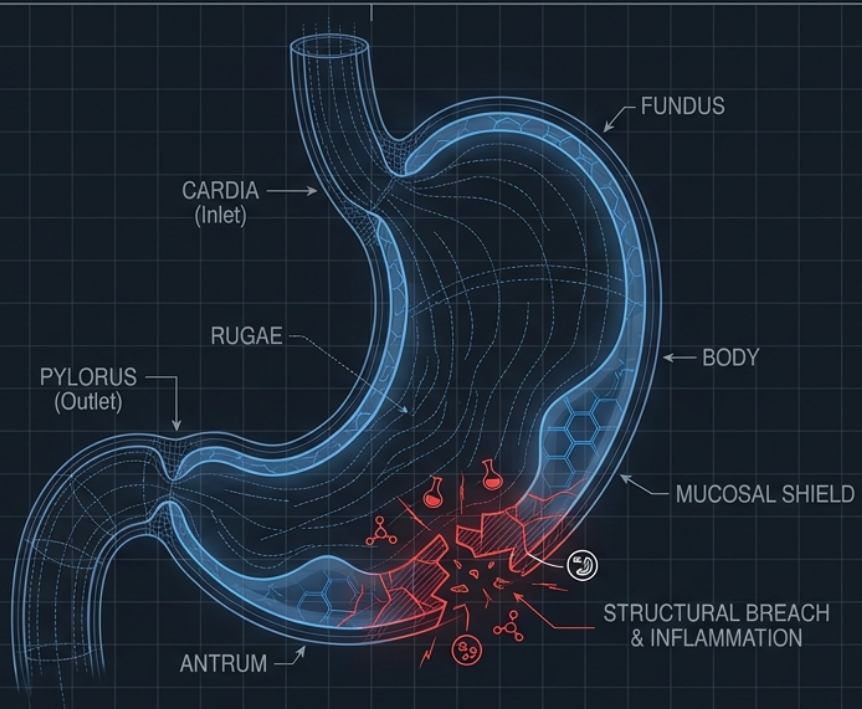


بِسْمِ اللَّهِ الرَّحْمَنِ الرَّحِيمِ

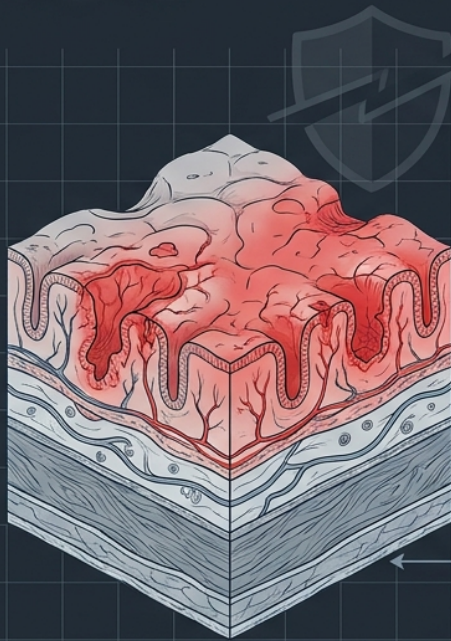


Gastritis: Breaching the Mucosal Barrier

A Clinical Blueprint of Etiology, Pathophysiology, and Management

The Target Profile: Defining Gastritis

The Condition



The fundamental inflammation of the gastric mucosa.

Characterized by disruption of the protective mucosal barrier, leading to tissue injury.

MUCOSA
(Inflammation & Damage)

SUBMUCOSA
(Vascular & Neural Support)

MUSCULARIS EXTERNA
(Motility & Structure)

SEROSA
(Protective Outer Layer)

Demographics

50-60

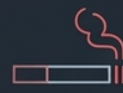
High-Risk Age
(Years of life)

Peak incidence and symptom



Gender Predominance
Significantly more common in men.

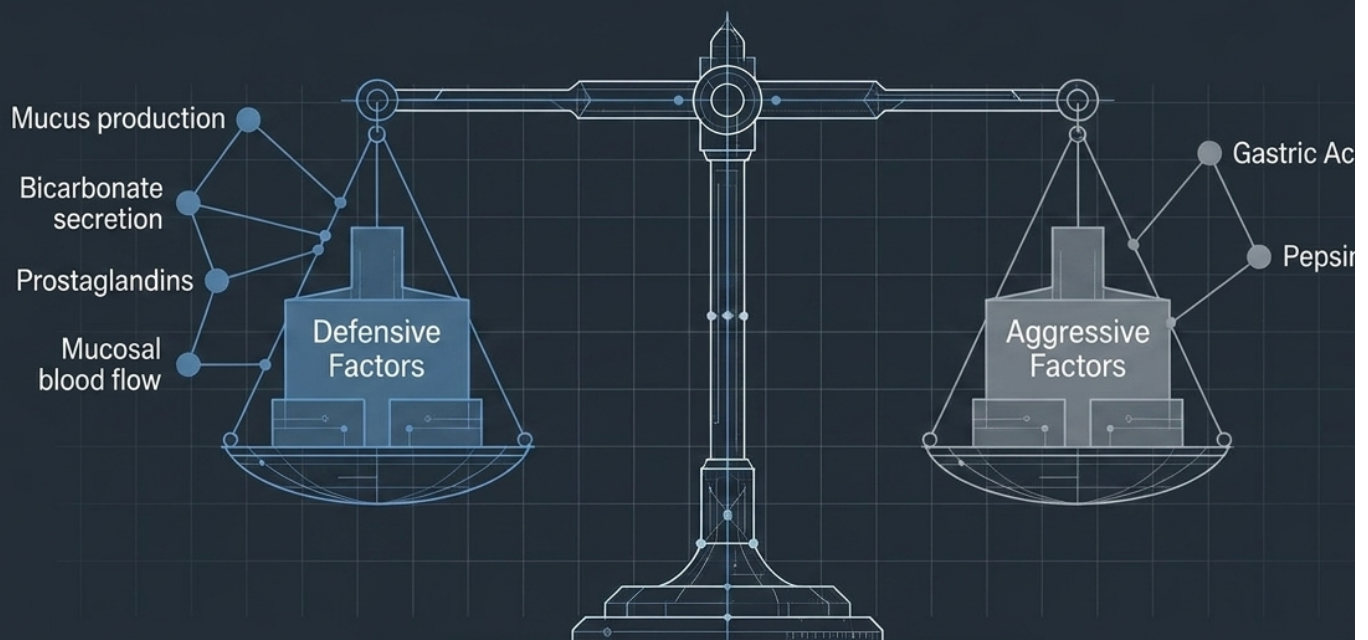
Associated with behavioral and physiological predispositions



Lifestyle Accelerators
Exponentially higher in heavy drinkers and smokers

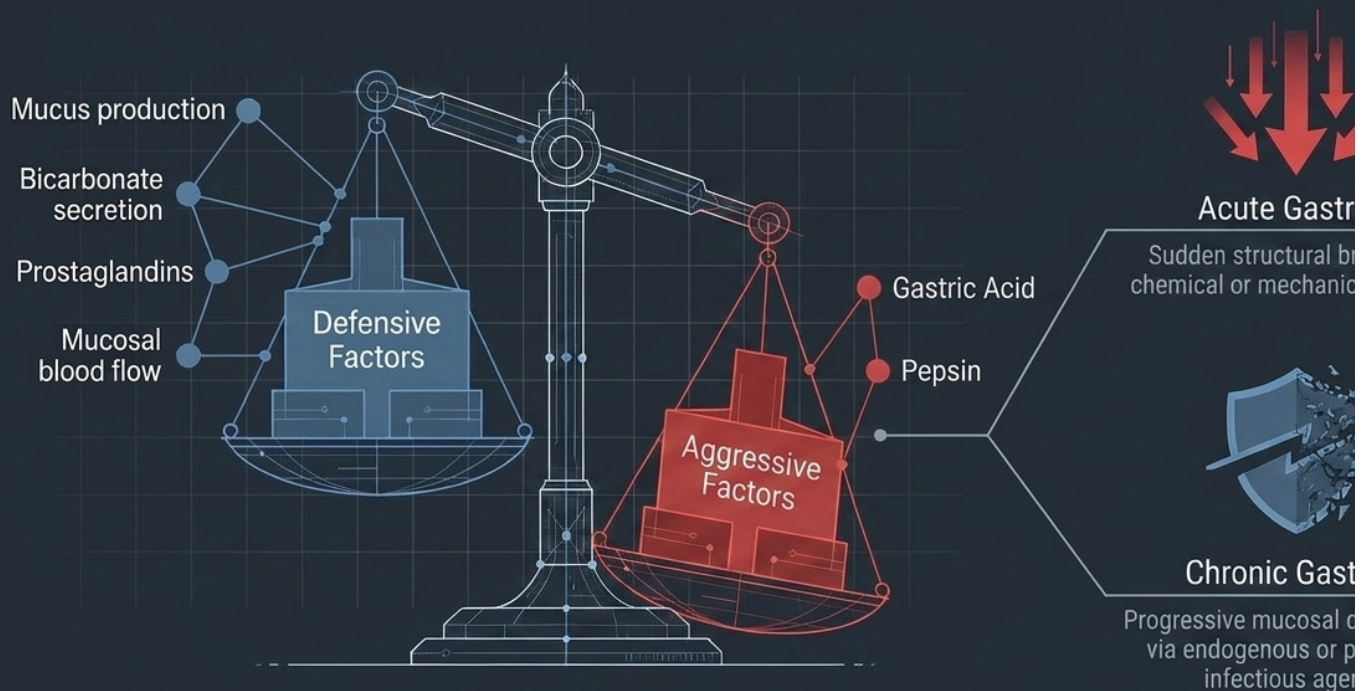
Chronic exposure compromises defenses and increases acid

The Baseline Equilibrium: The Gastric Shield



The gastric mucosa remains intact as long as defensive physiological mechanisms perfectly counter aggressive digestive forces.

The Tipping Point: System Failure



The Gastritis Spectrum: Acute vs. Chronic



Acute Gastritis

Onset: Sudden and immediate.

Triggers: Immediate irritants (NSAIDs, alcohol, acute stress/critical illness).

Cellular Infiltrate: Neutrophils (active inflammation).

Mechanism: Acid erosion of gastric mucosa due to decreased protective prostaglandins and mucus.

Trajectory: Typically short-term and reversible upon trigger removal; severe cases risk gastric ulceration.



Chronic Gastritis

Onset: Gradual and persistent (often silent stages).

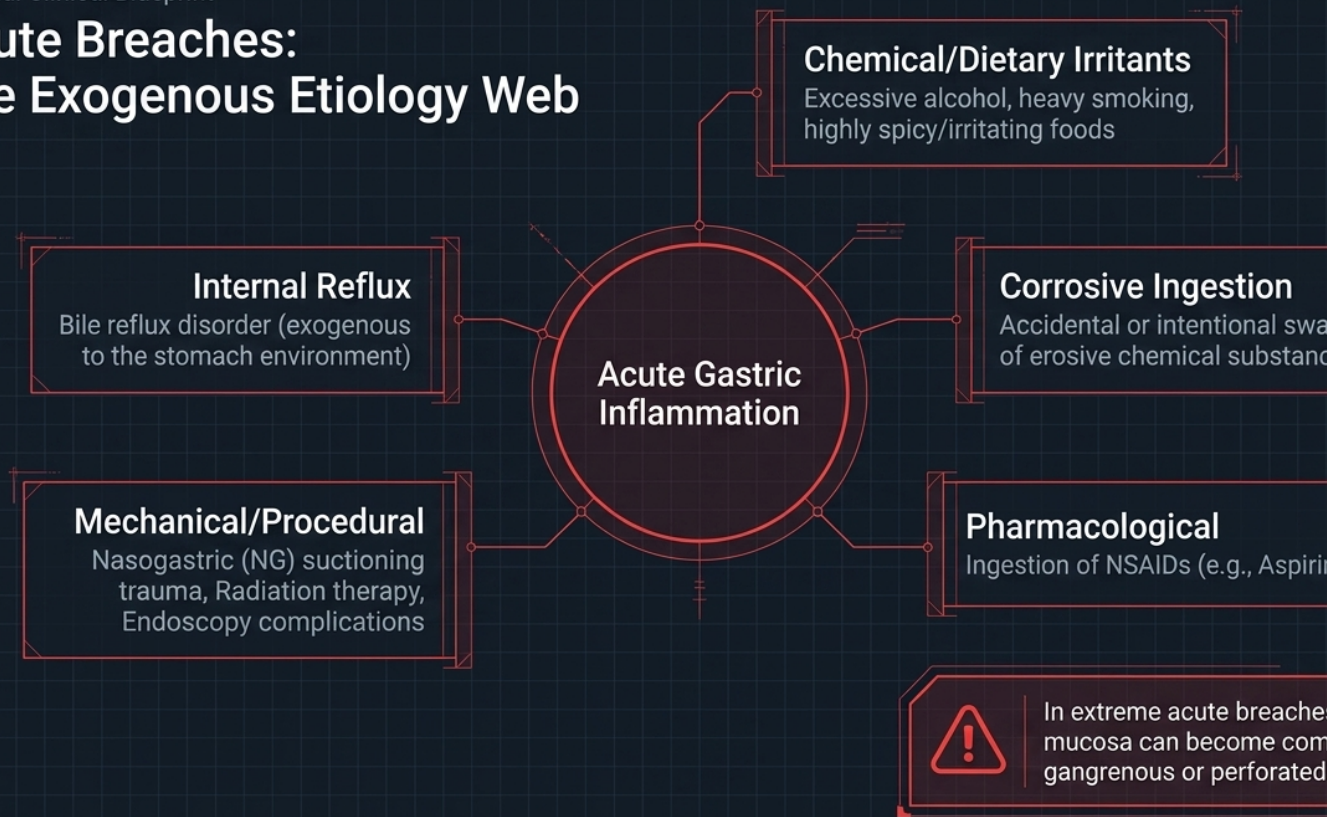
Triggers: Persistent factors (*Helicobacter* infection, autoimmunity, continuous irritants).

