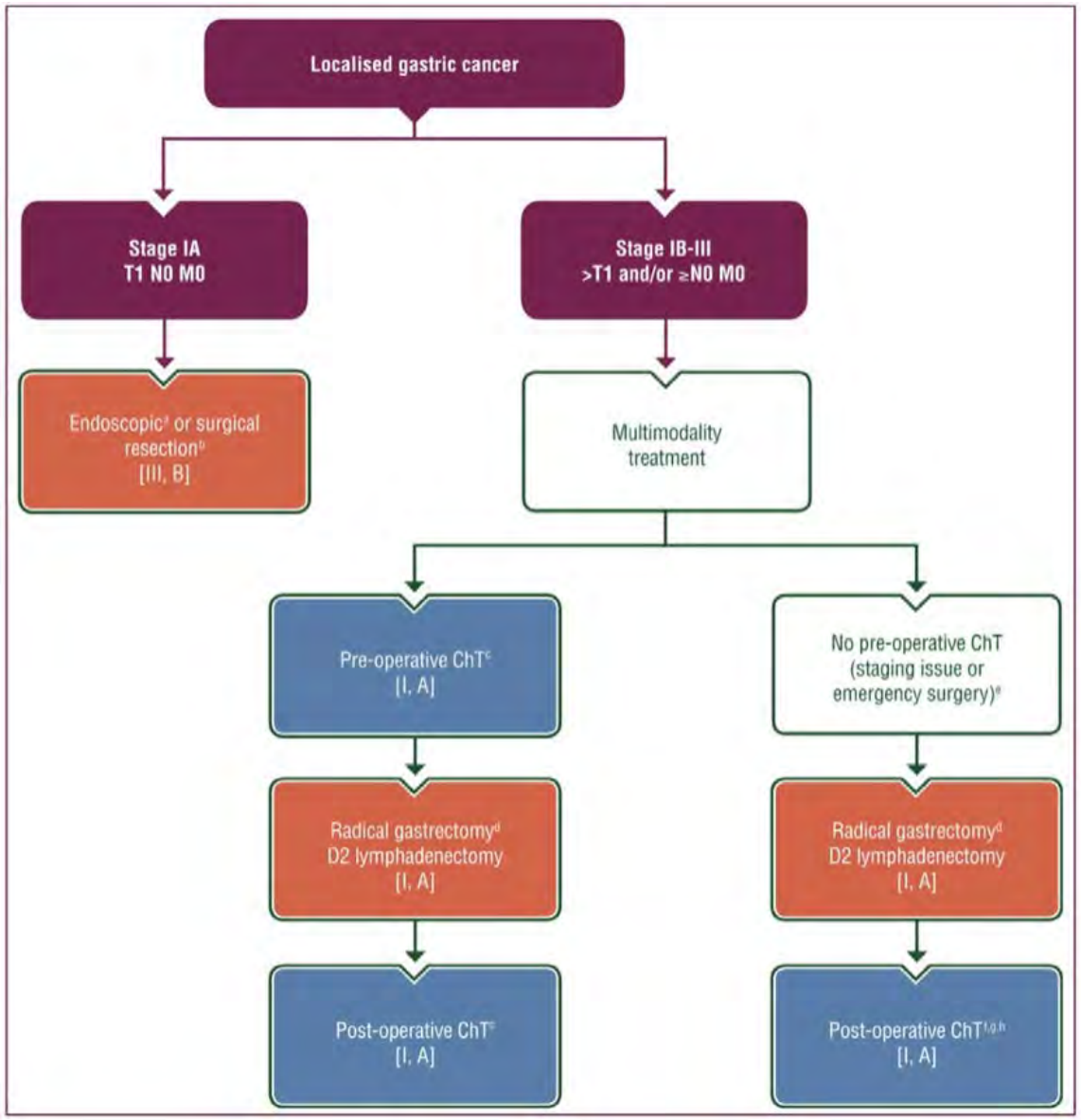


Fig. 54.8 Classification of gastric adenocarcinoma by depth of invasion (T classification). In the TNM classification, T denotes depth of invasion: Tis designates carcinoma in situ; T1 tumors are confined to the mucosa (T1a) and submucosa (T1b); T2 tumors invade the muscularis propria but not the serosa; T3 tumors penetrate the subserosal connective tissue without involving the visceral peritoneum or contiguous structures; and T4 tumors invade the serosa (visceral peritoneum) and may involve adjacent organs and tissues. In early gastric cancer, the disease is confined to the mucosa and submucosa (T1), regardless of nodal involvement.