Cellular Infiltrate: Lymphocytes and plasma cells (chronic inflammation).

Mechanism: Progressive destruction of glandular epithelial cells.

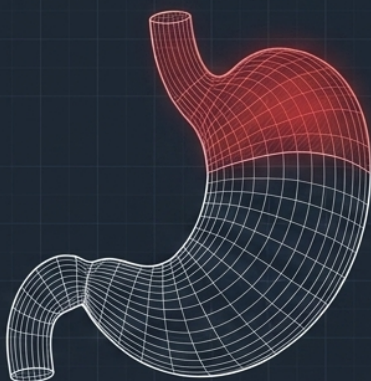
Trajectory: Long-term risk of irreversible gastric atrophy, metaplasia, and neoplastic complications.

Acute Breaches: The Exogenous Etiology Web



Chronic Gastritis: Type A vs. Type B

Type A (Autoimmune)

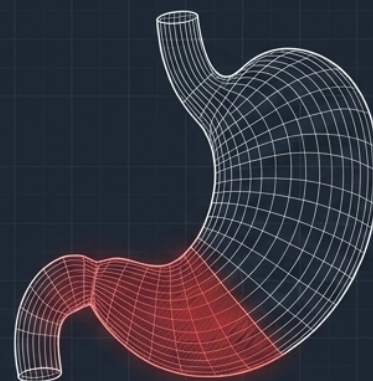


Anatomical Focus:
Fundus

Pathology: Autoimmune destruction. Faulty secretion of Castle's Intrinsic Factor.

Clinical Note: Usually asymptomatic in early stages; high risk for pernicious anemia.

Type B (Infectious)

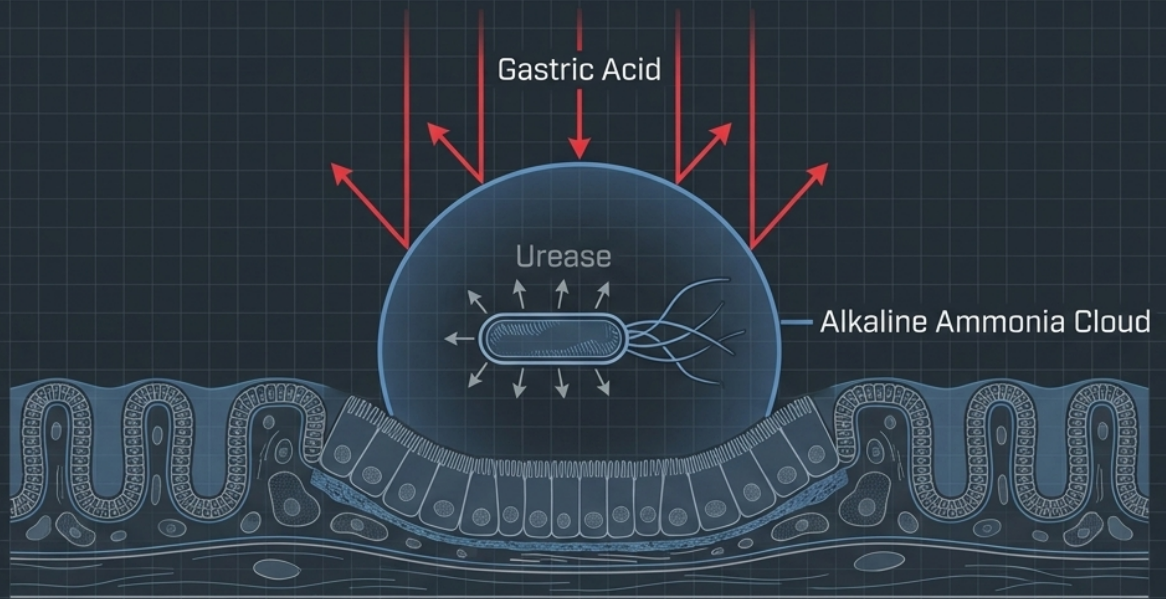


Anatomical Focus:
Antrum and Pylorus

Pathology: Associated heavily with *Helicobacter pylori* infection.

Clinical Note: Most common form of gastritis globally.

The H. Pylori Survival Model



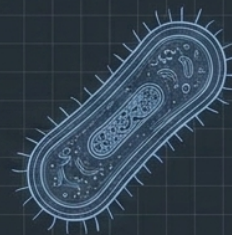
Urease production creates a localized alkaline micro-environment, neutralizing gastric acid and allowing *H. pylori* colonization.

Atypical and Granulomatous Forms



Viral Pathogens

Cytomegalovirus (CMV) infection.



Bacterial & Mycobacteria

Gastric Tuberculosis



Systemic Inflammatory

Crohn's disease involvement of the upper GI tract.



Idiopathic & Multisystem

Systemic diseases as Sarcoidosis, or idiopathic origins.

Sign & Symptoms

- Abdominal pain
- Dyspepsia
- Nausea and vomiting
- Loss of appetite
- Abdominal fullness
- Bloating
- Belching
- Fever and chills
- Hiccups
- Bleeding hematemesis
- Anemia (fatigue, lower of po, short of breathing)
- Nutrient deficiencies (b12)
- Gastric outlet obstruction
- Dehydration

Clinical Manifestations: The Patient Presentation

Acute & Systemic Symptoms

- Headache & Lassitude
- Hiccupping
- Acute Abdominal Pain & Tenderness

Chronic & Upper GI S

- Heartburn & Belching
- Sour taste in mouth
- Dyspepsia & Anorexi (loss of appetite)

Objective Clinical Signs (Warning)

- Hematemesis (Vomiting blood)
- Melena (Dark, tarry stools)
- Halitosis (Severe bad breath)
- Frequent Vomiting

Clinical Manifestations of AMAG



Hematologic Manifestations

- Iron-deficiency anemia (often the earliest clinical sign).
- Pernicious anemia (macrocytic anemia, hyper-segmented neutrophils).
- Hyperhomocysteinemia (carries a risk of thrombosis and pulmonary embolism).



Neurologic Manifestations

- Subacute combined degeneration (SCD) of spinal cord.
- Peripheral neuropathy, paresthesia, ataxia.
- Cognitive impairment, mood disorders, overt psychosis.
- Clinical Pearl: Neurological deficits may become irreversible if parenteral B12 treatment is delayed.



Gastroenterologic Manifestations

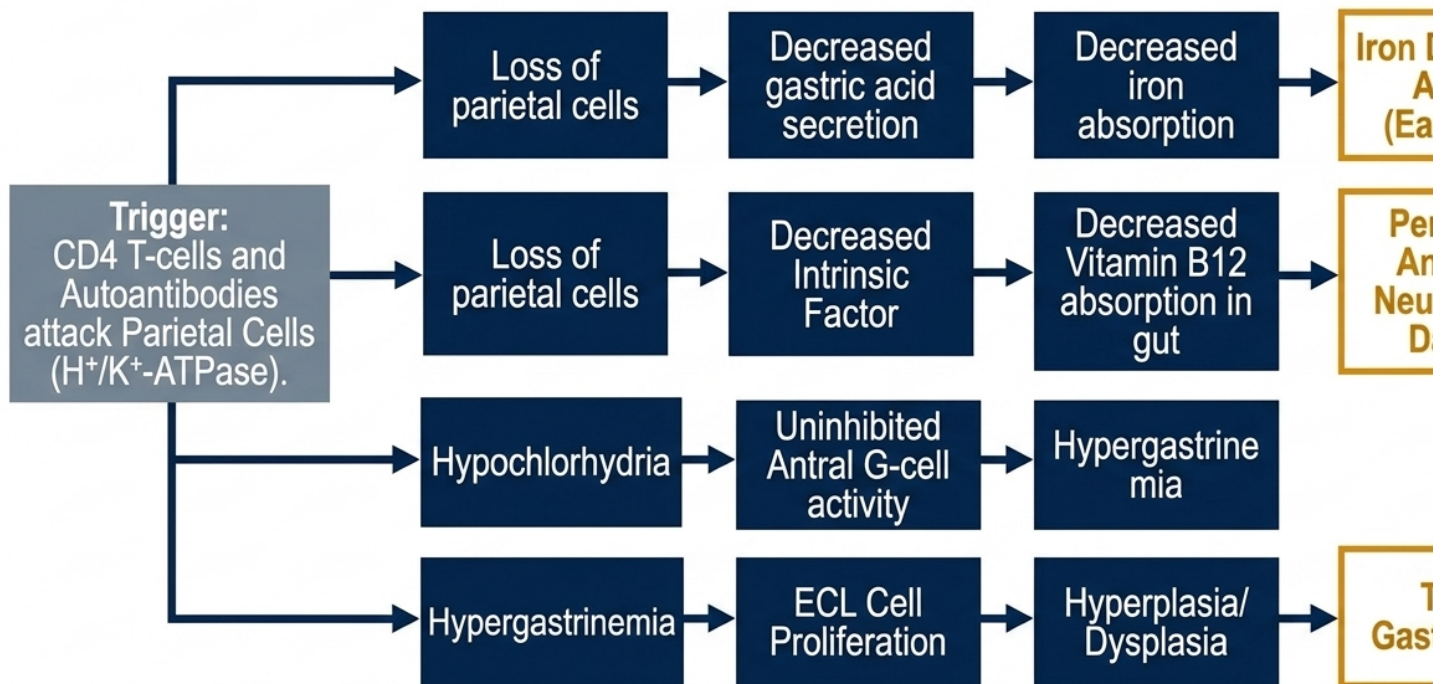
- Dysmotility-type dyspepsia, postprandial fullness, early satiety.
- Atrophic glossitis (burning sensation on tongue).
- Bacterial overgrowth of the small intestine due to achlorhydria.

Etiology Matrix: AMAG vs. HpAG

Feature	Autoimmune (AMAG)	Infectious (HpAG)
Pattern of Distribution	Corpus and fundus predominant (spares antrum)	Multifocal (includes antrum) and spreads to corpus
Cellular Hyperplasia	ECL cell & Parietal cell pseudohypertrophy are common	Rare (usually secondary to atrophy)
Inflammatory Depth	Involves lamina propria	Superficial layers (basal layer) with active inflammation
Biomarker: Gastrin-17	High	Low (but may be elevated in atrophic gastritis)
Biomarker: Pepsinogen I	Low	Normal
Antibodies Present	Against parietal cells (PCA) and intrinsic factor (IFA)	Against H. pylori

Shared Clinical Outcome: Both etiologies carry an increased risk of gastric adenocarcinoma and carcinoid

Pathogenesis of AMAG: A Systemic Domino Effect



Atrophic Gastritis (AG):

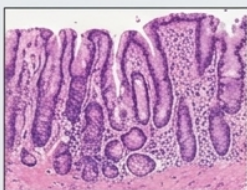
A preneoplastic condition defined by the loss of gastric glands ± metaplasia in the setting of chronic inflammation. Up to 15% prevalence in US populations.