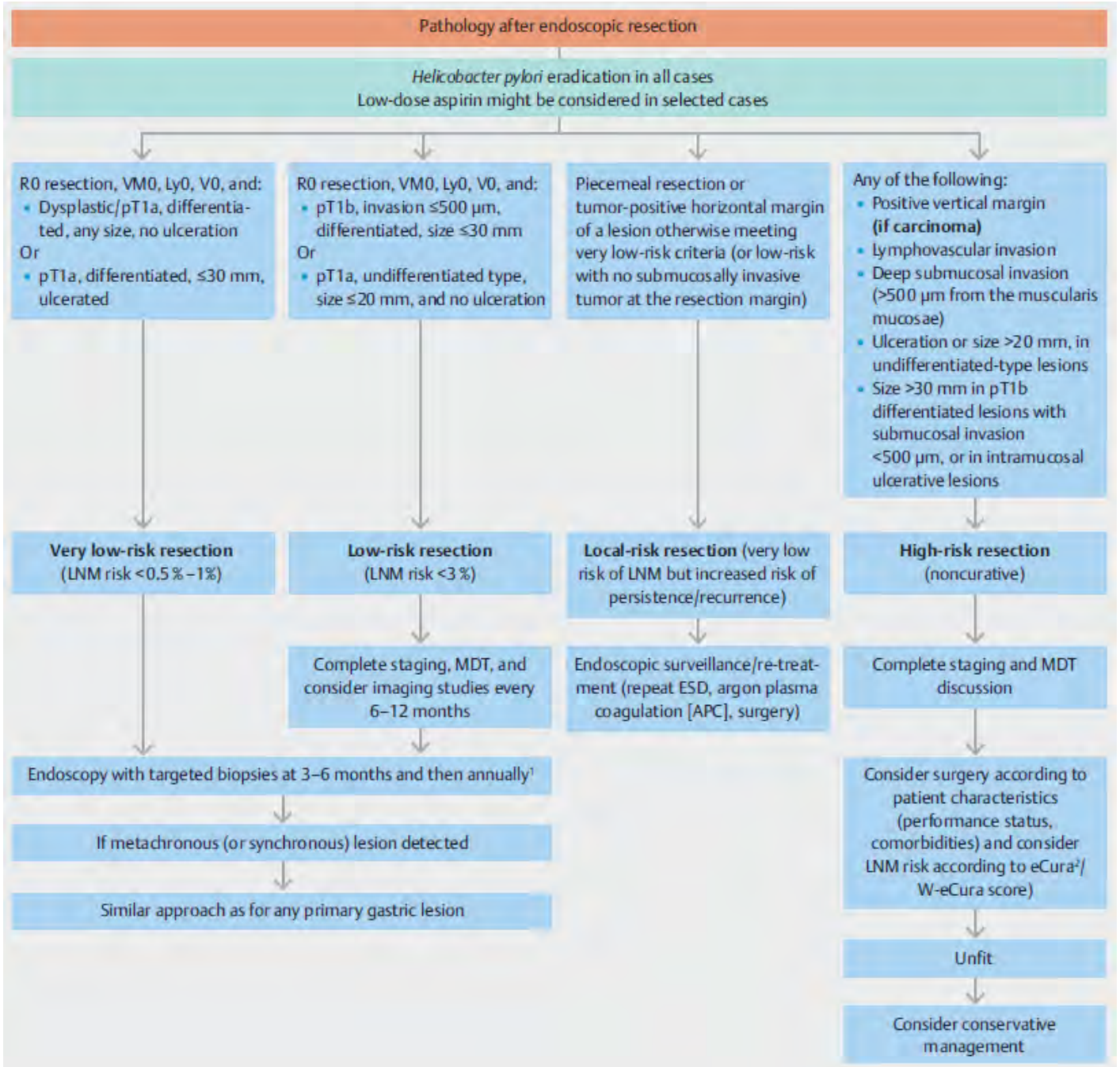

Localised gastric cancer
Stage IA
T1 N0 M0
Stage IB-III
>T1 and/or ≥N0 M0
Endoscopic[a] or surgical resection[b]
[III, B]
Multimodality treatment
Pre-operative ChT[c]
[I, A]
No pre-operative ChT (staging issue or emergency surgery)[e]
Radical gastrectomy[d]
D2 lymphadenectomy
[I, A]
Radical gastrectomy[d]
D2 lymphadenectomy
[I, A]
Post-operative ChT[c]
[I, A]
Post-operative ChT[f,g,h]
[I, A]

MANAGEMENT

MANAGEMENT

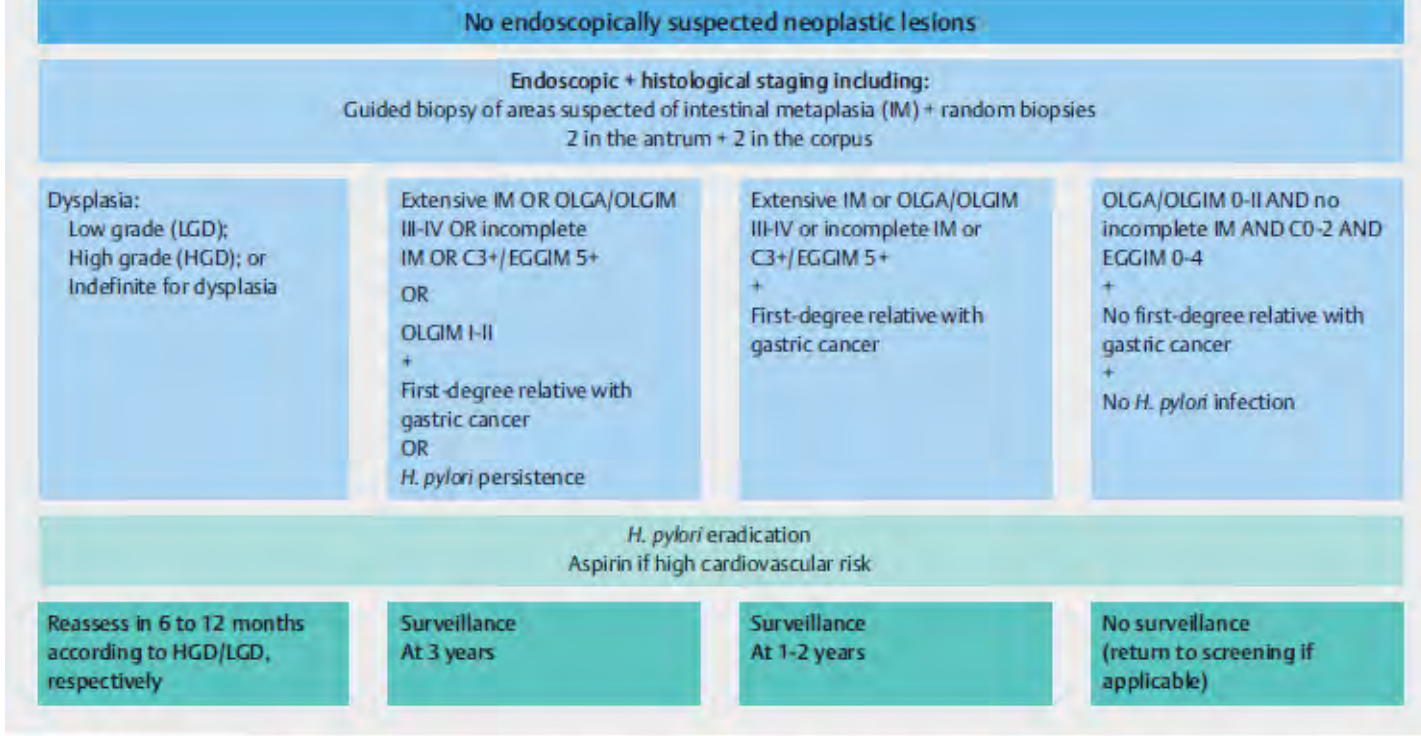
- According to MAPS III
  - ESD is treatment of choice for superficial gastric lesions
  - ESD clinically staged as dysplastic or as intramucosal carcinoma (any size if not ulcerated and <30mm if ulcerated)
  - EMR being an alternative if Paris 0-IIa lesions with size <10mm with low likelihood of malignancy
  - ESD for malignant lesions with no ulcerative findings
    - minimal submucosal invasion if differentiated and </= 30mm
    - Intramucosal when undifferentiated and </= 20 mm