The Correa Cascade



Normal

Node 1
Normal Mucosa: Round pit patterns (corpus), elongated (antrum).

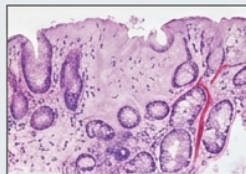


Normal mucosa
(Antrum/Fundus)



Atrophic gastritis

Node 2
Atrophic Gastritis (AG): Loss of folds, pallor, prominent submucosal venules.

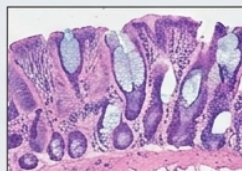


Atrophic gastritis
with glandular loss



Intestinal metaplasia

Node 3
Gastric Intestinal Metaplasia (GIM): Tubulovillous pattern, Light Blue Crests.

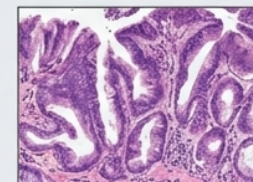


Intestinal metaplasia
(Complete/Incomplete)



Dysplasia

Node 4
Dysplasia: Irregular vessel patterns, disordered epithelial growth.

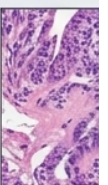


Dysplasia
(Low grade/High grade)



Early adenocarcinoma

Node 5
Gastric Adenocarcinoma (GA): (0.3% of c architecture)



Gastric Adenocarcinoma



Chronic AG carries a dual threat—of both Gastric Adenocarcinoma and Neuroendocrine Tumors (NETs) (



PATHOGEN-MEDIATED (CHRONIC)

Helicobacter pylori infection

> Mechanism

- Pro-inflammatory cytokines cause direct epithelial injury.

! Ultimate Threat

- Intestinal Metaplasia & Adenocarcinoma.



IMMUNE-MEDIATED (CHRONIC)

Autoimmune Metaplastic Atrophic Gastritis (AMAG)

> Mechanism

- CD4 T-cell autoimmune destruction of oxyntic mucosa.

! Ultimate Threat

- Pernicious Anemia & Type-1 Neuroendocrine Tumors.



ISCHEMIC / STRESS MEDIATED (ACUTE)

Critical Care Stress (SRMD)

> Mechanism

- Splanchnic hypoperfusion and mucosal barrier breakdown.

! Ultimate Threat

- Rapid, clinically significant gastrointestinal bleeding.

Diagnostic Blueprint: Confirming the Pathology

Active / Symptomatic Cases



1-Minute Ultra-Rapid Urease Test



Breath Test (*H. Pylori* confirmation)



Culture & Sensitivity



Chronic / Structural Investigation



Endoscopy (Direct visualization)



Upper GI Radiography



Histologic Assessment (Biopsy)



Serologic Antibody Test (Type A)

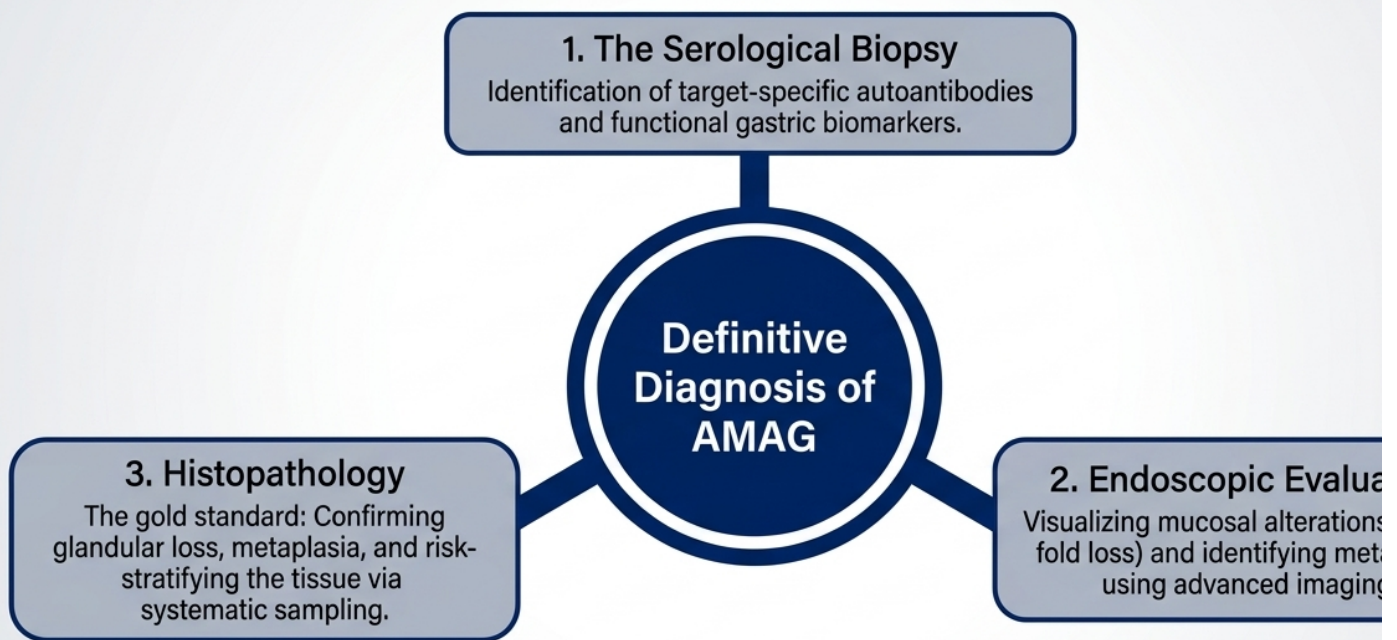


Gastric Acid Profiling
(Achlorhydria, Hypochlorhydria, Hyperchlorhydria)

! Adjuvant Tests (Triggered by severe vomiting) !

! Serum electrolytes | Hemoglobin (Hb) | Total Leucocyte Count (TLC) | Differential Leucocyte Count

The Diagnostic Triad



Takeaway Note: Clinical presentation alone is insufficient. Due to vague early symptoms, diagnosis requires this combined multi-modal approach.

The Serological 'Biopsy'

Parietal Cell Antibodies (PCA)



Status: Highly Sensitive (85-90%).

Caveat: False positives possible with *H. pylori*. May disappear in late-stage disease as the target oxyntic tissue is completely destroyed.

Intrinsic Factor Antibodies (IFA)



Status: Highly Specific (98%).

Clinical Value: Lower sensitivity (60%), but highly correlated with gastric atrophy and pernicious anemia when positive.

Pepsinogen I/II Ratio



Mechanism: PGI is secreted by oxyntic mucosa; PGII by pyloric glands. PGI drops as corpus atrophies.

Diagnostic Threshold: A PGI:PGII ratio < 8 is a highly specific, potential single-measurement screen for atrophy.

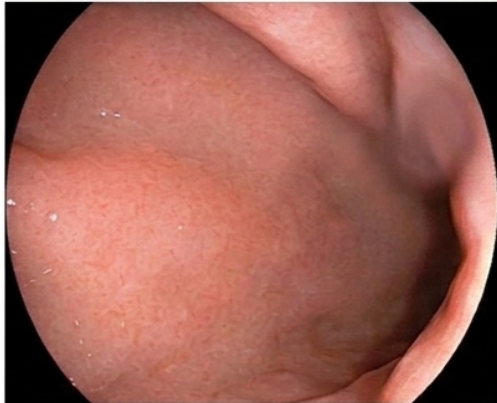
Gastrin-17



Mechanism: Elevated due to G-cell hyperplasia compensating for achlorhydria.

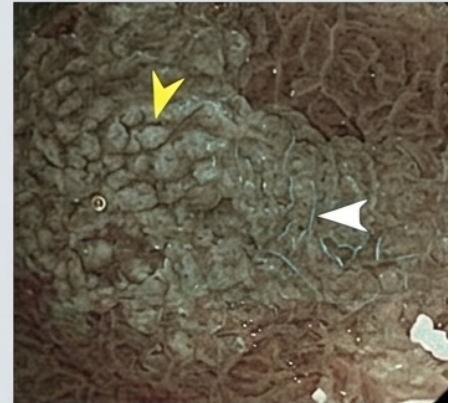
Endoscopic Appearance: Visualizing Atrophy

White Light Endoscopy (WLE)



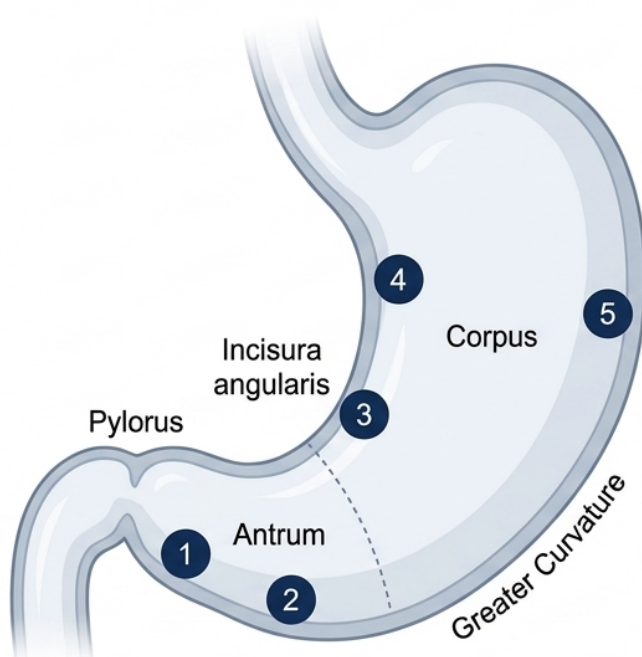
- **Visual Findings:** Pale mucosal appearance, flattened/absent rugal folds.
- **Key Sign:** Increased visibility of submucosal blood vessels due to profound thinning of the gastric mucosa ('mucosal desertification').

Narrow Band Imaging (NBI)



- **Visual Findings:** Essential for identifying Intestinal Metaplasia (IM).
- **Light Blue Crest (LBC) (Yellow Arrowhead):** blue-white lines on the crests of the epithelial surface.
- **White Opaque Field (WOF) (White Arrow):** Light scattering at microscopic lipid drops accumulating in IM mucosa.

The Sydney Biopsy Protocol: Systematic Sampling



The Protocol (5 Standard Biopsies):

- **Antrum: 2 biopsies** (lesser + greater curvature, from pylorus).
- **Incisura angularis: 1 biopsy.**
- **Corpus: 2 biopsies** (lesser curvature 4cm proximal to incisura + greater curvature 8cm from cardia).

Specimen Handling Rules:



- Standard Sydney biopsies must be placed into at least 2 separate jars (Antrum vs. Corpus) to map atrophy extent.
- Targeted biopsies from any visible abnormalities go in their own separate jar.

Endoscopy Pearls:

Distend gastric folds, clean mucosa with defoaming agent, use image-enhanced endoscopy (NBI).

The Sydney System Parameters

Core histological

☒ Chronic Inflammation

☒ Activity (Neutrophilic Infiltrate)

☒ Atrophy

☒ Intestinal Metaplasia

☒ Lymphoid Follicles



H. Pylori Diagr Correlation

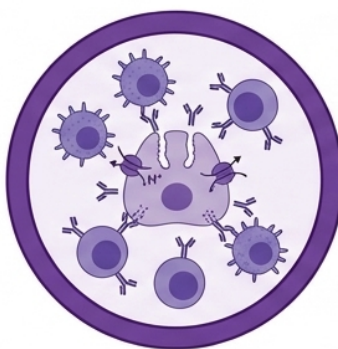
89.28

HP positivity is f
in cases of sev
inflammation

Clinical Insight:

Neutrophilic activity and follicle presence are high indicators for HP. The pre one of these histological is a strong indicator for tl

Autoimmune Targeting



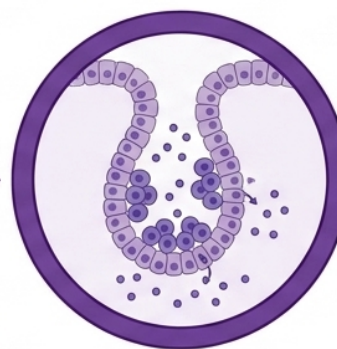
Gastric H⁺/K⁺-ATPase reactive CD4 T-cells infiltrate the lamina propria. B-lymphocytes produce anti-parietal cell antibodies (PCA) and anti-intrinsic factor antibodies (IFA).

Parietal Cell Destruction



Oxyntic glands in the corpus and fundus are destroyed (sparing the antrum). Result: Hypochlorhydria and a drop in Pepsinogen I.

G-Cell Hyperplasia



Absence of acid feedback induces excessive gastrin production in the antrum.

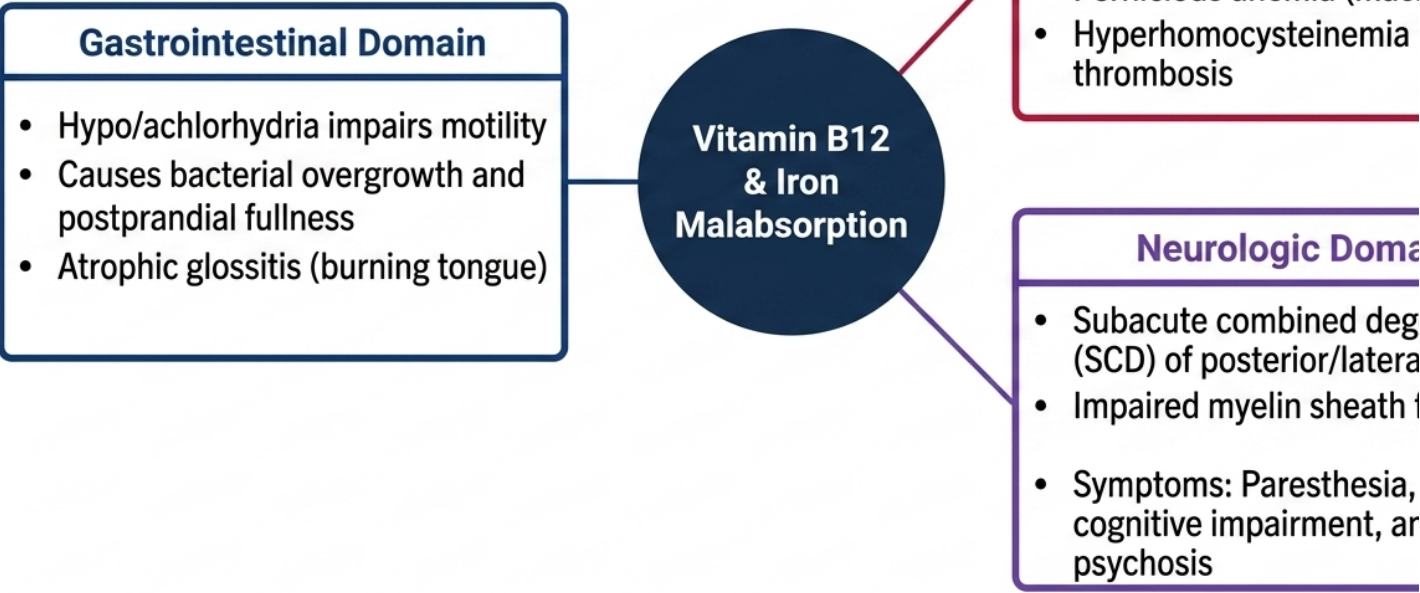
ECL Proliferation



Elevated gastrin stimulates Enterochromaffin cells, leading to linear/nodular hyperplasia and risking T neuroendocrine

Differential Diagnosis: Autoimmune vs. Pathogen-Induced Atrophic Gastritis

	 AMAG	 H. Pylori
Pattern of Distribution	Corpus and fundus (spares antrum)	Multifocal (including antrum)
ECL Cell Hyperplasia	Common	Rare
Inflammatory Depth	Lamina propria primarily involved	Superficial layers (active, infiltrative)
Gastrin-17 Level	High	Low (but may be elevated in antral disease)
Pepsinogen I Level	Low	Normal
Antibodies	Against parietal cells & intrinsic factor	Against H. Pylori

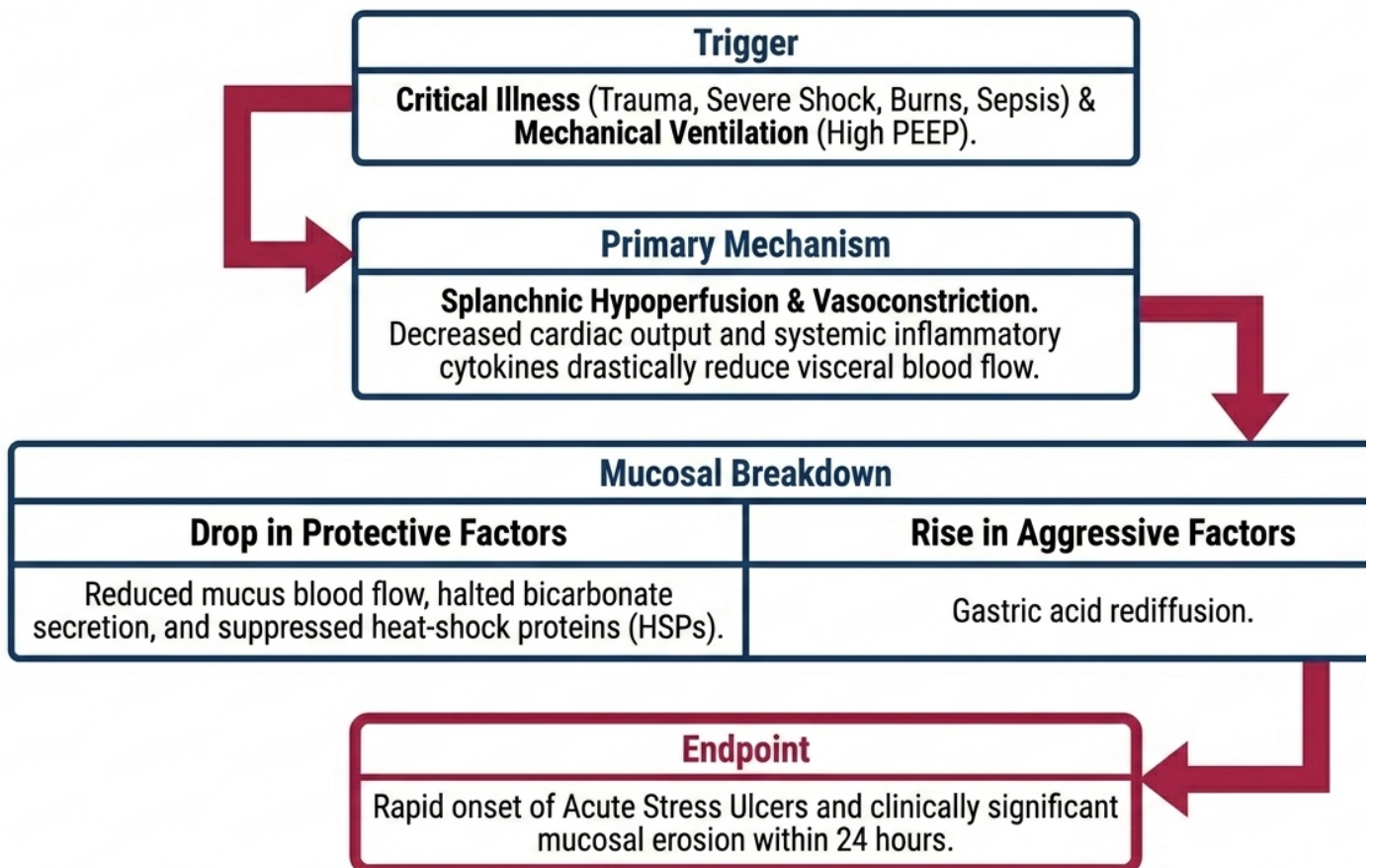




Endoscopic View: Advanced AMAG showing flattened rugal - folds and visible sub-mucosal vessels in the gastric corpus and fundus.



Histopathological View: H&E staining reveals atrophy, dense inflammatory infiltration, and intestinal metaplasia. Immunohistochemistry confirms hyperplastic changes.



Histological Assessment & Risk Stratification

Top Layer: Staging Systems for Cancer

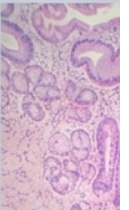
- **OLGA:** Assesses severity of both antrum and corpus. Stages III and IV indicate high gastric cancer risk.
- **OLGIM:** Considers only intestinal metaplasia stage. Underestimate risk if non-metaplastic atrophy is present.

Middle Layer: High-Risk Histological Markers

- **Incomplete GIM:** Colonic-type metaplasia without brush borders (high risk) vs. complete small-intestinal type.
- **Corpus-Extension:** Extensive involvement (e.g., antral GIM extending into corpus).
- **Severe Atrophy/GIM:** $\geq 2/3$ of glands involved in a specific gastric region.

Base Layer: Mandatory Pathology Reporting

Must report: Presence/absence of *H. pylori*, Intestinal Metaplasia (IM) subtype, and anatomical extent.



Stress Ulcer Prophylaxis: Risk Stratification Score

Tier 1: Highest Risk (Crimson Alert)

- ✓ Mechanical ventilation for > 48 hours
- ✓ Coagulopathy (Platelets < 50,000, INR > 1.5, or PTT > 2x control)
- ✓ Extensive burns (>30% of body surface area)

Tier 2: Other High-Risk Factors (Amber Alert)

- ✓ Shock, severe sepsis, multisystem trauma, traumatic brain/cord injury, kidney failure, steroid therapy

Clinical Context Bar

Context: Mucosal damage occurs in 75-100% of ICU patients within 24 hours. Clinically significant bleeding (BP drop, Hemoglobin drop > 2g/dL) occurs in < 5%, but drastically increases with higher risk factors.

Stress ulcer prophylaxis risk stratification scorecard

Minor risk

ICU >7 days

Occult GI bleeding ,

therapeutic anticoagulation ,

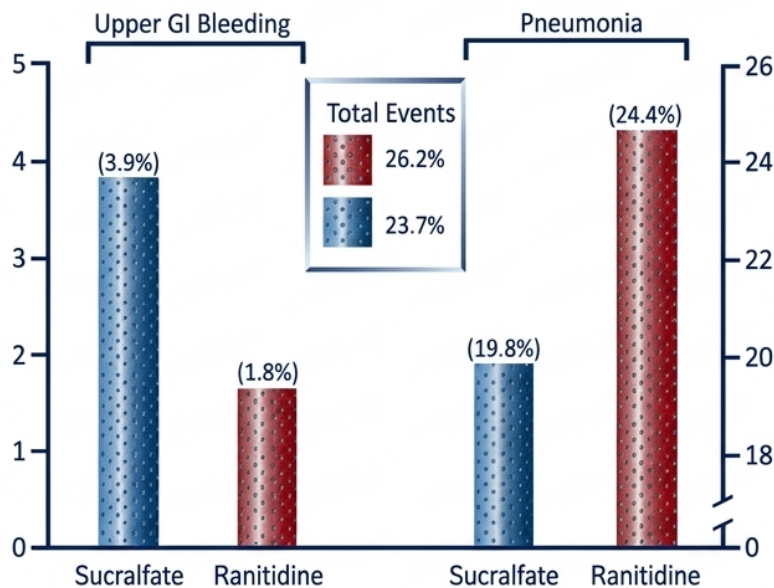
Dual antiplatelet therapy ,

Chronic NSAID use,

enteral nutrition not established'

INTERPRETATION

- *0-2 no stress ulcer prophylaxis*
- *3-4 consider prophylaxis based on clinical judgment*
- *>5 prophylaxis recommended*



Sucralfate lowers the risk of pneumonia but has a higher rate of upper GI bleeding compared to Ranitidine.

H2 Blockers (e.g., Famotidine 20mg IV)

Highly effective at reducing bleeding by blocking histamine receptors.

Risk: Increased infectious gastroenteritis and pneumonia.

Dose adjustment needed for renal failure.

PPIs (e.g., Pantoprazole 40mg IV)

Greater, longer acid reduction.

Inhibits H⁺/K⁺-ATPase.

Metabolized in the liver.