MAPS III GUIDELINES 2025

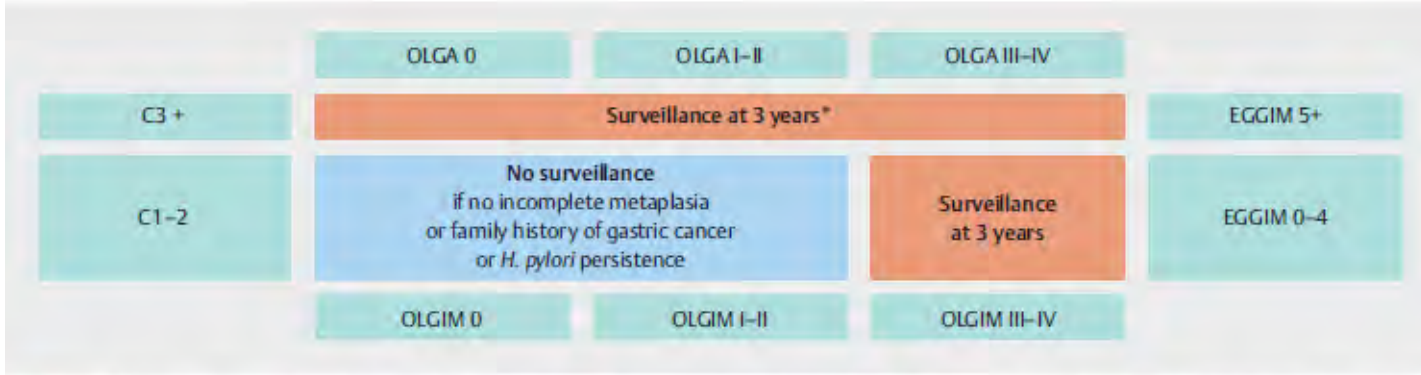

Pathology after endoscopic resection
Helicobacter pylori eradication in all cases
Low-dose aspirin might be considered in selected cases
R0 resection, VM0, Ly0, V0, and:
• Dysplastic/pT1a, differentiated, any size, no ulceration
Or
• pT1a, differentiated, ≤30 mm, ulcerated
R0 resection, VM0, Ly0, V0, and:
• pT1b, invasion ≤500 µm, differentiated, size ≤30 mm
Or
• pT1a, undifferentiated type, size ≤20 mm, and no ulceration
Piecemeal resection or tumor-positive horizontal margin of a lesion otherwise meeting very low-risk criteria (or low-risk with no submucosally invasive tumor at the resection margin)
Any of the following:
• Positive vertical margin **(if carcinoma)**
• Lymphovascular invasion
• Deep submucosal invasion (>500 µm from the muscularis mucosae)
• Ulceration or size >20 mm, in undifferentiated-type lesions
• Size >30 mm in pT1b differentiated lesions with submucosal invasion <500 µm, or in intramucosal ulcerative lesions
Very low-risk resection (LNM risk <0.5 %–1%)
Low-risk resection (LNM risk <3 %)
Local-risk resection (very low risk of LNM but increased risk of persistence/recurrence)
High-risk resection (noncurative)
Complete staging, MDT, and consider imaging studies every 6–12 months
Endoscopic surveillance/re-treatment (repeat ESD, argon plasma coagulation [APC], surgery)
Complete staging and MDT discussion
Endoscopy with targeted biopsies at 3–6 months and then annually[1]
If metachronous (or synchronous) lesion detected
Similar approach as for any primary gastric lesion
Consider surgery according to patient characteristics (performance status, comorbidities) and consider LNM risk according to eCura[2]/ W-eCura score)
Unfit
Consider conservative management