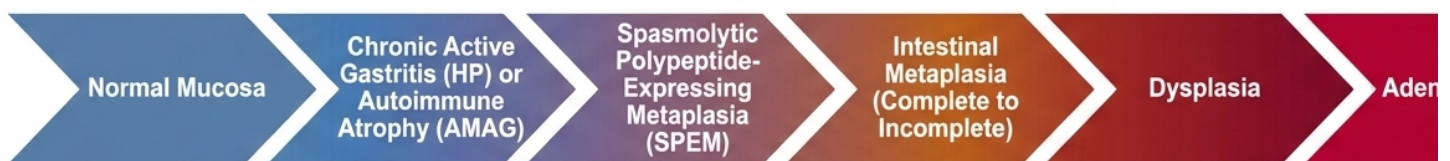
Risk: May inhibit clopidogrel activation.

Sucralfate (1g q6h)

Cytoprotective agent. Forms a viscous protective fluid without altering gastric pH.

Note: Enteral tube feeding provides a trophic effect and increases luminal pH. Patients successfully receiving enteral nutrition generally do not require pharmacological prophylaxis.

The Precancerous Pathway: Transition to ESGE MAPS III Guidelines

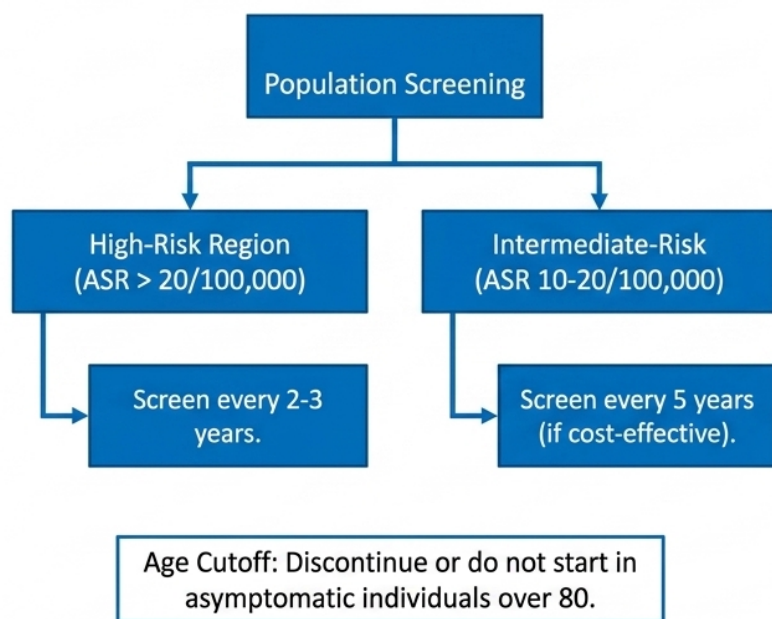


MAPS III Diagnostic Standard

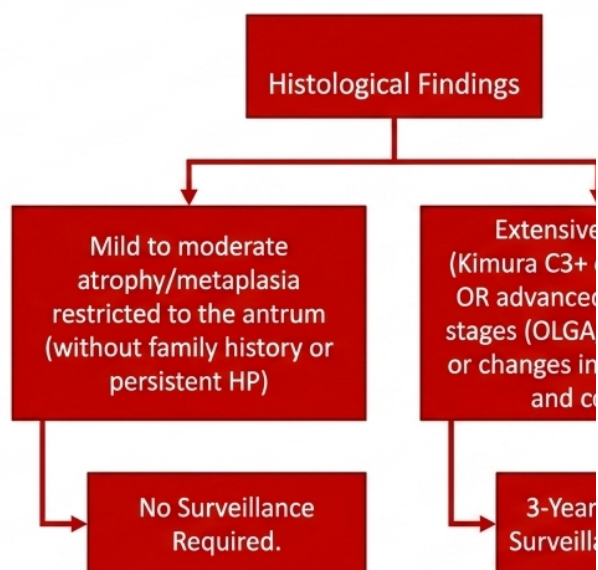
- High-quality endoscopy utilizing Virtual Chromoendoscopy (VCE) is mandatory for staging.
- Biopsies must be taken from at least two topographic sites (2 from antrum/incisura, 2 from corpus) in separate vials.

ESGE MAPS III: Screening and Surveillance Logic

Population Screening Logic

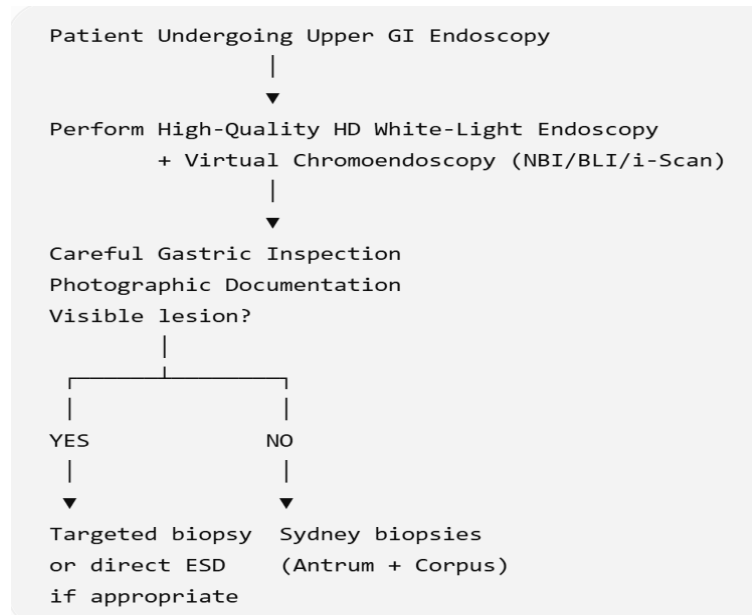


Histological Surveillance Logic



System	Method	What it assesses
Kimura-Takemoto	Endoscopy	Atrophy extent (Closed vs Open)
OLGA	Histology	Atrophy severity + topography
OLGIM	Histology	Intestinal metaplasia severity + topog
EGGIM	Endoscopy	Intestinal metaplasia extent (real-time)

Overall Clinical Algorithm



Standard Biopsy Protocol

No Visible Lesion

↓

Biopsy Protocol

2 × Antrum (+ incisura)

2 × Corpus

↓

Two Separate Containers

↓

Histology

OLGA

OLGIM

H. pylori Assessment

Risk Stratification

Endoscopic Staging

EGGIM Score

+

Kimura-Takemoto Atrophy

↓

Histologic Staging

OLGA

OLGIM

↓

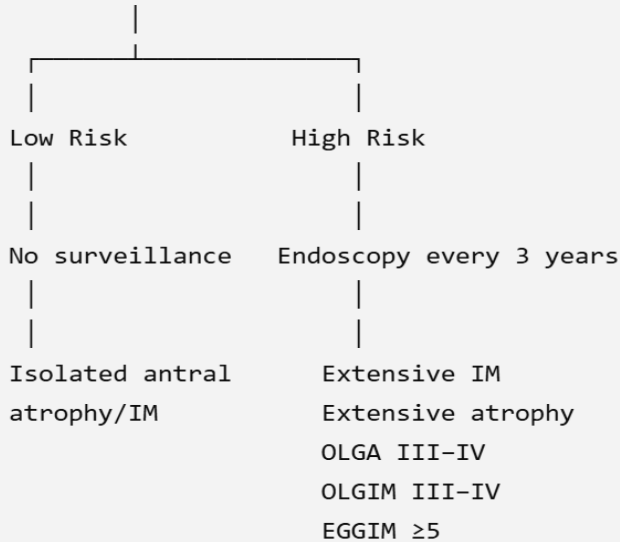
Overall Gastric Cancer Risk

High-risk findings include:

- Extensive intestinal metaplasia
- Corpus involvement
- OLGA III–IV
- OLGIM III–IV
- EGGIM ≥ 5
- Advanced endoscopic atrophy

Surveillance Algorithm

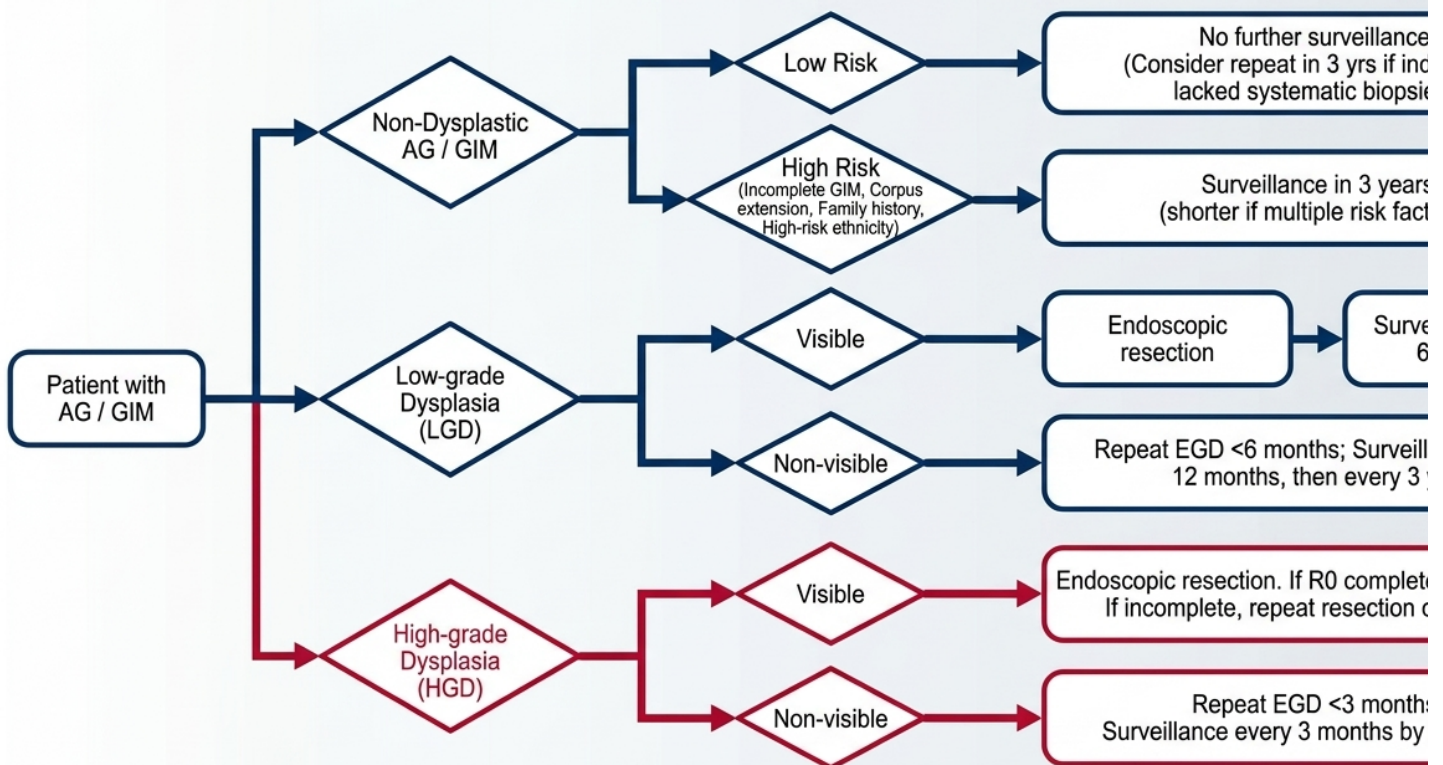
Risk Assessment



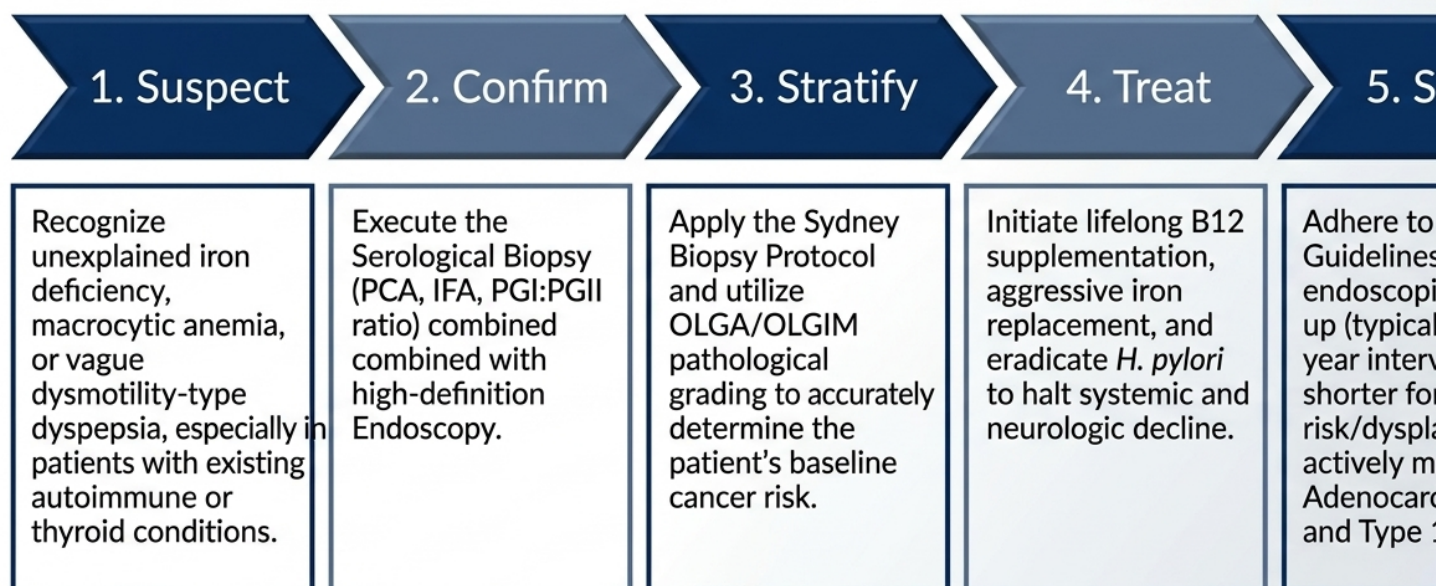
Additional factors increasing surveillance intensity:

- Family history of gastric cancer
- Persistent *H. pylori*
- Incomplete intestinal metaplasia

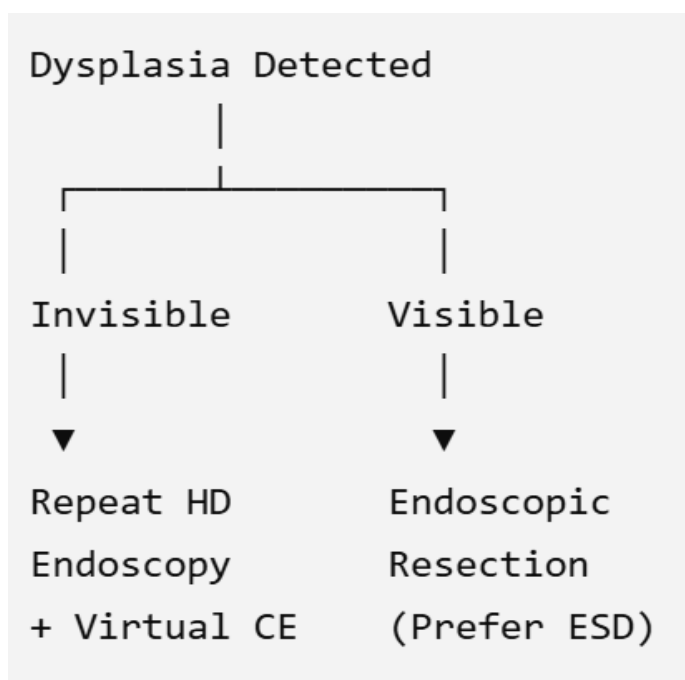
Endoscopic Surveillance Algorithm (ACG Guidelines)



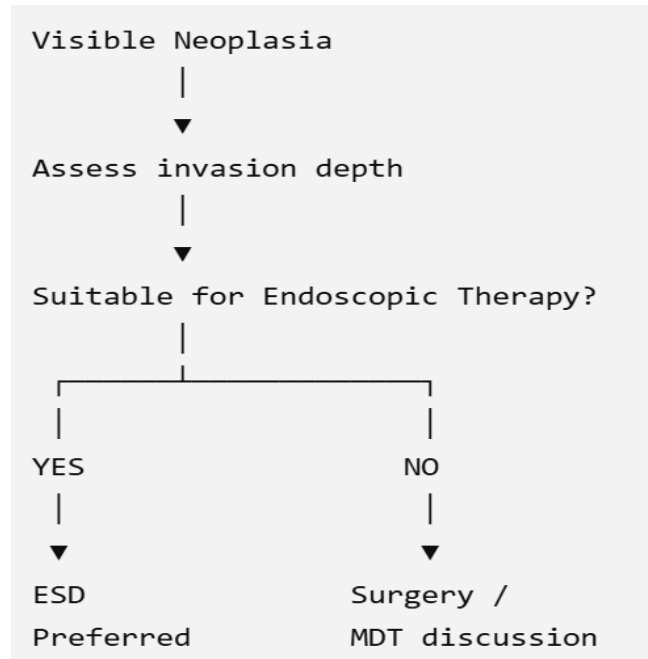
Synthesis: The AMAG Care Continuum



Dysplasia Management



Endoscopic Treatment



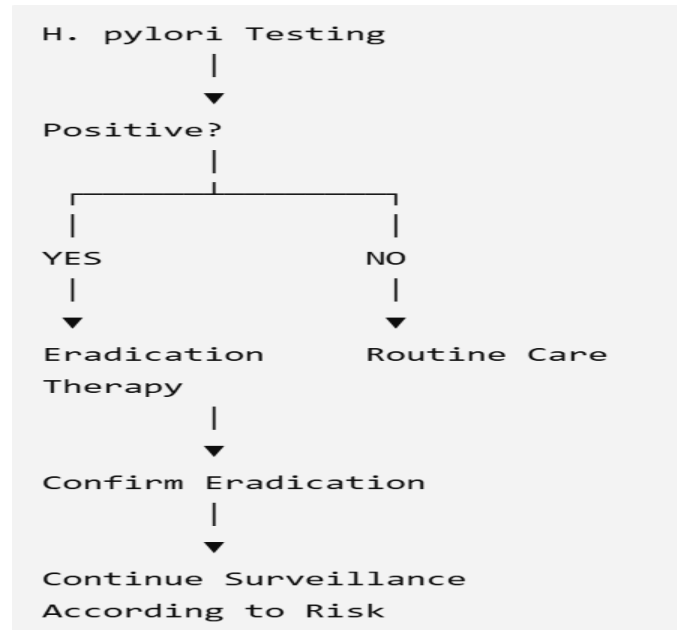
Preferred treatment

- Low-grade dysplasia
- High-grade dysplasia
- Intramucosal carcinoma
- Selected superficial submucosal cancers

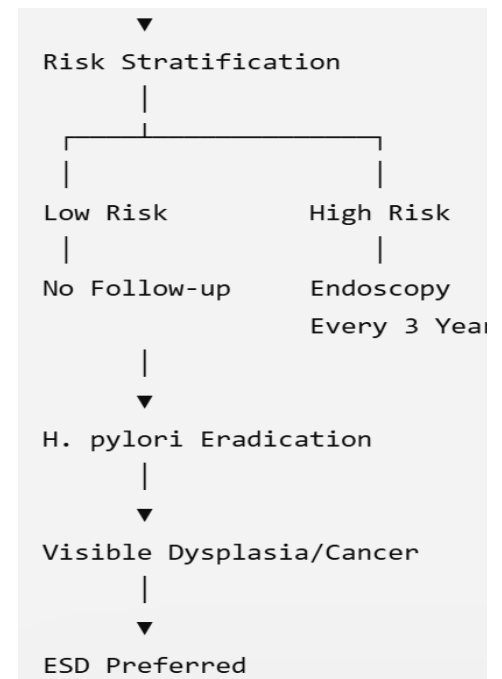
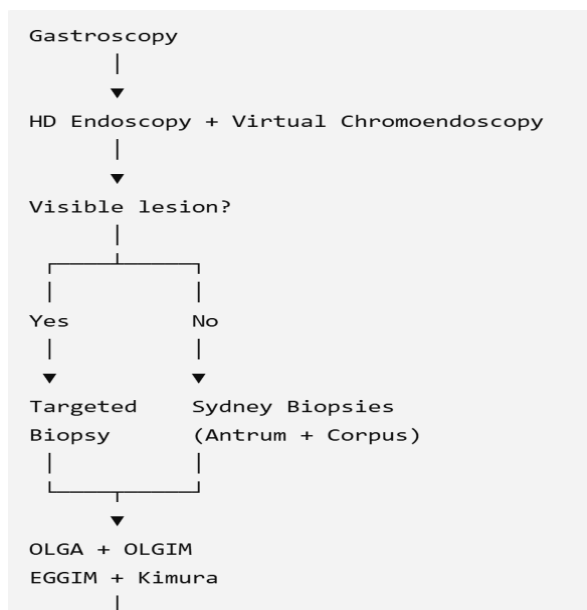
EMR only for selected small (≤ 10 mm), low-risk superficial lesions.

Routine EUS or CT before ESD is **not** recommended unless deep invasion is su

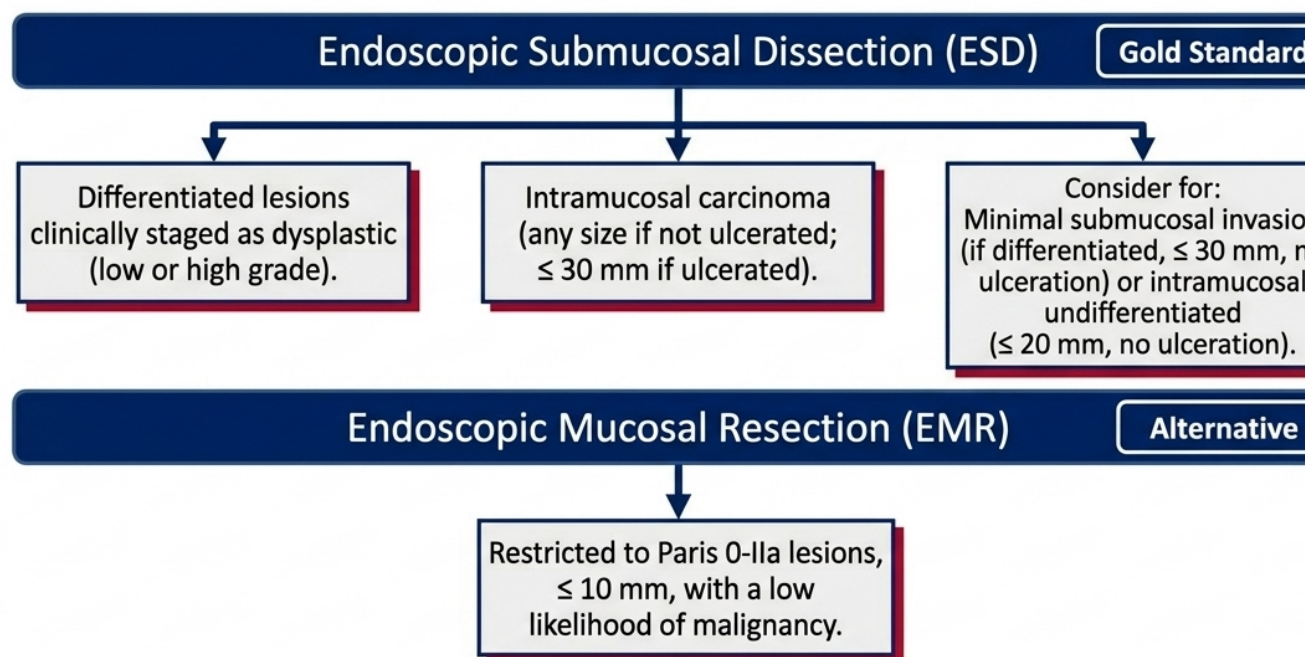
Helicobacter Pylori Management



MAPS III Clinical Pathway (One-Slide Summary)



Endoscopic Resection Criteria (ESD vs. EMR)