SURVEILLANCE

3 yearly Endoscopy	• Extensive endoscopic changes (C3+ or EGGIM 5+) • Advanced histological stages of atrophic gastritis (severe CAG or GIM, OLGA/OLGIM III/IV) • GIM at single location with GC family history or persistent H pylori
1 to 2 yearly Endoscopy	• FDR with the above features
No Surveillance	• Mild to moderate CAG or GIM restricted to the antrum

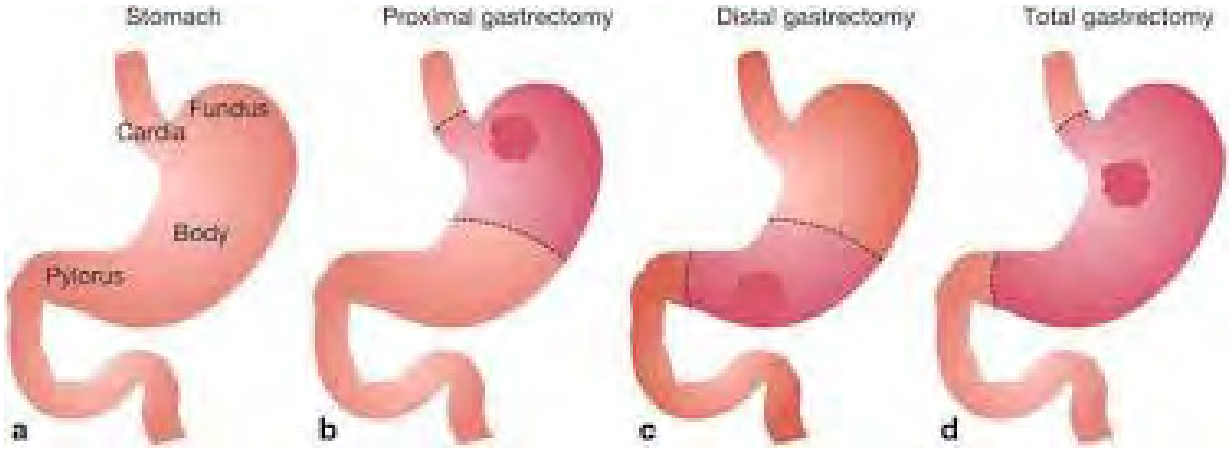


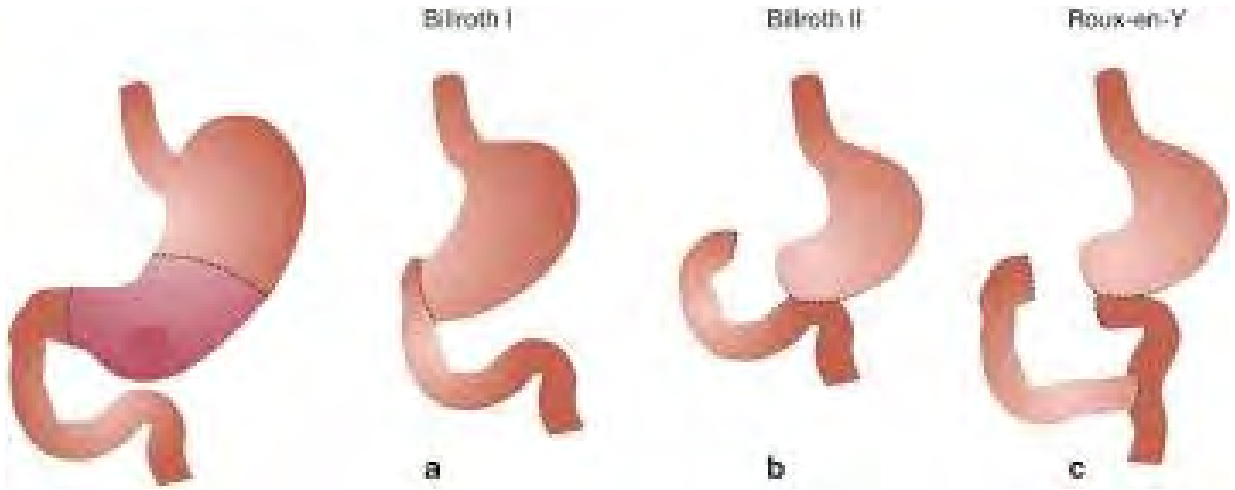
▶ **Fig. 10** Management of precancerous conditions (and nonvisible dysplasia or indefined). C3+, C0–2, Kimura–Takemoto classification; EGGIM, endoscopic grading of gastric intestinal metaplasia; OLGA, operative link on gastritis assessment; OLGIM, operative link on gastric intestinal metaplasia.



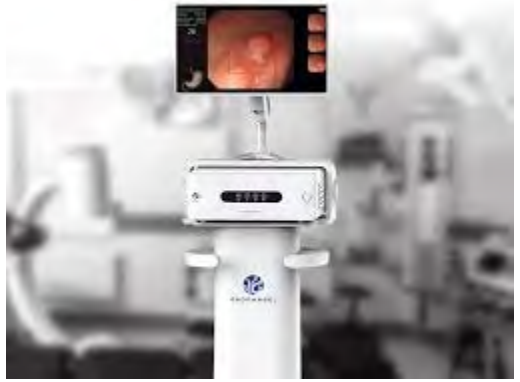
▶ **Fig. 11** Comprehensive approach: both endoscopic and histological information must be considered for stratification of risk and allocation of individuals to different surveillance regimes (if no autoimmune gastritis is diagnosed). C3+, C1–2, Kimura–Takemoto classification; EGGIM, endoscopic grading of gastric intestinal metaplasia; OLGA, operative link on gastritis assessment; OLGIM, operative link on gastric intestinal metaplasia. *Adjust to 1–2 years if first-degree relatives with gastric cancer.

MANAGEMENT


Stomach
Fundus
Cardia
Body
Pylorus
a
Proximal gastrectomy
b
Distal gastrectomy
c
Total gastrectomy
d


Billroth I
a
Billroth II
b
Roux-en-Y
c

THE FUTURE



<u>Artificial Intelligence for Early Gastric Cancer</u>

- Uses advanced computer vision and deep learning algorithms to detect subtle changes

- Convolutional Neural Networks (CNN), Real-time Endoscopy (EndoAngel, Olysense and GI Genius) and Lesion Delineation and Staging

- However, still not recommended for use in guidelines

Lei et al. Artificial intelligence-assisted diagnosis of early gastric cancer. 2025

TAKE HOME MESSAGES

- Gastric cancer develops through a stepwise inflammatory-metaplastic sequence.
- H. pylori remains the most important modifiable risk factor.
- High-definition endoscopy and NBI are central to detection.
- Extensive IM and OLGIM III–IV require surveillance.
- ESD is preferred for suitable early neoplastic lesions.
- MAPS III emphasises personalised risk-based surveillance.
- Every gastroscopy is an opportunity for gastric cancer prevention.

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