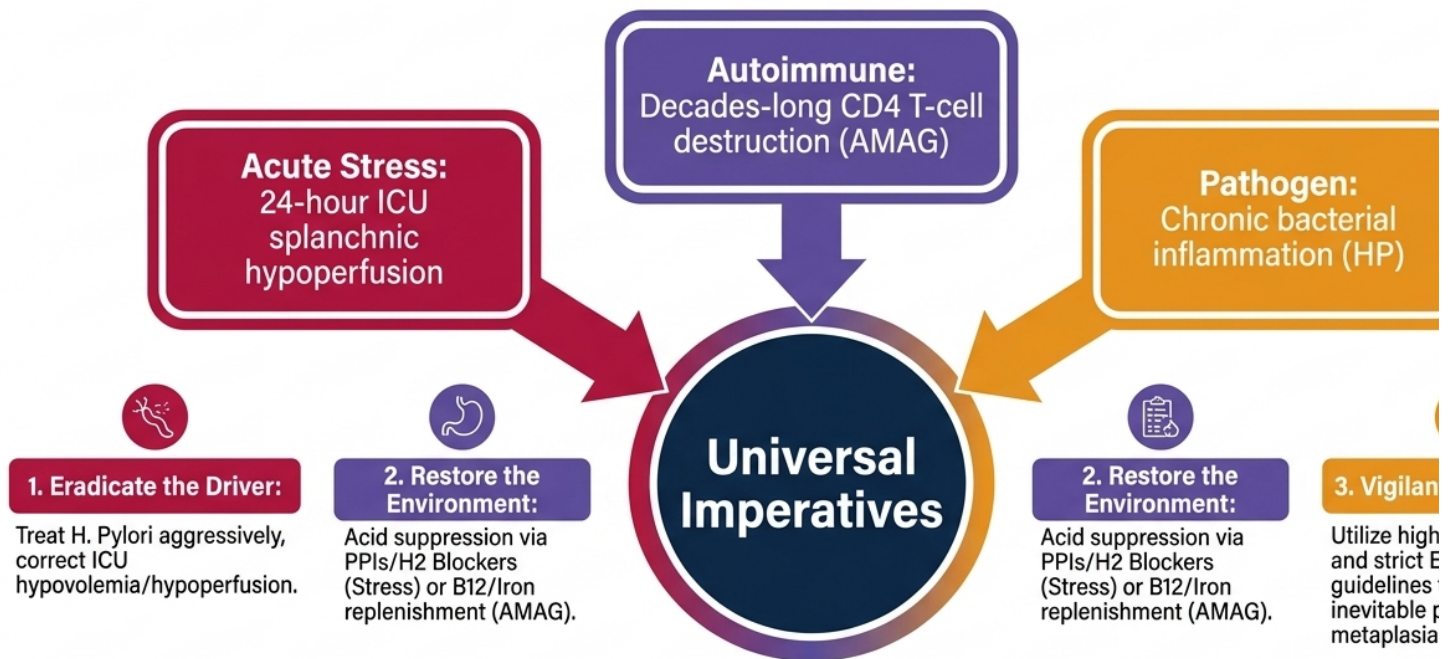


Pre-Op Rule Alert Box: Do NOT routinely perform EUS, CT, MRI, or PET prior to resection unless deep submucosal invasion is suspected.

Post-Resection Risk Grid: Pathological Staging & Action

Curative / Very Low-Risk En bloc R0. Differentiated, pT1a, no lymphovascular invasion. Any size if no ulcer; ≤ 30 mm if ulcerated. LNM risk < 0.5-1%. Action: No further treatment.	Curative / Low-Risk En bloc R0, no lymphovascular invasion. pT1b (≤ 500 µm, differentiated, ≤ 30 mm) or pT1a (undifferentiated, ≤ 20 mm, no ulcer). LNM risk < 3%. Action: Multidisciplinary discussion, generally no further treatment.
Local-Risk Very low LNM risk, but increased local persistence (e.g., piecemeal resection or tumor-positive horizontal margin of an otherwise very-low-risk lesion). Action: Endoscopic surveillance/re-treatment.	High-Risk / Noncurative Positive vertical margin, lymphovascular invasion, deep submucosal invasion (> 500 µm), poorly differentiated with ulcer or > 20 mm. Action: Complete staging, strong consideration for surgery.

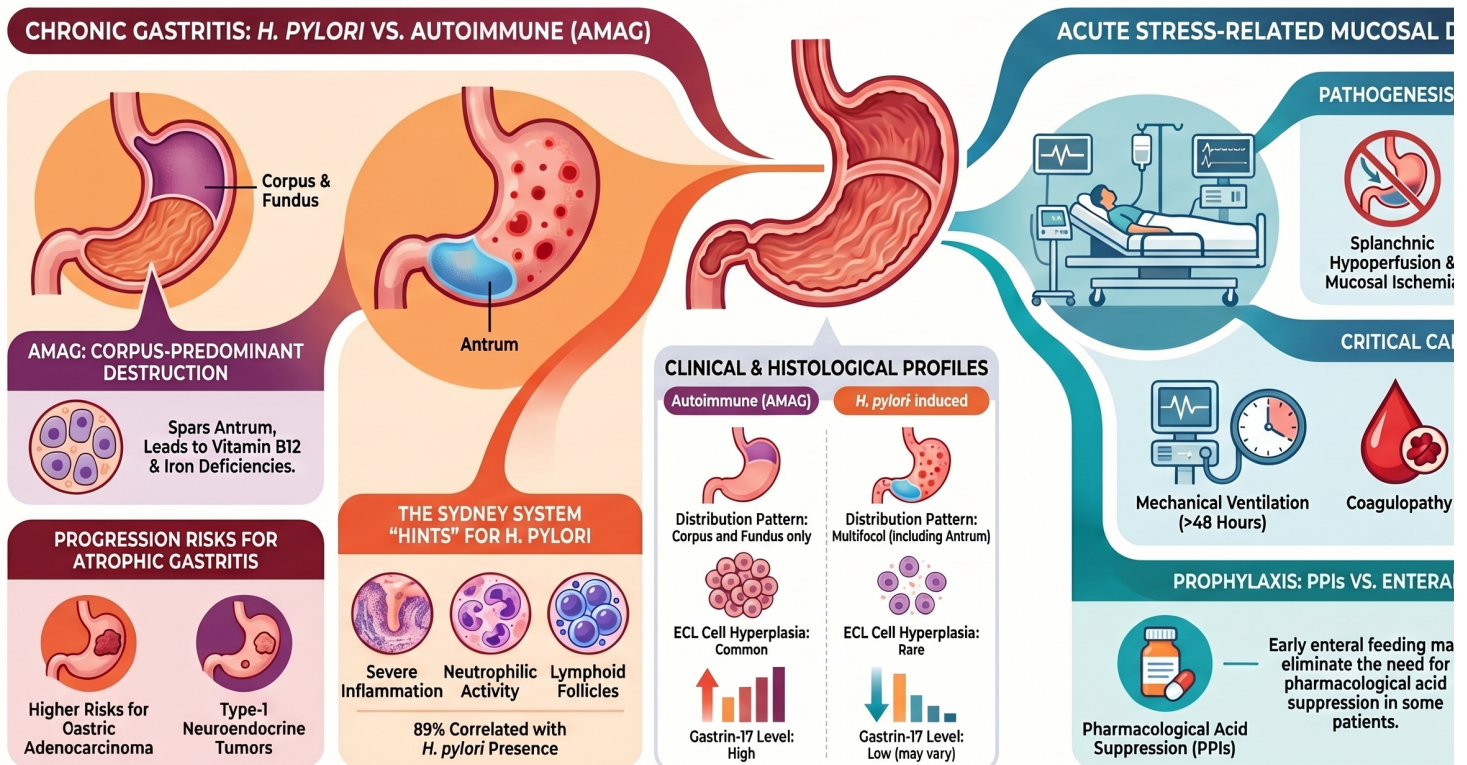
The Nexus of Gastric Management



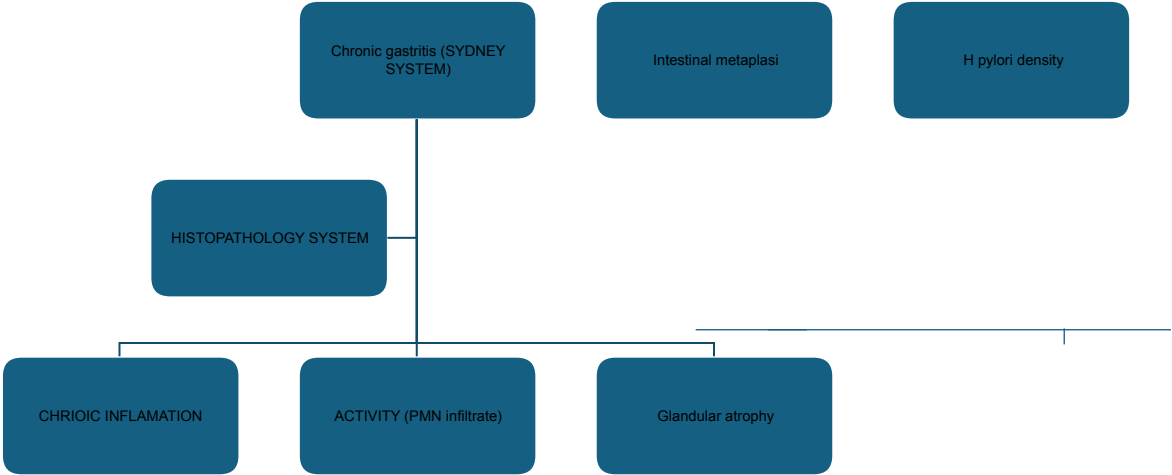
Closing Insight: The stomach is an environment defined by its balance of aggressive acids and defensive mucus. Mastery of gastric pathology requires intercepting the timeline precisely when that balance fails.

DECIPHERING GASTRITIS: CLINICAL MARKERS, SUBTYPES, AND DIAGNOSTICS

Overview of chronic *H. pylori* and Autoimmune (AMAG) subtypes, with key histopathological markers and clinical risks of acute stress-related mucosal damage.



Gastritis Types & Management



H. Pylori Correlation

Strong Association with Activity

High Positivity in Severe Inflammation

Lymphoid Follicle Indicator

Grading (Houston Update)

Absent (Score 0)

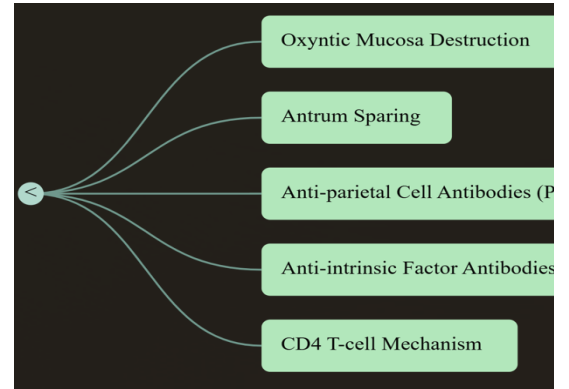
Mild (Score 1)

Moderate (Score 2)

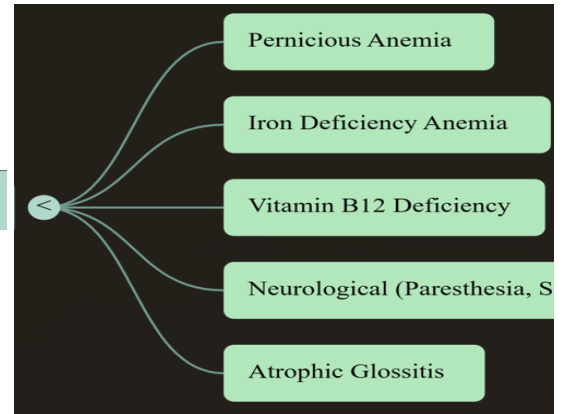
Severe (Score 3)

Autoimmune Metaplastic Atrophic Gastritis (AMAG)

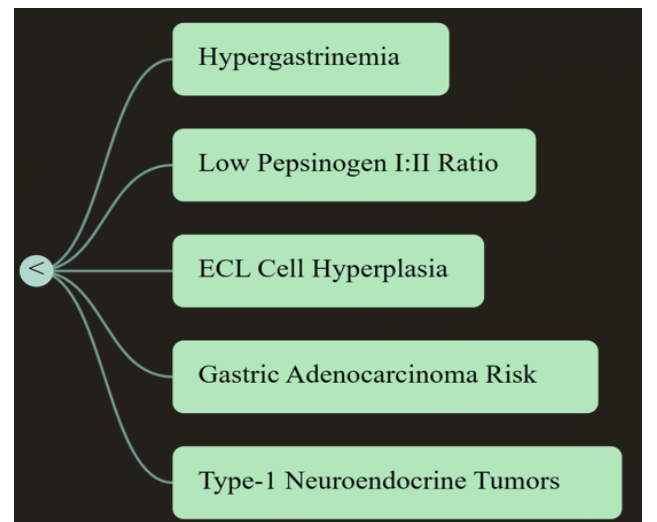
Pathogenesis

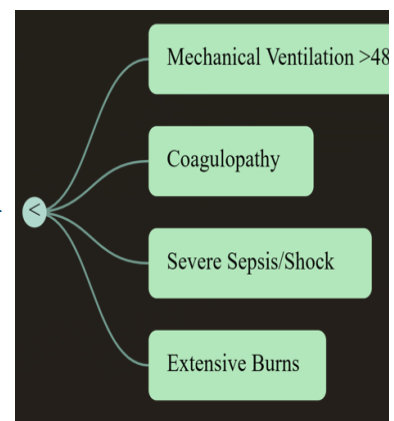
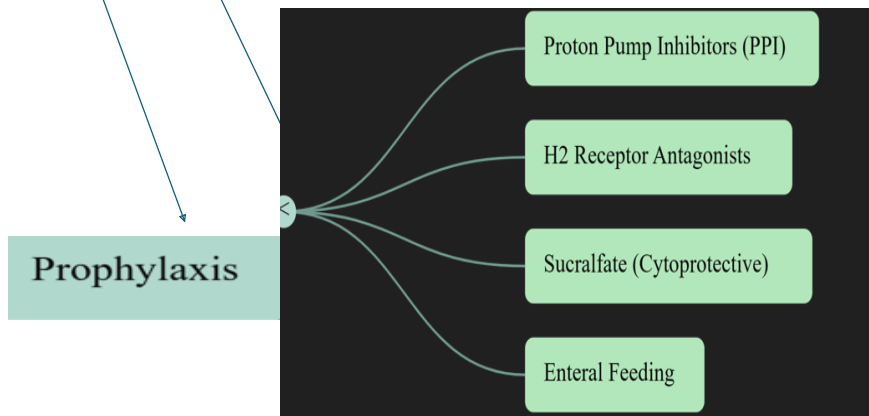
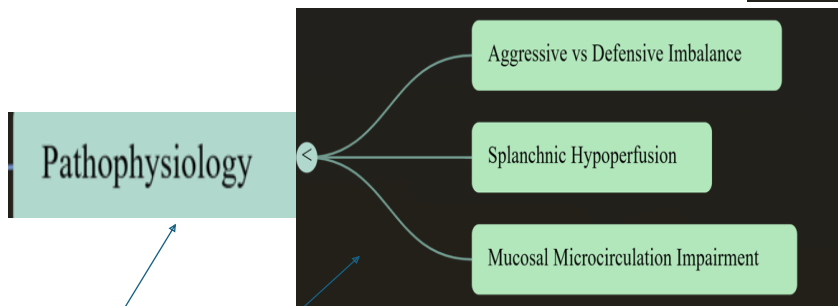
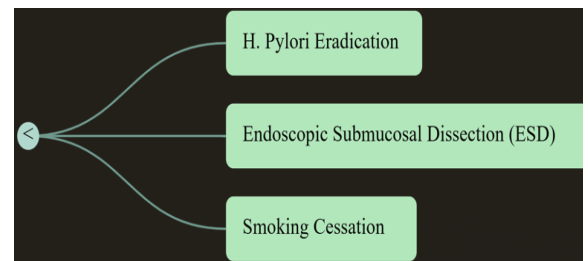
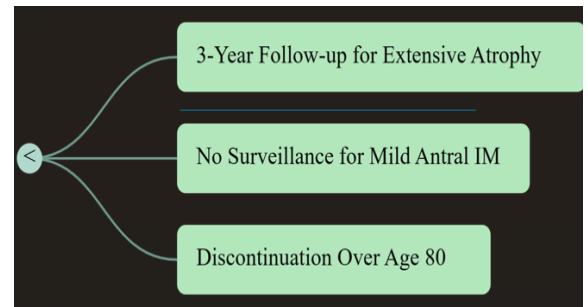
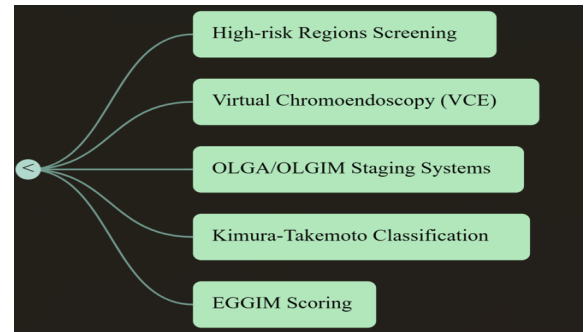
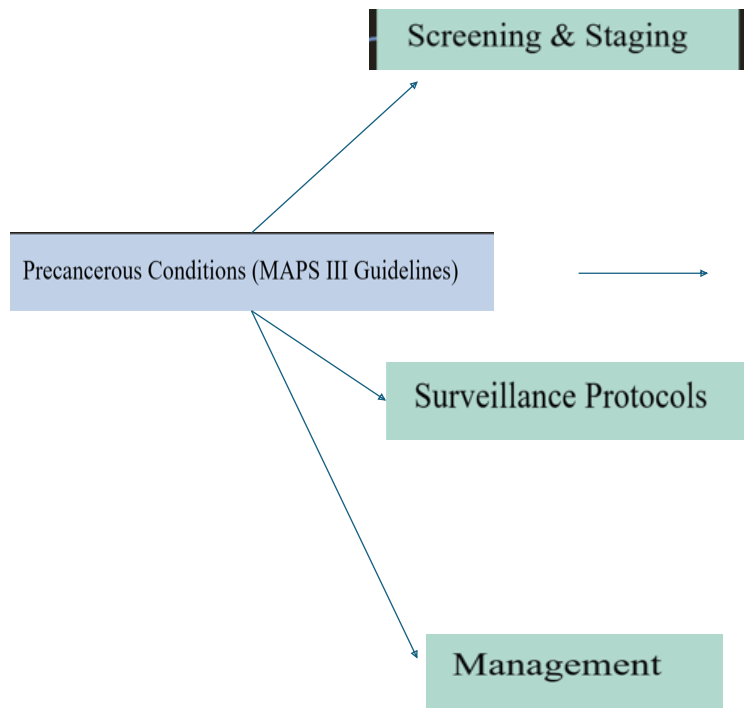


Clinical Manifestations

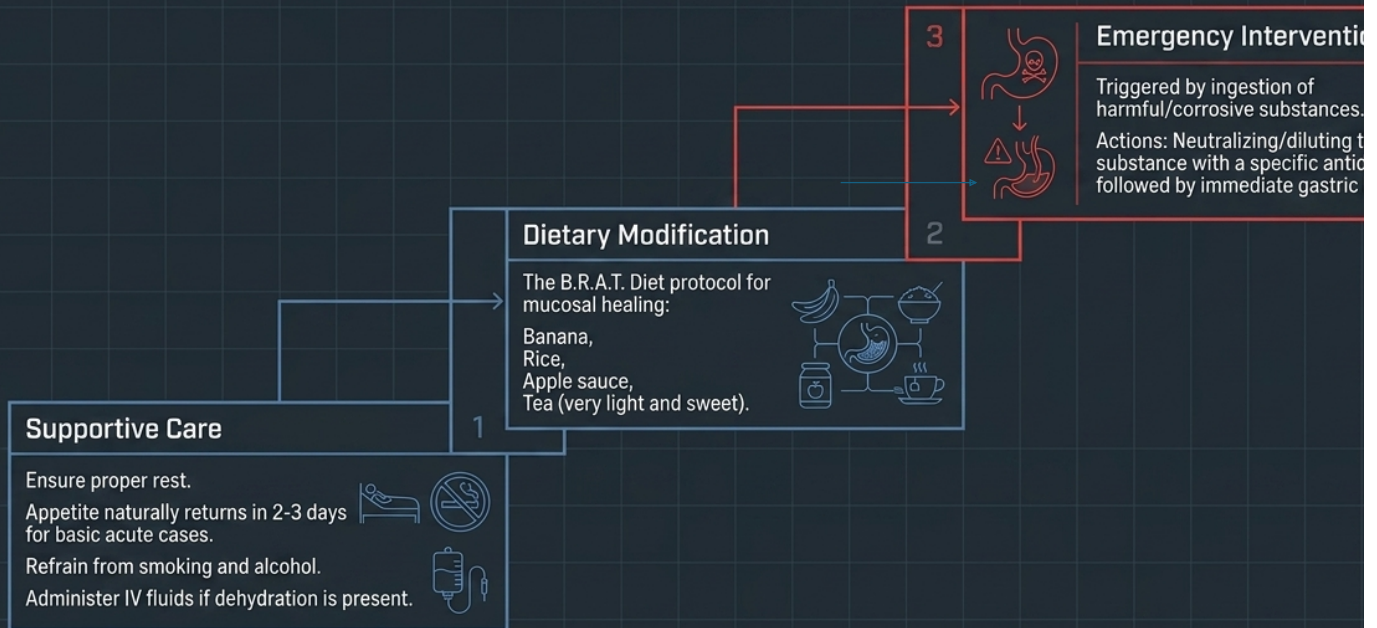


Diagnosis & Complications





The Intervention Ladder: Non-Pharmacological Management

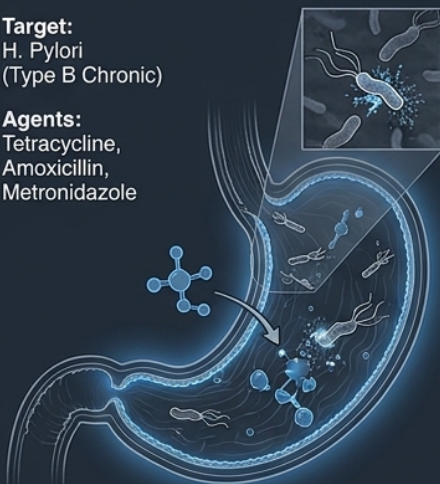


The Pharmacological Arsenal

Antibiotics (The Eradicators)

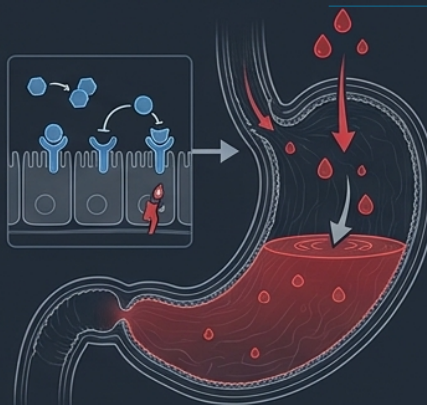
Target:
H. Pylori
(Type B Chronic)

Agents:
Tetracycline,
Amoxicillin,
Metronidazole



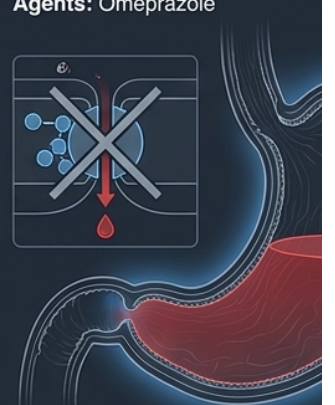
Histamine H2-Receptor Antagonists (The Reducers)

Target: Suppressing baseline acid production
Agents: Cimetidine, Ranitidine



Proton Pump Inhibitors (The Shutdown)

Target: Complete blockade of gastric acid secretion to allow mucosal healing
Agents: Omeprazole

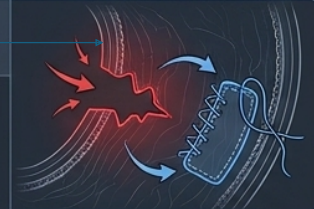


Surgical Fallbacks: When the Shield Fails

Surgical management is strictly not needed for standard acute gastritis, and is only rarely utilized in severe, chronic recurrent cases.

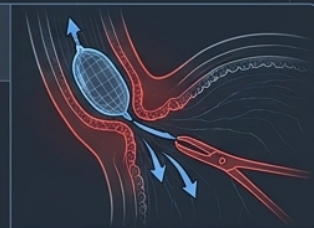
Pathway 1: Reconstruction of Perforation

Context: When acute ulcers or severe gangrene erode entirely through the gastric wall, requiring **immediate physical patching**.



Pathway 2: Cardiac Sphincter Stenosis Repair

Context: When chronic inflammation and scarring lead to a **stricture** at the gastroesophageal junction, requiring mechanical dilation or **surgical alteration**.



Gastritis: The Clinical Blueprint – From Mucosal Breach to Mana

